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Ergonomics in the operating room

Ergonomía en el quirófano

Abilene Cirenia Escamilla-Ortiz,* Josefina Serrano-Pérez‡

The word ergonomic comes from the Greek *ergos* –work– and *nomos* –laws, and rules–. It follows the natural laws of human work and nature; in short, everything is in the right way and place. Ergonomics is essential for the surgeon, but many do not consider it. The surgeon must adapt to the work environment.

The tasks that the surgeon performs daily not only require mental clarity, hand and eye coordination, concentration, and precision in the execution of movements but also to remain in the same posture for periods ranging from minutes to hours.

In recent years, the patient benefit has been weighted "first, not harm", but we have forgotten about the surgeon's care and well-being.[1] Lack of ergonomics training and subsequent implementation during surgical procedures leads to discomfort and pain that results in fatigue, which can affect speed, stamina, and concentration.[1]

Work-related musculoskeletal disorders are repetitive strain injuries that can damage muscles, nerves, and joints (neck, back, waist, wrist, and hands).[2]

In vaginal surgery procedures, injuries are reported from 54 to 87%, laparoscopic surgery from 73 to 100%, robotic surgery from 23 to 80%, and open surgery from 66 to 94%. Surgery is analogous to playing sports; it can be physically and mentally demanding, so good health and nutrition are necessary to prevent injuries.[2] Safe and effective exercises can be done before entering the operating room to improve the torso and abdomen muscles. A stretching routine should be part of any surgeon's routine to provide flexibility and decrease musculoskeletal injuries.[2,3]

Before the surgical event, the surgeon must anticipate everything that may be required during the surgery, for example, the adjustment of the lights, how the patient's arms will be positioned, and other procedures which will allow the surgeon to be comfortable. Likewise, the height of the table should be checked since if it is not adjusted, it can cause back and neck problems, so it is suggested that the neck should be flexed at 20 degrees, minimize trunk torsion, distribute the load and not block the knees, maintain a good position of the arms about the shoulders in laparoscopic surgery, as well as the placement of the monitors to avoid twisting; abduction of the shoulders should be kept at 30 degrees or less.[2,3] When holding the camera in laparoscopic surgery, the wrists should be kept in a neutral position avoiding flexion or extension for a long time. In open surgeries, the way the instruments are taken is also important to avoid injuries, for example, introducing the whole finger in the rings instead of only introducing the tips of the fingers.

The incidence of sprains or muscle strain secondary to maintaining prolonged and uncomfortable postures, as well as remaining static while holding a retractor with manual force, is frequently reported; for example, in the cervical region, it has been reported in 58.1%, dorsal 40.5%, lumbar 52.7%, wrist 27.1% and

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in the shoulders 24.3%. The percentage may vary depending on the type of surgery and the surgeon's gender; however, it is in the female gender where a higher progression is reported in the upper torso.[4]

Surgeons have challenges in the operating room; there is still much to investigate on this subject since the current tools for the study of ergonomics are limited, so measurement and improvement instruments that can be reproduced and are easy to use should be developed. Hospitals with training programs for residents should implement protocols in which ergonomics is taught to prevent conditions that could put their professional life at risk.

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Ten years experience in an extracurricular undergraduate surgery course in medicine

Experiencia de 10 años de un curso extracurricular de cirugía en pregrado de medicina

Carlos Agustín Rodríguez-Paz,* Víctor Hugo Gámez-Huerta‡

Keywords:
instructors, surgery, teaching, experimental, constructivism.

Palabras clave:
instructores, cirugía, docencia, experimental, constructivismo.

ABSTRACT

Introduction: the different proposals to bring undergraduate medical students to surgical courses have allowed the development of surgical skills. The constructivist school provides surgical teaching with new opportunities by allowing a scaffolding where several additional skills are integrated. **Objective:** to describe our experience with an experimental undergraduate surgical group. **Material and methods:** by a descriptive study, we review the students prepared in eight years in 11 courses under an evaluation scheme of the Serrano/Anaya system in live models under a routine, awaiting their products. The students who emerged from this course were prepared as coaches or instructors. **Results:** based on 109 students, an initial qualification of 32 points and a final qualification of 70 to 80 points were obtained; 60 (70%) were students, and 22 (18%) were instructors. Additionally, 15 (13%) presented papers at congresses. **Conclusions:** the Serrano/Anaya process evaluation model allowed a sequential preparation similar to that of postgraduates, and the constructivist model provided the possibility of generating products such as encouraging students to create research projects.

RESUMEN

Introducción: *las diferentes propuestas de acercar a los alumnos en pregrado de medicina a cursos de cirugía han permitido desarrollar destrezas quirúrgicas. La escuela constructivista al permitir un andamiaje donde se integran diversas habilidades adicionales brinda la posibilidad a la docencia quirúrgica de nuevas oportunidades.* ***Objetivo:*** *describir nuestra experiencia con un grupo de cirugía experimental en pregrado.* ***Material y métodos:*** *por estudio descriptivo se reseña los alumnos preparados en ocho años en 11 cursos bajo un esquema de evaluación del sistema de Serrano/Anaya en modelos vivos bajo una rutina, esperando sus productos. Se prepararon como coaching o instructores a los alumnos que emergieron de este curso.* ***Resultados:*** *con base en 109 alumnos se obtuvo calificación inicial de 32 puntos y final de calificación de 70 a 80 puntos, 60 alumnos (70%), 22 alumnos fueron instructores (18%). Adicionalmente, 15 (13%) presentaron trabajos en congresos.* ***Conclusiones:*** *el modelo de evaluación de procesos de Serrano/Anaya permitió realizar una preparación secuencial similar a la de los postgraduados, asimismo el modelo constructivista brindó la posibilidad de generar productos como incentivar al alumno a crear proyectos de investigación.*

INTRODUCTION

The challenge of preparing a surgeon encompasses both the intellectual spheres (the written material in books and journals),[1,2] of skills and competencies (knowing the material and how to use it through manual actions),[3,4] the discipline, and if we were ambitious, creating an indoctrination of innovation,[5] above all foreseeing to train the surgeon to provide an immediate response to that unexpected transoperative events[6] seeking new answers to clinical-surgical problems.

Surgery from ancient times until the eighteenth century was a Lancastrian process

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by which students learned from their tutors rudimentarily according to the patients they encountered.[7] From the 19th century onwards, William Halsted established an educational system with a defined study plan and a series of scientific elements; the surgical resident performed surgical procedures under the supervision of a graduate surgeon until he demonstrated that he had mastered the technique (Halstedian method).[8-10] At the end of the 20th century, a new system was established based on competencies and skills development through a learning curve with elements of internet-based programs to complement the non-face-to-face hours.[11]

If we recognize that the training of the resident is based on careful preparation at the undergraduate level, we will find that this foundation will depend on the elements provided in the School of Medicine; hence the importance of including morphological, physiological, biomolecular, microbiological and, of course, clinical subjects, providing not only an anatomical approach but also a biomolecular physiological approach to support clinical decisions.[1] Although it is not the intention of the undergraduate program to train surgeons but to provide the essential elements for the student to aspire to be a surgeon someday, this stage has been considered crucial to develop skills independent of their formal curriculum with which they must comply in the bachelor's degree.[12,13]

An ideal scenario where the undergraduate student acquires his or her first real surgical experience is the university's experimental surgical laboratory within such facilities.[14-16] An ideal model to provide this teaching element is the assembly of a laboratory with the indispensable elements proposed by Dr. Luis Padilla-Sánchez at the end of the last century, meaning respecting national and international regulations regarding the right to life of experimental animals. Experimental surgery practices can be performed on small species,[17,18] being a successful model for postgraduate students that influenced some undergraduate students in the medium term.[17]

Getting a student to incorporate the theoretical elements into his practice requires models that allow preserving that knowledge.[19] The models in surgical teaching help preserve the students' improvement and base a practical experience, which leads them to form skills and master simple techniques.[6,20] This experience is very similar to the constructivist school of teaching initiated by Piaget, in which the subjects only learn through reflexive abstraction, making the live models of surgical practice an ideal means to consolidate this knowledge. The student is building this new knowledge,[20] where the elements taught are beams of a scaffolding that not only allow building a piece of new knowledge on the existing one, forming a reality different from positivism,[21] but also allowing the development of tangible products through practical exercises from the acquired elements.

Under the motivation of Dr. Anaya-Prado's three-phase evaluation scheme,[22] the student was prepared in the first theoretical phase. In the second, a practical demonstration is given by an instructor. In the third, the student practices under supervision, evaluating three skill fields and observing the changes acquired.[23] In order to propose a live model, the NOM-062-ZOO-1999 regulations[24] for the use of animals and in the creation of the Sun Lee system for the use of experimental Wistar rats, and based on Dr. Padilla-Sánchez's teaching model for the use of small species for the practice of surgical procedures in an experimental microsurgical environment,[18] essential skills are developed (laparotomy, inguinal plasty, hepatic biopsy, and unilateral nephrectomy).

The constructivist teaching sense within the surgery is completed by having a didactic model that allows teaching and evaluation actions using a demonstrable product that makes it evident that the student acquired such knowledge; for the surgical area, it is a practical skill much more tangible and easy to evaluate according to Dr. Anaya-Prado's model.[22] There still needs to be a surgical group with published articles based on constructive teaching proposals, where

it is easier to apply this teaching theory in medicine.

Our objective was to describe how many students took the surgery course with the use of live biological models, how they were evaluated, how many of them are still in the career, how many concluded it, and how many have published or are about to publish papers within our School of Medicine of the Universidad Cuauhtémoc at San Luis Potosí (EMUCSLP).

MATERIAL AND METHODS

Based on a total of 11 surgery courses with the use of a live animal model between 2009 and 2019, in this case, the 250 g male Wistar type rat was used with the observation of the requirements of NOM-062-ZOO-1999,[24] as well as the ethical norms of respect for species for use in experimental surgery laboratories contained in the respective manuals,[25,26] placing our attitude more towards the bioethical personalist school of Hans Jonas than towards the utilitarian one of Hugo T Engelhardt by respecting its particularity as a living being.[27,28]

The students in the course were medical students from the second to the eighth semesters. They were taught a sequence of four blocks of knowledge to support the manipulations in the living biological model, ethical aspects, and respect for the life of small species, indispensable knowledge to perform the manipulations in the rat, fundamental knowledge to know the material to be used with the biological model, knowledge of the technique to be used and a practice module in the living model.

The sequence of knowledge to be acquired is suggested by Dr. Anaya-Prado's method,[22] in which these elements are acquired theoretically, the material to be used is known, and a teacher shows the technique to be developed. We describe for this work the results of two courses; the students perform three stations: 1) general laparotomy and biopsy, 2) nephrectomy, and 3) splenectomy.

The course is directed by a certified general surgeon and supported by a group of undergraduate medical students (coaching system),[29] of whom have already taken at least one similar course in experimental surgery in live biological models. These instructors have developed additional skills to their initial instruction and collaborate in monitoring the constant development of the skills of the new students in this course;[6] these skills were evaluated from the eighth course in 2014. The Anaya-Serrano system was used with the modification proposed by one of our alumni to evaluate the domains of knowledge, mastery of the instruments to be used, and mastery of the technique to be evaluated.[30]

The variables studied were the number of students who took the experimental surgery course in eight years, sex, and whether they managed to conclude the procedures without their rat dying, as a cross-sectional range. In a follow-up, students who reached the level of general instructors and senior instructors (general coordinator of instructors) were considered. Of all trainees, how many went on to medical school and how many presented papers at congresses are shown as percentages.

RESULTS

Out of a total of 11 courses, 149 students graduated, 137 belonged to EMUCSLP, and the rest (n = 12, 8.05%) belonged to other schools; 120 of them are still in the course, and the rest dropped out (n = 17; 12.4%); demographic data are shown in *Table 1*.

Our undergraduate group began using the evaluation system from the eighth course onwards; we observed that performance differed between generations. For example, we describe the results between two courses, each being group A and group B shown in *Table 2*; the overall average of the three modules was 7.9 in group A and 8.2 in group B. The so-called stations referred to, in number one, laparotomy plus biopsies. The so-called stations referred, at number one, to laparotomy plus liver biopsy, station two, nephrectomy, and station three, splenectomy. The evaluation was structured as follows: at each station, there was a knowledge stage, another stage

Table 1: Demographic results of undergraduate students in 11 years of an experimental surgery course at the Universidad Cuauhtémoc San Luis Potosí, 2009-2019.

	n	%
Subgroup developed by students		
Total number of students	149	100.00
Female	75	50.34
Male	74	49.66
Data of students who graduated from the course		
Concluded medicine	109	100.00
Students nominated as instructors	22	20.18
Students nominated as instructors' coordinators	4	3.67
Students with research projects presented at conferences	15	13.76
Instructors with an award at a scientific event	3	2.75

Table 2: Module ratings of the skill stations according to the Anaya-Serrano-Gámez model of undergraduate students in 11 years of an experimental surgery course at the Universidad Cuauhtémoc, San Luis Potosí, 2009-2019.

	Qualifications	
	Group A	Group B
Knowledge test		
Station one	8.5	8.4
Station two	6.8	8.6
Station three	9.6	8.2
Average	8.3	8.4
Examination of equipment and instruments		
Station one	7.2	7.7
Station two	6.7	8.5
Station three	9.5	7.9
Average	7.8	8.0
Skills test		
Station one	8.2	7.6
Station two	5.6	8.5
Station three	9.0	8.2
Average	7.6	8.1
Course average	7.9	8.2

to evaluate the knowledge of the material, and, at the end, the skills stage. In this way, we have the result of the student's performance in its theoretical aspect, knowledge of the material, and skills in three different procedures, with which an evaluation based on evidence was made and not on an affective-qualitative scale, but on an impartial quantitative scale, thanks to which it was possible to give feedback to the student in the areas where he could improve such skills or knowledge and, of course, also to the teacher himself. This approach helped us to propose the most outstanding students as instructors based on these averages. This way, emotional conflicts were avoided and helped support each student's final grade in the different courses.

As an unexpected fact, our study found that of the 120 students who completed the course, 15 (12.5%) have presented research projects in various forums, three of them won a place in a congress competition, and one of them has already published his work. In 2022 a specific manual for the course was published.

DISCUSSION

The initial approach we intended in the first four courses was to provide elements to undergraduate students to develop skills and bring them closer to abilities, which, although they are found in their undergraduate surgical area subjects, without intending to form a pre-resident,[1] we found that these skills allowed us to develop a dynamic model, maintaining the ethical principles regarding minor species[28] proposed by Sun Lee through the courses for postgraduates that are developed in our country.[14,16-18] Applying them to the undergraduate allowed us, from the eighth course of 2014, to implement a constructivist system, which obtained a quantifiable result, where the student's performance was evaluated, no longer in a practical way, but also more fairly and objectively allowed us to correct the defects of the three items to be perfected in the student,[22,23,30] eliminating the skinner's effect of only issuing a grade,

improving that intended scaffolding that in the future will allow the student to develop other skills. An incredible achievement was the teacher training of 22 instructors and the inclusion of 15 as research prospects by motivating them to present papers in congresses and a published article;[30] these last two, although they were not expected products of the course, were taken as a medium-term benefit of the constructivist actions. The achievement was not only to create a surgical scaffolding but also to develop another complementary scaffolding that would attract the student to surgical research, a fundamental piece in the indispensable innovation in our area.[5] We can say that we leave as a proposal for further studies that constructivism surpasses behaviorism not only by providing a single objective but also by developing more than two achievements in students who are prepared with this system in our experience (development of surgical skills and development of research skills).

Although Evans proposes that actual sessions should be reserved for complex procedures,[31] our experience in subjecting students to real situations was an approach that even motivated an unexpected by-product, such as the production of completed research papers. This motivation is described in other groups that manage the so-called "surgery clubs".[15,19,32,33] Although it is challenging to compare quantitative achievements with other Latin American groups, what is certain is that the success of all of us is to bring undergraduate students closer to actual surgical exercises, our variable being a constructivist system.

Programs and proposals with the help of simulators should be included to increase this skills[34] in different scenarios.[29,31] It is interesting to note that the construction of pre-delineated elements in surgical teaching provides opportunities to develop, in turn, new by-products such as the Moulton study, where from that scaffolding[6] elements were provided to students to respond to unforeseen surgical situations. The definition of these teaching model schemes is still pending.

CONCLUSIONS

It is also pending to develop programs that give congruence in the undergraduate student, not only to train as a clinician in our area (we do not intend to make specialists from this level), but to encourage the inclination towards surgical research and cultivate curiosity at the academic level, awakening their skills,[9] being personally a necessary combination of all of them.

As an unexpected aspect in our description, it was observed that students, in addition to the constructivist exercise of delivering a product, their simple qualification or the performance of their surgical practice, there was an additional product which was their intervention in research projects in 10% of those enrolled, which we consider that an additional achievement of the course was that students joined research projects with personal achievements of presentation at congresses, The course was a promoter of research in our school, fulfilling the expectation of how to bring the millennial generation, of cybernetic complexity,[35] to an actual practice with the development of intellectual skills in research and surgery, achieving a scaffolding that allows acquiring new elements in their personal development.

What is clear to us in terms of experience is to implement, at low cost and high impact, a constructivist system such as that of Anaya-Serrano-Gámez, not only in the development of surgical skills but also to motivate the student's natural approach to research, both tangible by-products (research papers, congress posters, and publications).

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Ethical considerations: concerning the live models (Wistar rats), the indications of NOM-062-ZOO-1999 were followed in order not to mistreat this species, prevent its suffering, and respect its integrity as a living being; in the case of survival to the practices, the students took charge of these animals, committing themselves

to take care of them as pets. In the case of the students, only the general productivity data were taken without requiring informed consent for each of them, considering not to mention the names but the general percentage data of each generation. The research committee with registration CEI-HGS-015-17 authorized the teaching studies.

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Type I and II sphincter of Oddi dysfunction: a case-control study

Disfunción del esfínter de Oddi tipo I y II: estudio de casos y controles

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Keywords:
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Palabras clave:
coledocolitiasis, disfunción del esfínter de Oddi, discinesia, estenosis.

ABSTRACT

Introduction: the sphincter of Oddi is a valvular complex that regulates bile flow and pancreatic secretion. The sphincter of Oddi dysfunction is divided into stenosis (type I) or dyskinesia (type II). This study aims to describe this pathology's scenario, compare it with cases of choledocholithiasis, and demonstrate if there are differences or similarities. **Material and methods:** a case-control study was performed where patients sent to gastrointestinal endoscopy with a diagnosis of benign biliary tract obstruction were analyzed between the period from January 2019 to December 2021. **Results:** there was no statistically significant difference between the characteristics of patients with sphincter of Oddi dysfunction and proven choledocholithiasis. Verifying the statistic revealed differences in cannulation strategies or post-endoscopic retrograde cholangiography pancreatitis was also impossible. **Conclusions:** type I and type II sphincter of Oddi dysfunction should be considered as the same entity and treated with the same therapy (endoscopic retrograde cholangiopancreatography with sphincterotomy). Choledocholithiasis and sphincter of Oddi dysfunction behave as similar pathological spectra, since the clinical features involved do not show relevant statistical differences.

RESUMEN

Introducción: *el esfínter de Oddi es un complejo valvular que regulariza el flujo biliar y la secreción pancreática. La disfunción del esfínter de Oddi se divide en estenosis (tipo I) o discinesia (tipo II). El objetivo de este estudio es describir el escenario de esta patología, hacer una comparativa con casos de coledocolitiasis y demostrar si existen diferencias o similitudes.* ***Material y métodos:*** *se realizó un estudio de casos y controles donde se analizaron pacientes enviadas a endoscopia gastrointestinal con diagnóstico de obstrucción benigna de la vía biliar entre el periodo de enero de 2019 a diciembre de 2021.* ***Resultados:*** *entre las características de las pacientes con disfunción del esfínter de Oddi y coledocolitiasis comprobada no hubo diferencia estadísticamente significativa. Tampoco fue posible verificar diferencias estadísticamente reveladoras en las estrategias de canulación ni en la pancreatitis postcolangiografía retrógrada endoscópica.* ***Conclusiones:*** *la disfunción del esfínter de Oddi tipo I y II deberá considerarse como una misma entidad, tratarse con una misma terapéutica (colangiopancreatografía retrógrada endoscópica con esfinterotomía). La coledocolitiasis y la disfunción del esfínter de Oddi se comportan como espectros patológicos similares, ya que las características clínicas implicadas no muestran diferencias estadísticas relevantes.*

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INTRODUCTION

The sphincter of Oddi is a valvular complex composed of smooth muscle that regulates bile flow and pancreatic secretion into the duodenal lumen. It has a resting pressure of 15 mmHg. It comprises a biliary sphincter and a pancreatic sphincter joined at their distal portion to form the ampullary sphincter at the level of the second duodenal portion.[1]

Sphincter of Oddi dysfunction (SOD) is a diagnosis of exclusion and encompasses a variety of disorders that result in inappropriate function (stenosis or dyskinesia) of this valve.[2] This dysfunction is associated with abdominal pain (although

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a non-painful variant exists),[3] elevation of liver and pancreatic enzymes, bile duct and pancreatic duct dilatation, and may also be associated with pancreatitis.[4] Although both genders can be affected, it is more common in women aged 20-50 years.[5] The prevalence of this disease in patients with biliary pain after cholecystectomy is 14%.[6]

In both variants of SOD (stenotic and dyskinetic), it is suggested that its etiology is similar, and trauma is necessary (probably from a litho smaller than 5 mm), which, when passing through the sphincter of Oddi, causes inflammation and the consequent formation of a fibrotic ring by scarring (in half of the cases) leading to the SOD syndrome.[4]

This entity has been controversial since its first description, initially stratified according to the Milwaukee classification *(Table 1)* and later modified by Rome IV scale *(Table 2)*. This study aims to describe the scenario in a second-level center facing this pathology, compare it with cases of choledocholithiasis, and demonstrate if there are differences or similarities.

MATERIAL AND METHODS

A case-control study was conducted where female patients referred to the gastrointestinal endoscopy service diagnosed with benign bile duct obstruction (diagnosis of choledocholithiasis referral) were studied from January 2019 to December 2021. Patients were selected for the case group who met the criteria for SOD: biliary pain, altered LFTs (liver function tests) and bile duct dilatation, absence of choledocholithiasis and structural alteration in the bile duct (Rome IV criteria), who had a history of uncomplicated cholecystectomy (open or laparoscopic). As

Table 1: Milwaukee classification for sphincter of Oddi dysfunction.

	Type I	Type II	Type III
Biliary pain	+	+	+
Altered LFTs	+	±*	–
Bile duct dilatation	+	±*	–
Delayed biliary drainage	+	±*	–

LFTs = liver function tests with alanine aminotransferase (ALT) and aspartate aminotransferase (AST) twice the average on two or more occasions.
Bile duct dilatation ≥ 12 mm on ultrasound or > 10 mm on cholangiography.
Delayed biliary drainage = drainage of contrast medium delayed for > 45 minutes after ERCP.
* One or two positive factors.

Tabla 2: Criterios diagnósticos para disfunción de esfínter de Oddi de tipo biliar según Roma IV.

Must include:
1. Biliary-type pain
2. Elevated liver enzymes or dilated bile duct (> 6 mm)
3. Absence of choledocholithiasis or other structural alterations of the biliary tract

Support criteria:
1. Normal amylase/lipase
2. Abnormal sphincter of Oddi manometry
3. Abnormal hepatobiliary scintigraphy

Suspected sphincter of Oddi dysfunction: biliary type pain + at least one associated objective finding.
Episodic functional abdominal pain: biliary-type pain without any other alteration.

support criteria, patients with amylase/lipase within normal parameters at the time of the study were included. For the control group, 60 female patients with endoscopic retrograde cholangiopancreatography (ERCP)-confirmed diagnoses of choledocholithiasis whose resolution occurred at the endoscopic event were selected. In both groups, an inclusion criterion was that they had not undergone previous ERCP.

The following were evaluated: age, weight, height, body mass index (BMI), age over 55 years, previous pancreatitis, bilirubin and their differential, alanine aminotransferase, aspartate aminotransferase, extrahepatic bile duct (EHBD) size, difficult cannulation, cannulation attempts, precutting, and post-ERCP pancreatitis.

Two groups were pooled, the group with SOD versus choledocholithiasis, and the established variables were analyzed. Values were expressed as absolute values and percentages for categorical variables. They were compared with the χ^2 test or Fisher's exact test. In contrast, quantitative variables are expressed as averages, ± standard deviation, and were compared with Student's t-test (for variables of normal behavior) or Mann-Whitney U test (for non-normal behavior variables). A value of less than 0.05 was considered statistically significant. The analyses were performed with SPSS Statistics, Version 25.0 (Armonk, NY: IBM Corp).

RESULTS

Twenty-two cases with a diagnosis of SOD were studied, and 60 control patients with choledocholithiasis were included. Patients who met the criteria for suspected DEO were referred with a diagnosis of suspected choledocholithiasis. Four patients (18.2%) had a previous diagnosis of pancreatitis. The papilla of native characteristics was found in all 22 patients. Cholangiography evidenced increased caliber without filling defects in all patients; a pencil-point termination and adequate drainage of contrast medium were observed after sphincterotomy. Ten patients (45.4%) were cannulated with difficulty criteria with an average of 3.8 attempts. Precut papillotomy was used to cannulate in four patients (18.2%), and in all 22 patients (100%), cholangiography and sphincterotomy were performed as treatment of the presumed diagnosis of SOD. Extrahepatic bile duct sweeping was performed in 17 patients (77.3%) as part of bile duct securing. Three patients (13.6%) had post-ERCP pancreatitis as a complication (in one of these patients was severe), but there was no mortality.

In the characteristics of patients with SOD and proven choledocholithiasis, there was no statistically significant difference in any of the morphological or laboratory variables *(Table 3)*.

Nor could it prove statistically significant differences in cannulation strategies or post-ERCP pancreatitis *(Table 4)*.

DISCUSSION

SOD is a broad functional disorder involving a valve with inappropriate spasm or relaxation and stenosis. It has an estimated prevalence of 1.5% in the general population; however, it appears underestimated due to the lack of biochemical markers for its identification. Manometric studies reveal that up to 10% of biliary tract interventions involve the papilla, even with no lithosclerosis lesions.[7] In a study carried out in the *Hospital Juárez de México*, a prevalence (probable diagnosis) of DEO of 16.5% was observed,[8] while in the *Hospital Central Militar*, the prevalence was 18.9% (52 cases in 269 CPREs),[9] while in another study published by our group a prevalence of 20% was reported.[10] In patients with chronic or idiopathic pancreatitis, the prevalence of SOD can reach 59 and 72%, respectively.

In our study, the mean age of patients with SOD was lower than in another study performed in Japan (50.5 versus 62 years); however, our study was performed only in women, whereas in the study mentioned above, women accounted for 69.4% of the participants.[11] In that same study, previous pancreatitis was observed in 22%, whereas in our analysis, the history of previous pancreatitis was 18.2%. Regarding the caliber of the SBV, in the Japanese study, it was 12.2 mm, while in our study, it was 9 mm.

The Milwaukee classification was first used in SOD; however, this classification could lead to confusion, so the Rome IV criteria (which avoid using manometry) are now used to diagnose.[12] According to Rome IV, type I SOD no longer exists and should be classified as benign papillary stenosis (mechanical obstruction), not a functional disorder. In contrast, type III SOD is considered a functional entity that appears to be unrelated to the sphincter of Oddi per se.[3] Thus, type II SOD (according to Rome IV) is currently classified as the true SOD.[13] It will take some time to separate benign papillary stenosis from functional disorders (Rome IV). This diagnosis will be permanently linked to SOD and will probably continue to be referred to as type I SOD (even if manometry is not used to make the diagnosis).

Table 3: Patient features.

	SOD (N = 22)	Choledocholithiasis (N = 60)	p
Age [years]*	50.5 ± 16.4	46.6 ± 16.2	0.3‡
Weight [kg]*	76.2 ± 17.4	76.7 ± 15.9	0.9‡
Height [m]*	1.58 ± 0.8	1.62 ± 0.8	0.08§
BMI [kg/m²]*	29.9 ± 5.05	28.9 ± 5.7	0.5‡
> 55 years, n (%)	9 (40.9)	11 (27.5)	0.3¶
Pancreatitis prior to ERCP, n (%)	4 (18.2)	5 (12.5)	0.7¶
TB [mg/dl]	3.5 ± 1.9	3.6 ± 2.5	0.8‡
DB [mg/dl]	2.2 ± 1.2	2.1 ± 1.6	0.9‡
IB [mg/dl]	1.3 ± 0.7	1.5 ± 1.01	0.4‡
ALT	264 ± 215.5	250.8 ± 192.6	0.8§
AST	228 ± 271	204.1 ± 192.6	0.8§
EBD size [mm]	9 ± 3.7	11.2 ± 5.2	0.6§

SOD = sphincter of Oddi dysfunction. BMI = body mass index. ERCP = endoscopic retrograde cholangiopancreatography. TB = total bilirubin. DB = direct bilirubin. IB = indirect bilirubin. ALT = alanine aminotransferase. AST = aspartate aminotransferase. EBD = extrahepatic bile duct.
* Data are mean ± standard deviation. ‡ Student's t-test. § Mann-Whitney U. ¶ Pearson's χ^2.
Source: IMSS electronic file HGZ No. 35.

Table 4: Sphincter of Oddi cannulation.

Variable	SOD (N = 22) n (%)	Choledocholithiasis (N = 40) n (%)	p
Cannulation (difficult)	10 (45.5)	14 (35)	0.4‡
Cannulation attempts*	3.8 ± 2.5	2.9 ± 2.01	0.18§
Precut	4 (18.2)	12 (30)	0.35¶
Post-ERCP pancreatitis	3 (13.6)	5 (12.5)	0.6¶

* Data are mean ± standard deviation. ‡ Pearson's χ^2. § Mann-Whitney U test. ¶ Fisher's exact test.
SOD = sphincter of Oddi dysfunction.
Source: IMSS electronic file HGZ No. 35.

It is questionable whether SOD is a primary pathologic process or a consequence of a traumatic alteration of the sphincter of Oddi. The surgical history suggests that it is the second option, so a patient with recurrent symptoms after cholecystectomy (due to cholelithiasis) may be a case of secondary benign papillary stenosis or type I SOD. Differentiating the purely dysfunctional process (dyskinesia) from the stenotic process is very complex. Since the treatment is similar, it can be stated that patients with a history of cholecystectomy could suffer from stenosis-type dysfunction. In contrast, those without a history of cholecystectomy and evidence of gallbladder or common bile duct lithiasis could be considered fully functional (dyskinesia).

Diagnosis is complex, and overlooking it leads to complications such as recurrent biliary symptoms, elevated liver enzymes, and even pancreatitis.[14] There will be controversy regarding the pain of SOD because the characteristic is biliary type, which is related to food (there are authors who refer that pain in SOD is not related to food), usually lasting from 30 minutes to a few hours and resolves spontaneously. The diagnostic suspicion starts with the pain clinic and laboratories, including bilirubin, liver enzymes, amylase, and lipase. Alkaline phosphatase may provide a clue for diagnosis without increased bilirubin or pancreatic enzymes.[7] It is imperative to rule out the presence of choledocholithiasis or other biliopancreatic or ampulla of Vater alterations, which could condition the picture.[6]

Differentiating it from choledocholithiasis was not possible in this study; there were no characteristics with statistically significant differences, so in our environment, ERCP has a current role in the diagnosis and treatment of this entity, even without a previous diagnostic suspicion.

The gold standard for diagnosis is the sphincter of Oddi manometry, whose pressure > 40 mmHg (three standard deviations above average) makes the diagnosis. Patients with benign papillary stenosis (SOD type I) may have normal manometry up to 15-35%,[15] while patients with dyskinesia dysfunction (SOD type II) may have normal manometry up to 45%.[15] This suggests that the pure increase in the sphincter of Oddi pressure is insufficient to cause the disease's symptoms.[15]

ERCP with sphincterotomy is the management in patients with type I (stricture) and type II (dyskinesia) SOD with a short-term success rate greater than 90%;[11] while other series report a success rate of 60 to 94% in patients whose diagnosis was not based on manometry.[16] The recurrence rate after sphincterotomy treatment is 32% within six months; however, this recurrence is related to the presence and development of functional dyspepsia.[11]

The rates of post-ERCP pancreatitis in SOD range from 0 to 30% (if ERCP is accompanied by manometry), so performing manometry seems to be a risk factor for this complication.[2] In this study, the rate of post-ERCP pancreatitis was 13.6% lower compared to a Japanese study, where the rate of post-ERCP pancreatitis was 36%;[11] this is a very high rate even for those patients undergoing sphincterotomy. Mortality from adverse events after ERCP is 0.08%.[17] In our study, all cases underwent sphincterotomy as treatment with a rate of post-ERCP pancreatitis acceptably like controls with choledocholithiasis (13.6 versus 12.5%, p = 0.6). ERCP with sphincterotomy is indicated in patients with SOD who meet the criteria of biliary-type pain, altered liver function tests, and bile duct dilatation[4] with a greater than 90% success rate in patients.[18]

It is recommended that during ERCP for type I and type II SOD, indomethacin 100 mg or diclofenac 75 mg rectally before or after the procedure are administered,[19] and place a pancreatic stent 5 Fr and 4 cm in case of unintentional cannulation of the pancreatic duct,[16] and that epinephrine be sprayed on the papilla after the procedure.[17]

In our region (Ciudad Juarez), there is no access to the sphincter of Oddi manometry studies, and this scenario is constant in most of the country. Moreover, this procedure is less and less used due to the added risk of pancreatitis that it entails. Other study methods include scintigraphy with a lower sensitivity than manometry (which shows

delayed emptying).[20] In public and second-level medical centers, there is also no access to imaging studies of the biliary tract, such as magnetic resonance cholangiography, so ERCP is still valid as a diagnostic study? The diagnosis of SOD in most hospitals is based on the Rome IV criteria or post-ERCP findings (in post cholecystectomy patients). It is stated that 10% of patients may have a complete diagnostic workup, and this percentage needs to be revised.[19] In these cases, how prudent is treating them even without complementary studies? In all the cases in this study, the diagnosis was made post-ERCP, and to make this diagnosis, the presence of choledocholithiasis or an ampullary tumor had to be excluded.[4]

This study has weaknesses: it is a retrospective study in a single hospital center, the lack of follow-up of patients to observe the resolution of their symptoms, and the long-term response rate after sphincterotomy.

CONCLUSIONS

Both type I (stenosis) and type II (dyskinesia) SOD should be considered as the same entity that is treated with the same therapy (ERCP with sphincterotomy). Type III SOD will be reassigned to a functional entity in its entirety, the treatment of which will be purely medical. Ideal medicine is far from our reality, and international guidelines only sometimes fit the national scenario. Considering and treating type I and type II dysfunction with the only thing we have (sphincterotomy) may be risky, but it is still justified. Choledocholithiasis and SOD show that their clinical characteristics involved do not present relevant statistical differences.

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Cholecystectomy in the bariatric patient: before, during, or after gastric bypass?

Colecistectomía en el paciente bariátrico: ¿antes, durante o después de bypass gástrico?

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Keywords:
obesity, cholelithiasis, cholecystectomy, bariatric surgery.

Palabras clave:
obesidad, colelitiasis, colecistectomía, cirugía bariátrica.

ABSTRACT

Vesicular lithiasis is a frequent pathology and involves significant costs to health systems; moreover, it is the primary gastroenterological diagnosis in hospitalized patients. The incidence of cholelithiasis is one of the leading health problems affecting the adult population in Mexico and the world. Among the risk factors for biliary pathology are female sex, age, overweight and obesity, and rapid weight loss. Many of these factors are present in patients undergoing bariatric surgery. Some studies report up to 20% of cholelithiasis or a cholecystectomy history in patients who will undergo bariatric surgery. Patients undergoing bariatric surgery have a low incidence rate of biliary complications, and cholecystectomy at the same surgical time increases the risk of postoperative complications and operative time. If cholecystectomy is not indicated, patients should be followed carefully, with particular attention to the development of biliary complications. In this review, we searched the different times when laparoscopic cholecystectomy can be performed in a bariatric patient.

RESUMEN

La litiasis vesicular es una patología muy frecuente e implica grandes costos a los sistemas de salud; además, es el principal diagnóstico gastroenterológico en los pacientes hospitalizados. La incidencia de colelitiasis es uno de los problemas principales de salud que aquejan a la población adulta en México y el mundo. Dentro de los factores de riesgo de la patología biliar se encuentran el sexo femenino, la edad, el sobrepeso y la obesidad, así como la pérdida rápida de peso. Muchos de estos factores se presentan en pacientes sometidos a cirugía bariátrica. Algunos trabajos reportan hasta 20% de colelitiasis o antecedentes de colecistectomía en sus pacientes que serán sometidos a cirugía bariátrica. Los pacientes sometidos a cirugía bariátrica tienen una tasa de incidencia baja de complicaciones biliares y la colecistectomía en el mismo tiempo quirúrgico aumenta el riesgo de complicaciones postoperatorias y el tiempo operatorio. Si la colecistectomía no está indicada, se debe hacer un seguimiento cuidadoso de los pacientes con atención especial a desarrollo de complicaciones biliares. En esta revisión efectuamos una búsqueda en los distintos tiempos en los que se puede realizar la colecistectomía laparoscópica en el paciente bariátrico.

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INTRODUCTION

Vesicular lithiasis is a frequent pathology and involves great costs to health systems; it is also the primary gastroenterological diagnosis in hospitalized patients.[1] The incidence of cholelithiasis is one of the leading health problems affecting the adult population in Mexico and the world. In the United States, 10-15% of the adult population is estimated to suffer from cholelithiasis, and approximately 800,000 new cases are diagnosed yearly.[2] The prevalence of biliary pathology in morbid obesity has not

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been well-studied. Some studies report up to 20% of cholelithiasis or a history of cholecystectomy in patients who will undergo bariatric surgery.[3]

Because of bariatric surgery, the decrease in body weight is also considered a risk factor for the development of cholelithiasis. Different epidemiological studies in different populations have demonstrated this effect;[4,5] a decrease between 4 and 10 kg in two years is associated with a 44% increase in the risk of developing cholelithiasis compared to those who only decreased less than 4 kg, even in those whose decrease was more significant than 10 kg the risk increased by 94%.[6]

Follow-up studies have shown that body mass index (BMI) is associated with the likelihood of having undergone cholecystectomy, particularly among women with a BMI > 30, in whom the likelihood is increased up to threefold.[7]

There is still controversy about whether cholecystectomy should be performed prophylactically at the same surgical time as Y-de-Roux gastric bypass (YRGB). Some have advocated a selective approach using intraoperative ultrasound and selective cholecystectomy followed by prophylactic ursodeoxycholic acid, but compliance with ursodiol was only 41%.[8] Laparoscopic cholecystectomy in obese patients usually has a similar conversion rate, complication rate, and length of stay as in non-obese patients. It can be performed after YRGB surgery if clinically indicated.[9] It appears that laparoscopic YRGB combined with cholecystectomy is safe and feasible without altering port placement; however, it increases operative time and hospital stay.[10]

ACUTE CHOLECYSTITIS AND OBESITY

Acute cholecystitis in 90-95% of cases corresponds to a complication of cholelithiasis, usually due to cystic duct obstruction, distension and inflammation, and secondary bacterial infection. The clinical presentation is characterized by abdominal pain in the right upper quadrant, hypersensitivity, and resistance to palpation, classically described as Murphy's sign. Diagnosis is based on clinical, laboratory findings, and imaging studies.

Acute cholecystitis can result in significant morbidity and mortality, especially in older and immunocompromised adults, so the treatment of choice is laparoscopic cholecystectomy. Regarding the timing of this surgery, it is currently recommended to perform it in the same episode of cholecystitis (or up to seven to 10 days) to avoid progression to complications such as empyema, necrosis, cholangitis, and sepsis.[11]

We know that obesity is associated with an elevated risk of cholelithiasis, and several studies have shown a correlation between body mass index (BMI) and the risk of cholelithiasis.[12]

In women, a strong positive correlation exists between body mass index and the relative risk of gallstones. In one study, the age-adjusted relative risk in mildly overweight women was 1.7 compared to normal-weight individuals and increased to 6.0 in markedly overweight women.[13]

The association of gallstones with obesity in men has been more challenging to demonstrate. Some studies indicated that body mass index was not different in men with or without gallstones, regardless of age.[14,15] However, in extensive epidemiological studies, men and women observed a positive association between body mass index and gallstones.

Gallstone disease has been associated with central adiposity with a positive correlation between waist-to-hip ratio and observed in obese individuals; this often appears symptomatic.[16]

Most gallstones in obese individuals are cholesterol stones. Three main factors have been implicated in forming cholesterol stones: supersaturation of bile with cholesterol, a nucleation defect, and impaired gallbladder motility.[17]

Morbid obesity is often considered a risk factor for laparoscopic cholecystectomy due to increased operative time morbidity and conversion rate to open cholecystectomy. Laparoscopic cholecystectomy (LC) in obese

patients can be a generally more demanding technique, as illustrated by the experience of Angrisani et al.[18] and Nies and his team,[19] who reported significantly longer operative times. And to the significantly higher conversion rates (4.5% versus 1.8%) in the series published by Champault et al.[20] There may be difficulties with exposure to Calot's triangle due to a fatty omentum, bulky transverse colon and enlarged fatty liver.[21]

However, it has recently been proven that laparoscopic cholecystectomy can be safely performed in obese and overweight patients with increased operative time, complications such as surgical site injury, or risk of conversion. If adequate equipment and skilled surgeons are available, symptomatic patients should be offered the benefits of laparoscopic cholecystectomy regardless of body mass.

Laparoscopic cholecystectomy and gastric bypass at the same operating time

Rapid weight loss after bariatric surgery is one of many known risk factors for gallstone development, along with age, female gender, parity, race, obesity, genetics, very low-calorie diets, short bowel syndrome, gallbladder motor dysfunction, diabetes, medications, and gastrointestinal surgery, among many others.[22]

Sustained weight loss after gastric bypass is achieved by a combination of gastric restriction and a variable degree of mismanagement and therefore has a higher risk of gallstone development than purely restrictive procedures such as adjustable gastric banding. The appropriate management of gallstones and gallbladder disease in these patients is still under debate. Several therapeutic modalities are used, including simultaneous cholecystectomy to all patients at the time of gastric bypass regardless of the presence or absence of gallstones and symptoms (prophylactic approach), simultaneous cholecystectomy only to patients with gallstones (elective or selective approach), and expectant management with or without prophylactic administration of ursodeoxycholic acid.[23-25]

Prophylactic approach

It consists of performing simultaneous cholecystectomy on all patients during gastric bypass, regardless of the presence or absence of gallstones or symptoms.

The rationale for this approach is based on the high incidence of gallstone development after gastric bypass secondary to rapid weight loss and the low sensitivity and specificity of ultrasound for detecting gallstones in morbidly obese patients.

Guadalajara et al. performed simultaneous cholecystectomy in 89 patients undergoing open gastric bypass and observed a postoperative incidence of gallstones of 24%, while the incidence of preoperative ultrasound was only 16%.[26]

Cholecystectomy during laparoscopic YRGB has been extensively studied, but the results are inconclusive. Many reports have shown that the combined procedure can be performed safely but is not complication-free. Additional ports may be required, increased operative time and length of hospital stay, and specific adverse events such as wound infections, gastrointestinal leakage, pneumonia, and renal failure have been reported.[10,27]

A large study using the ACS NSQIP database from 2005 to 2009 showed that cholecystectomy at the same surgical time increased the risk of adverse events in laparoscopy but not in open YRGB. That same study suggested that it should be reserved for patients with previously symptomatic disease because access to the biliary system after YRGB would not be possible by endoscopic retrograde cholangiopancreatography (ERCP).[28]

Expectant management

Several studies have suggested that the prophylactic use of ursodeoxycholic acid may prevent gallstone formation after different bariatric procedures.

Sugerman et al.[29] concluded that a daily dose of 600 mg of ursodiol is an effective prophylactic agent for gallstone formation following gastric bypass. However,

compliance and dosage of the medication are variable, and data on the effect of ursodeoxycholic acid on symptomatic gallstones requiring cholecystectomy are limited. One study with cost-effectiveness analysis reported that the additional cost of prescribing ursodiol does not justify its use after YRGB.[30]

More extensive controlled studies are needed to establish the effective use of ursodeoxycholic acid in the bariatric patient.

Laparoscopic cholecystectomy following gastric bypass

The incidence of cholecystectomy is higher during the first six months after bariatric surgery but is generally low (4%). Gastric bypass patients are more likely to require cholecystectomy than band and sleeve patients.[31] An estimate of the standardized incidence rate (SIR) for cholecystectomy for the period 1987-2008 in Sweden, based on data from the National Patient Registry, showed a 10-fold peak in the SIR for cholecystectomy during the first seven to 24 months after bariatric surgery.[32,33]

The increased risk of cholesterol gallstones after bariatric surgery involves several determinants, including altered biliary cholesterol homeostasis during weight loss. Also, gallbladder hypomobility, increased pro-nucleating factors, and altered intestinal motility appear to contribute to cholesterol gallstone formation.[34]

Preoperative use of statins is a protective factor for cholecystectomy. In theory, statins may reduce the risk of gallstones by lowering hepatic cholesterol levels, but the effect has yet to be consistently demonstrated in the literature.[35] If the association between the use of higher doses of statins and gallstone formation is confirmed, the effect of prescribing statins in moderate or high doses on gallstone formation, biliary symptoms, and cholecystectomy should be investigated.

One study showed that patients who undergo cholecystectomy after YRGB rather than before experience twice as many complications.[36] A multivariate analysis determined a higher risk of perioperative and aggregate postoperative complications at 30 days when cholecystectomy is performed after YRGB compared with before. There was a higher risk of reoperation when cholecystectomy was performed after YRGB compared with before YRGB ($p = 0.034$) when acute cholecystitis was the indication for cholecystectomy.

In addition, complications experienced after the first procedure independently increased the risk of complications in the subsequent procedure ($p < 0.001$); and 61.7 minutes ($p < 0.001$) was added to the surgical time.[36]

CONCLUSION

Symptomatic gallstone disease and cholecystectomy risk increase in the first years after bariatric surgery. Different strategies have been used to reduce the risk of symptomatic gallstones, including postoperative pharmacological treatment with ursodeoxycholic acid and concomitant cholecystectomy in patients with ultrasound-verified cholelithiasis with or without symptoms.

Therefore, how to address this potential complication is an ongoing discussion in the bariatric community.

The patient and surgeon should have an open and shared decision-making conversation to discuss together the risks and benefits of the timing of bariatric and gallbladder surgery. These decisions should be individualized, considering the patient's symptoms, anatomy, and other risks of surgery.

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Compartment syndrome in extremities

Síndrome compartimental en extremidades

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Keywords: syndrome, compartment, extremities, fasciotomy, trauma, burn.

Palabras clave: *síndrome, compartimental, extremidades, fasciotomía, traumatismo, quemadura.*

ABSTRACT

An analysis of the manuscripts published in the scientific literature on compartment syndrome was carried out to evaluate a patient with compartment syndrome and identify the clinical presentation and the bases necessary to establish the diagnosis and timely treatment. There is little recent literature less than five years old describing the characteristics of compartment syndrome, which can be caused by multiple etiologies, of which trauma is the most frequent, in addition to burns, fractures, contusions, coagulopathies, postischemic edema or very tight casts. A review of selected studies was carried out, among which those with the highest academic content were selected. Each selected study was analyzed, and data that could be included in each subtopic of the review article were identified. Acute compartment syndrome corresponds to a surgical emergency in that, in the case of not establishing an early intervention, the probability of irreversible sequelae increases notably. In compartment syndrome, there is an increase of interstitial pressure in the body compartments that compromises the adequate perfusion of the soft tissues, derived from an ischemic process that presents as clinical manifestations of sudden intense pain, pallor, absence of pulses, paresthesia, and paralysis, which accompanied by a directed anamnesis integrates the diagnosis with the physical examination. The decompression of the affected muscle space is a surgical emergency, requiring timely detection and early treatment to avoid sequelae and serious complications.

RESUMEN

Se llevó a cabo un análisis de los manuscritos publicados en la literatura científica sobre el síndrome compartimental para la evaluación de un paciente con síndrome compartimental e identificación de la presentación clínica y las bases necesarias para instaurar el diagnóstico y tratamiento oportuno. Es escasa la bibliografía reciente menor de cinco años que describa las características del síndrome compartimental, el cual puede ser provocado por múltiples etiologías, de las cuales los traumatismos son la más frecuente, además de las quemaduras, fracturas, contusiones, coagulopatías, edema postisquémico o yesos muy apretados. Se realizó una revisión de estudios elegidos, entre los cuales fueron seleccionados aquellos con mayor contenido académico. Se analizaron cada uno de los estudios seleccionados y se identificaron los datos que podrían incluirse en cada uno de los subtemas del artículo de revisión. El síndrome compartimental agudo corresponde a una emergencia quirúrgica que, en caso de no establecer una intervención precoz, la probabilidad de secuelas irreversibles se incrementa notablemente. En el síndrome compartimental existe aumento de la presión intersticial en los compartimentos del cuerpo que compromete la adecuada perfusión de los tejidos blandos, derivado de un proceso isquémico que presenta como manifestaciones clínicas dolor súbito intenso, palidez, ausencia de pulsos, parestesia y parálisis, lo cual acompañado de una anamnesis dirigida integra el diagnóstico junto al examen físico. Corresponde a una urgencia quirúrgica la descompresión del espacio muscular afectado, por lo que requiere detección oportuna y tratamiento precoz para evitar secuelas y complicaciones graves.

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INTRODUCTION

The compartment syndrome (CS) is defined as a surgical emergency characterized by an elevation of interstitial pressure within a closed osteomyofascial compartment above capillary perfusion pressure with compromised blood flow in muscle and nerve that conditions tissue damage triggering an ischemic process that jeopardizes viability and reversibility.[1-3] In acute compartment syndrome (ACS), intra-compartmental pressure (ICP) > 30 mmHg is described as a valuable threshold to aid in diagnosis; however, it is considered a clinical diagnosis, so a single regular ICP reading does not exclude acute compartment syndrome.[3]

Historically, one of the first descriptions of CS and its complications in the literature was attributed to skeletal muscle ischemia, made by Richard von Volkmann, a 19th-century surgeon and considered one of the fathers of orthopedic surgery in 1881, of ischemic contracture of muscle tissue due to constriction, often by bandages.[4] Subsequently, surgical management by fasciotomy for battlefield injuries was put into practice as early as World War II; however, it is Norman Rich who is credited with emphasizing the liberal and timely use of fasciotomy as a treatment for post-trauma CS.[5]

When excessively increased pressure within a muscle compartment limits adequate capillary perfusion to soft tissues, nerves, and muscles, it compromises viability and leads to potential damage 8 hours after the onset of ischemia; such tissue damage secondary to prolonged ischemia will be irreversible.[6]

The human body has approximately 46 spaces containing muscle, nerves, and blood vessels. Of these, nine are located in the trunk and the remaining 37 in the extremities in confined spaces with nerves and vessels that innervate and irrigate the muscle tissue covered by a membrane of aponeurotic connective tissue called fascia, with the function of insulation, support, and protection.[7,8]

Pathophysiologically, several theories have been proposed where the common feature of all CS, regardless of etiology or anatomical location, is an increase in intra-compartmental pressure (ICP) within a rigid fascial envelope that alters tissue perfusion,[1] since at the capillary level, the external pressure at all points of the compartment decreases blood flow with an elevation of postcapillary venular pressure and increases hydrostatic pressure with fluid extravasation, edema, nervous and muscular ischemia, leading to necrosis of the structures involved.[9]

One way of classifying CS includes acute compartment syndrome: symptoms compatible with increased compartment pressure that does not resolve independently. Subacute compartment syndrome: does not present with the characteristic symptoms of acute compartment syndrome but still leads to the typical sequelae (ischemic Volkmann's contracture). Recurrent or chronic compartment syndrome: occurs in athletes during physical activity, with muscle weakness and pain where the patient is intermittently asymptomatic, without long-term alterations, and the intra-compartmental pressure at rest is usually elevated.[10,11] During exercise, there is deficient tissue oxygenation secondary to decreased venous return and insufficient muscle tissue perfusion, with increased compartment pressure, accompanied by pain and temporary neurological deficit.[12,13]

The fourth type, crush syndrome, is described as an extreme entity of the acute syndrome, with systemic manifestations of muscle injury.[10] Crush syndrome is defined as the systemic alteration resulting from post-traumatic rhabdomyolysis due to crushing forces; it is an alteration that is frequently reported after catastrophic earthquakes worldwide, and it is due to trauma in extremities by direct force or by the highly stressful position maintained for the circulation of an extremity. Its importance lies in the fact that there is a rupture of muscle cells, which releases myoglobin and potassium into the circulation, causing hyperkalemia, severe inflammatory

response, fluid loss to the third space, metabolic acidosis, acute renal failure, and shock.[14]

EPIDEMIOLOGY

Acute compartment syndrome (ACS) most commonly develops soon after significant trauma, mainly when long bone fractures occur; however, it can be seen from non-traumatic causes.[6,15] It may be due to intrinsic factors (e.g., swelling, hemorrhage) or extrinsic or post-injury factors that restrict the ability of the fascial envelope to expand. It is more common in the lower extremity than in the upper extremity. The calf is the most common site affected in the lower extremity, and the forearm is the most common site in the upper extremity. Fasciotomies are less frequently needed in the upper extremities, accounting for approximately 20% of all extremity fasciotomies.[16,17] ACS is most commonly seen in young men < 35 years with the highest incidence, particularly after tibial diaphysis and distal radius fractures.[6,18]

ETIOLOGY

Several studies showed that fractures are the most common cause of ACS, accounting for about 69-75% of cases.[19] Multiple other etiologies can cause ACS; among all, trauma is the most frequent, particularly long bone fractures, where the tibia is most frequently affected, and approximately 1-10% of fractures develop ACS. Fractures of the forearm are the second most affected bones in the upper extremities. Other traumatic causes of CS include penetrating extremity trauma with vascular (arterial, venous) injury, intra-compartmental hemorrhage, contusions, and crush or burn injuries. Non-traumatic causes include prolonged external compression, animal bites and stings, coagulopathies, tight casts, and postischemic edema. All these causes are presented below *(Tables 1 to 3)*.[2,20,21]

Other important traumatic etiologies to describe are crushed and burn injuries. Crush injuries occur when the limb is trapped for a prolonged duration with

Table 1: Etiology of compartment syndrome due to upper or lower extremity involvement.

Causes in upper and lower extremities
Long bone fracture
Acute extremity ischemia with reperfusion
Burn injury
Crush injury
Animal bites and stings
Spontaneous hemorrhage/hematoma
Soft tissue infection
Non-traumatic myositis/myonecrosis/rhabdomyolysis
Systemic inflammatory response syndrome/mass fluid resuscitation
Leading causes in upper extremities
Animal or insect bites and stings
Iatrogenic high-pressure injection (liquid or gas)
Intravenous extravasation injury
Accidental/intentional intra-arterial injection
Birth injury (e.g., neonatal compartment syndrome)
Leading causes in lower extremities
Prolonged immobilization
Snakebite (due to the location of most snakebites)

Table 2: Etiology of compartment syndrome due to extrinsic and intrinsic causes.

Intrinsic causes	Extrinsic causes
They increase the volume inside the compartment	Restrict dilatation of the compartmental envelope
Bleeding:	Tight bandages, splints, or circumferential casts
Trauma (fracture, vascular injury)	Burn eschar
Coagulopathies (hemophilia, post-resuscitation coagulopathy)	Surgical closure of facial defects
Anticoagulant therapy (heparin, coumarins, thrombolytic agents)	
Edema:	Prolonged use of a tourniquet
Ischemia/reperfusion (arterial lesions, embolism, and thrombosis)	Lithotomy position during surgery
Electrical injuries	Military anti-shock pants (MAST)
Venous thrombosis (phlegmasia cerulea *dolens*)	
Exercise or rhabdomyolysis	

Table 3: Traumatic and non-traumatic causes of acute compartment syndrome.

Traumatic causes	Non-traumatic causes
Fractures of long bones	Intravenous fluids: fluid extravasation; massive fluid resuscitation (e.g., severe thermal burns, sepsis, etc.)
Blunt trauma (crush injury/crush syndrome)	Hematologic: ischemia-reperfusion injury, thrombosis, bleeding disorders, vascular disease, spontaneous hemorrhage
Burn injuries	Anticoagulation
Constrictive bandages, splints, or casts	Toxics: animal poisonings and bites; recreational drug injections
Penetrating trauma	Prolonged limb compression (following severe drug or alcohol intoxication; malposition during surgery)
High-pressure injection	Revascularization procedures (limb bypass surgery, embolectomy, thrombolysis)
Injury to vascular structures	Nephrotic syndrome (decreased serum osmolarity)
	Group A streptococcus infections of the muscle; systemic inflammatory response syndrome

increased compartment pressure resulting from external compression that induces CS determined by the state and duration of the crush or, after blunting, direct trauma. Burns can cause ACS by several mechanisms, mainly circumferential full-thickness burns, as the burn eschar may restrict tissue swelling. Other mechanisms include tissue edema (especially thermal and electrical burns) and secretion of chemical mediators that cause systemic inflammatory response syndrome (SIRS).

Any condition that decreases the capacity of a compartment or increases the volume of fluid within a compartment increases intra-compartmental pressure and puts the patient at risk for developing compartment syndrome.[21] Common sites include the leg and forearm;[15,21] however, it can also occur in the foot, thigh, and gluteal region.

ANATOMY OF THE COMPARTMENTS

The vital thing to know about the compartments in compartment syndrome is their relationship to the clinic.

At the thigh level, there are three compartments:

- **Anterior:** the sartorius and quadriceps muscle is found, passing the femoral nerve and the superficial femoral artery, which provide irrigation to this area.
- **Medial:** composed of the pectineus muscle, obturator externus, gracilis, and abductor muscles. Through here runs the obturator nerve that innervates this compartment.
- **Posterior:** in the posterior compartment are the biceps femoris, semimembranosus, and semitendinosus muscles; the nerve structure found here is the sciatic nerve, and the deep femoral artery is also found; all of them provide both innervation and irrigation to the compartment.

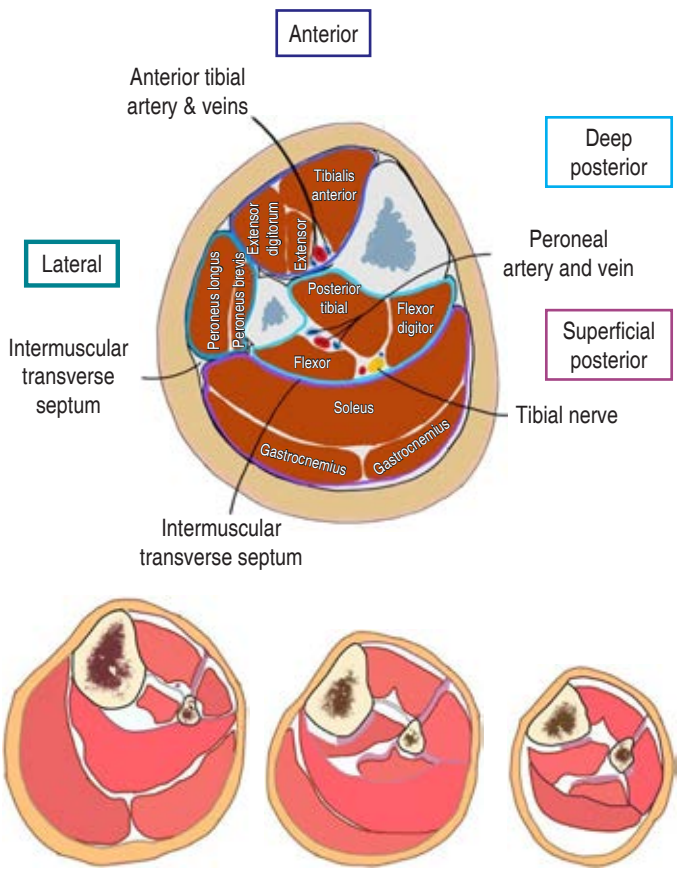

Figure 1: *Leg compartment.*

At the level of the leg *(Figure 1)*, a compartment is added and divided into anterior, lateral, superficial posterior, and deep posterior, which are formed as follows:

- **Anterior:** tibialis anterior muscle, extensor muscles of the foot, peroneal muscle. The anterior tibial artery and the deep perineal nerve are part of its irrigation and innervation.
- **Lateral:** the peroneus longus and peroneus lateralis brevis muscles are found here, the superficial perineal nerve runs through here, and the branches of the anterior tibial artery are found, which provide irrigation and innervation to these muscles.
- **Posterior superficial:** the gastrocnemius, soleus, and plantaris muscles are found, the tibial nerve provides innervation to these muscles, the sural arteries (medial and lateral) are also found, which provide irrigation to the gastrocnemius, while the popliteal, posterior tibial and peroneal arteries irrigate the soleus muscle.
- **Deep posterior:** the posterior tibial muscle, the foot flexor muscles, and the popliteal muscles are found. The tibial nerve and the posterior tibial and peroneal artery innervate it.

The foot *(Figure 2)* has medial plantar, central plantar, lateral plantar, interosseous, and dorsal compartments.

- **Medial plantar:** the abductor hallucis and flexor hallucis brevis are found, as well as the medial plantar nerves and vessels.
- **Central to the sole:** the short and long flexors of the toes are found, as well as Stan, the nerve, and plantar vessels.
- **Lateral to the sole:** the abductors and flexors of the little toe.
- **Dorsal:** which is limited by the dorsal fascia; it is the one that is more on the surface of the compartments.[21]

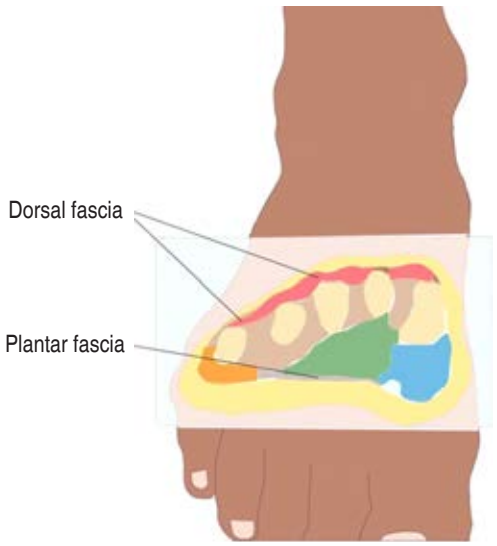

Figure 2: *Plantar and dorsal fasciae of the foot.*

At the level of the upper extremity *(Figures 3 to 5)*, specifically in the arm, there are anterior and posterior compartments.

- **Anterior:** contains the elbow flexor muscles (biceps brachii, brachialis) and the ulnar and median nerves.
- **Posterior:** contains the elbow extensor muscles (triceps) and the radial nerve.

PATHOPHYSIOLOGY

Multiple theories have been elaborated on the pathophysiology of compartment syndrome; however, they all conclude in the scenario of tissue anoxia.[20]

Ischemia begins once the local blood flow is restricted by increased compartment pressure, causing the metabolic demands of the local tissue to be unsatisfied.[21] Any etiology that causes increased fluid or pressure within a muscle compartment will elevate the internal pressure of the compartment due to the null distensibility capacity of the muscle fascia.[20]

The arteriovenous pressure gradient theory is the most widely accepted theory of acute compartment syndrome in extremities. Increased compartment pressure restricts local tissue perfusion by decreasing the arteriovenous pressure gradient, which consists of a decrease in arterial pressure and an increase in venous pressure. In turn, it will lead to a stage of cellular anoxia that will affect nervous and muscular tissue. A cycle is generated in which the effects of tissue hypoperfusion increase vascular permeability and, in turn, internal pressure.[20]

When intra-compartmental pressure rises within 10-30 mmHg of diastolic pressure, muscle oxygenation decreases as tissue pressure approaches mean arterial pressure, meaning that acute compartment syndrome of the extremities develops as a function of both compartmental and systemic arterial pressures.[20]

Nerve conduction is impaired when there is a difference between compartment pressure and a diastolic pressure of less than 30 mmHg or when compartment pressure is greater than 30 mmHg. If the pressure rises, conduction is wholly interrupted, and motor paralysis occurs-progression of ischemia results in cell death and myocytolysis. The severity of muscle damage is relatively proportional to the duration of ischemia of the affected limb;

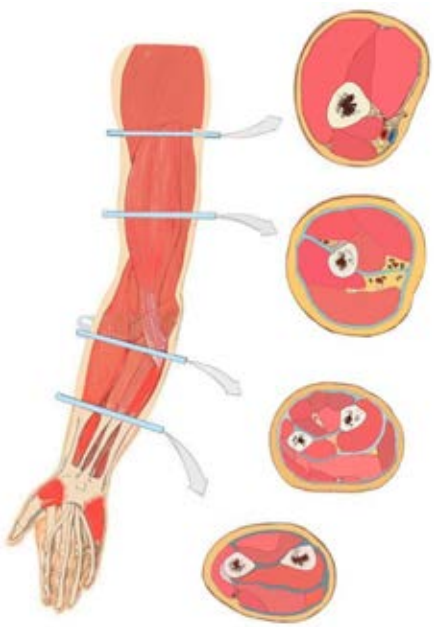

Figure 3: *Compartments of the upper extremity.*

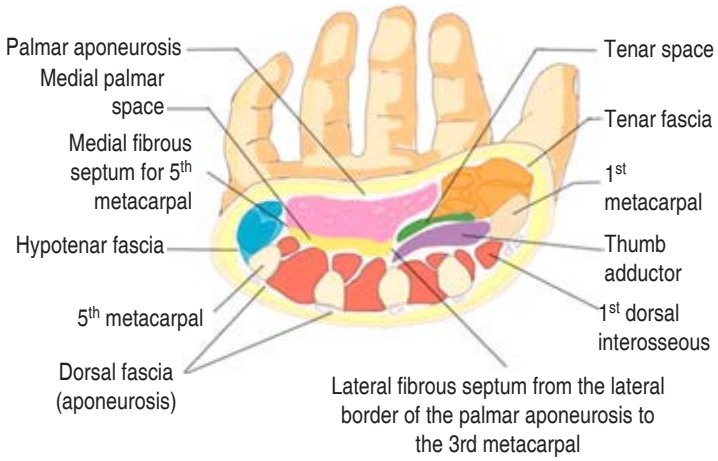

Figure 4: *Compartments at hand level.*

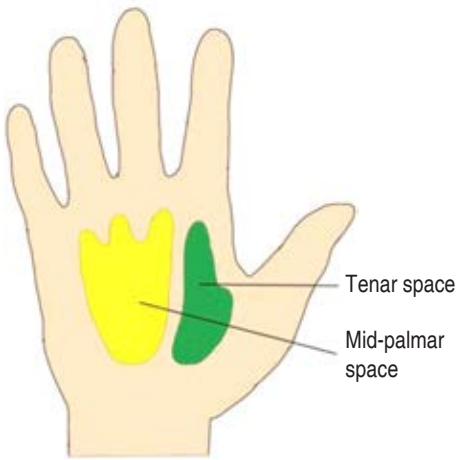

Figure 5: *Tenar and mid-palmar space.*

these effects will be irreversible after four to eight hours.[20]

CLINICAL PICTURE

Signs and symptoms of compartment syndrome (CS) include the "6 P's", of which pain out of proportion to the injury and increased analgesic requirements should raise suspicion of developing compartment syndrome,[22] other signs appear late and indicate delayed diagnosis, including pallor, paralysis, and absence of pulses.

1. **P**aresthesias: corresponds to the first indication of nervous ischemia and is the first symptom manifesting as a tingling sensation, burning, numbness, and loss of discrimination between two points.
2. **P**ain: intense and disproportionate to the lesion of stabbing or deep type, may be localized or diffuse, exacerbated by passive movement or direct compression of the affected compartment or elevation of the limb, and does not subside with narcotics.
3. **P**ressure: greater than 30-40 mmHg inside the compartment, measured by continuous infusion method or Stryker® (normal pressure 0-10 mmHg). The extremity is tense and warm on palpation, the skin taut and shiny.
4. **P**allor: late sign associated with progressive compromise of tissue perfusion, accompanied by cold and stiff skin with prolonged capillary filling (> 3 seconds).
5. **P**aralysis: late sign, weak or absent movement of distal joints, absence of response to direct neurological stimulation (damage of the myoneural junction).
6. The absence of pulses (Pulselessness) is a late sign. Clinically verified by palpation and absence of audible Doppler.[2]

Eliminating pain proportional to a muscle group's injury or passive stretching is considered the most important clinical finding; however, an assessment may be impaired due to altered consciousness, sedation, and mechanical ventilation.

Some general symptoms that can be found are abdominal pain or distension, dyspnea, dizziness, weakness, and malaise. Despite these clinical data, physical examination of the abdomen is a poor predictor of ACS.[23] Paralysis and paresthesias are less valuable data acutely, as they may result from neural trauma. Pulselessness is a late sign of CS but may be seen more frequently in combat wounds, firearm injury, arterial injury, or an expanding hematoma.

Other clinical data are tachycardia, hypotension, the elevation of jugular venous pressure, peripheral edema, and hypoperfusion data such as cold skin, obnubilation, restlessness, or lactic acidosis.[23]

DIAGNOSIS

The diagnosis of CS can be made clinically using the "6 P's"; however, there are more specific methods to determine the mmHg in a compartment to identify whether compartment syndrome is present. Signs and symptoms generally appear stepwise, although specific findings' appearance time varies.[15] When the diagnosis of ACS is suspected on clinical grounds, it is often confirmed by measuring compartment pressures. A careful and thorough neurological examination of the extremity should be performed and documented as part of the initial evaluation. A table describing signs and symptoms is provided *(Table 4)*.

MEASUREMENT OF COMPARTMENT PRESSURES

In remote areas and hospitals with limited surgical coverage, this approach is only sometimes possible but is preferable.

Direct measurement techniques

A hand-held manometer (e.g., Stryker device®), a simple needle manometer system, and the wick or slit catheter technique are the main approaches for direct measurements. The hand-held manometer method is most often used because it is portable, simple, and relatively accurate.[24] The slit catheter technique involves inserting a catheter into the compartment and monitoring the pressure through a transducer connected to a pressure amplifier and recorder.[25] The tip of the catheter or device should be within approximately 5 cm of the fracture level to obtain the maximum intra-compartmental pressure measurement.

Interpretation of measurements: normal tissue compartment pressure is between 0 and 8 mmHg.[26] Clinical findings associated with ACS generally correlate with the degree to which tissue pressure within the affected compartment approaches systemic arterial pressure: capillary blood flow is compromised when tissue pressure increases between 25 and 30 mmHg from mean arterial pressure. Pain may develop when tissue pressures reach 20 to 30 mmHg. Ischemia occurs when tissue pressures approach diastolic pressure.[27,28]

Indirect measurement techniques

There is a difference between diastolic blood pressure and compartment pressure (delta pressure) of 30 mmHg or less as a threshold for diagnosing ACS.[29,30] The delta pressure is found by subtracting the compartment pressure from the diastolic pressure, i.e.:

1. ACS delta pressure = diastolic blood pressure – measured compartmental pressure.

Table 4: Signs and symptoms.

Pain disproportionate to the apparent lesion (early and common finding)
Persistent deep aching or burning pain
Paresthesias (onset within approximately 30 minutes to 2 hours in acute compartment syndrome; suggests ischemic nerve dysfunction)
Pain with passive stretching of the muscles in the affected compartment (early finding)
Tight compartment with a firm "wooden" feel
Pallor due to vascular insufficiency (rare)
Decreased sensitivity
Muscle weakness (onset within approximately 2 to 4 hours in acute compartment syndrome)
Paralysis (late finding)

Modified from: Hammerberg EM.[21]

2. ACS delta pressure < 20 to 30 mmHg indicates the need for fasciotomy.[29,30]

If left untreated, ACS can lead to muscle necrosis, sensory deficits, paralysis, infection, fracture nonunion, and limb amputation.[6] Rhabdomyolysis may occur with muscle ischemia, resulting in myoglobinuria and possible renal failure requiring dialysis.

Treatment

In most cases, decompressive fasciotomy is the definitive treatment for acute compartment syndrome, where immediate open fascial decompression of all affected compartments is an emergency because delaying fasciotomy increases morbidity, including the need for amputation. Patients with late presentation or diagnosis (more than 12 hours) are at high risk for complications with surgery. Decision-making is complex and should involve two consultants. Upon clinical suspicion of compartment syndrome, immediate action should be taken by relieving external pressure on the compartment, which includes removing any bandages, splints, casts, or other restrictive coverings to examine the limb.[31,32]

Following fasciotomy, patients should undergo re-exploration at approximately 48 hours, or sooner if indicated, with the early involvement of a plastic surgeon to achieve adequate soft tissue coverage.[32]

Placement of the limb at the level of the heart is recommended; this helps to avoid reductions in arterial flow and elevations in compartment pressures due to swelling.

Indications and contraindications for fasciotomies are described below.[33]

Indications

Fasciotomy is indicated in clinical evidence of acute compartment syndrome (impending or established), which in some cases may be supported by objective evidence of elevated compartment pressure.

In impending compartment syndrome, symptoms may not be classic, and compartment pressure may not meet the criteria for acute compartment syndrome. However, there is a high likelihood of progression to compartment syndrome. Fasciotomy is performed earlier in this population based on the clinical judgment that the compartment is highly likely to develop. Fasciotomy should be performed immediately after worsening symptoms among those for whom careful management has been chosen.

Contraindications

Sometimes fasciotomy is not indicated or may not be necessary; for example, it should be avoided when the muscle is already dead. Fasciotomy, in such cases, provides no benefit and may increase the risk of infection. The definitive treatment for this type of injury often involves amputation.

A fasciotomy may not be necessary for specific low-risk circumstances, but the treating surgeon should make this decision.

CONCLUSION

In compartment syndrome, there is an increase of interstitial pressure in the body compartments that compromises the adequate perfusion of soft tissues, derived from an ischemic process that presents as clinical manifestations of sudden intense pain, pallor, absence of pulses, paresthesia, and paralysis. Multiple etiologies can cause an ACS, such as trauma, fractures, contusions, coagulopathies, burns, postischemic edema, or very tight casts, integrating the diagnosis with the physical examination.

The decompression of the affected muscle space is a surgical emergency, requiring timely detection and early treatment to avoid sequelae and serious complications.

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Recurrent hypoglycemia secondary to pancreatic insulinoma

Hipoglucemia recurrente secundaria a insulinoma pancreático

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Paola María Blanco-Pertuz,§ Gloria Caterine Pérez-Mingan§

Keywords:
pancreas, insulinoma, pancreatic neuroendocrine tumor, diagnosis, management.

Palabras clave:
páncreas, insulinoma, tumor pancreático neuroendocrino, diagnóstico, manejo.

ABSTRACT

Pancreatic neuroendocrine tumors are rare neoplasms of erratic behavior, which can be functioning or non-functioning according to their capacity to produce hormones and, therefore, generate diverse symptoms in affected patients; an accurate diagnosis will determine integral management, to improve the patient's quality of life, with emphasis on a complete resection that avoids the presence of residual disease. We present the case of a patient with recurrent hypoglycemic syndrome and imaging studies that showed a tumor lesion in the body of the pancreas, which was surgically operated on with total resection of the lesion, obtaining a diagnosis of pancreatic neuroendocrine tumor (insulinoma) in the pathological anatomy study.

RESUMEN

Los tumores neuroendocrinos pancreáticos son neoplasias poco frecuentes y de comportamiento variable, los cuales pueden ser funcionantes o no de acuerdo con su capacidad para producir hormonas y, por ende, generar diversos síntomas en los pacientes afectados; un diagnóstico certero va a determinar un manejo integral, con el cual se busca mejorar la calidad de vida del paciente, con énfasis en una resección completa que evite la presencia de enfermedad residual. Se presenta el caso de una paciente con síndrome hipoglucémico recurrente y con estudios de imagen que evidenciaron una lesión tumoral en cuerpo de páncreas, la cual es intervenida quirúrgicamente con resección total de la lesión, obteniendo como resultado en el estudio de anatomía patológica el diagnóstico de tumor neuroendocrino pancreático (insulinoma).

INTRODUCTION

Neuroendocrine neoplasms (NEN) are a group of tumors that originate from neuroendocrine cells located in all organs, mainly in the lung, gastrointestinal tract, and pancreas; at the pancreatic level, these lesions have a biological behavior according to their capacity to produce hormones and relatively different clinical management compared to adenocarcinomas; their incidence is less than or equal to one case per 100,000 individuals per year, and they only comprise 1 to 2% of pancreatic neoplasms, their incidence is increasing.[1]

In general, these neoplasms have a sporadic presentation; however, they are also associated with various hereditary entities such as multiple endocrine neoplasias (MEN) type 1, Von Hippel Lindau syndrome (VHL), and neurofibromatosis type 1 (NF-1).[2]

The diagnosis of these pathologies will depend on their functional capacity. Clinically, those with hormone production tend to have an earlier diagnosis and a smaller tumor size compared to non-functioning ones, in which the diagnosis is mainly due to incidental findings through imaging studies motivated by other causes unrelated to the lesion.[3]

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Within the pancreatic NEN, insulinomas are the pancreas' most common functioning endocrine neoplasm. Their main symptom is hypoglycemia, and their etiology is unknown, they occur in one to four persons per million in the general population, can be seen at any age, with an equal gender distribution; up to 90% of insulinomas are benign, 90% are solitary, > 90% occur in intrapancreatic sites, and 90% are < 2 cm in diameter, their distribution is uniform throughout the pancreas. Extrapancreatic localization is rare (incidence < 2%) with a higher frequency of wall duodenum; 90% occur in intrapancreatic sites, and 90% are < 2 cm in diameter; their distribution is uniform throughout the pancreas, and extrapancreatic location is rare (incidence < 2%) with greater frequency in the duodenal wall.[4]

PRESENTATION OF THE CASE

A 47-year-old female patient with no relevant personal or family history was referred from a less complex hospital for a clinical picture of 15 months of evolution due to recurrent episodes of asthenia, adynamia, pallor, and diaphoresis, including loss of consciousness, with documentation of glycemic levels below 40 mg/dl; these clinical manifestations entirely resolved after administration of unquantified glucose.

Due to severe and recurrent hypoglycemia not associated with the intake of antidiabetic or exogenous hypoglycemic agents, a 72-hour fasting test was performed with measurement of glucose and insulin levels, confirming hypoglycemia with normal insulin levels; a tomography of the skull and sella turcica was performed without finding lesions; However, an abdominal CT scan showed the presence of a focal increase in the size of the adrenal gland, so it was decided to characterize with MRI of the abdomen in which a single focal lesion of 13 × 10 mm was identified at the junction of the pancreatic body and tail *(Figure 1)*, described as hypointense in T1 sequences fat saturation techniques (FAT SAT), slightly hyperintense in T2 sequences with discrete peripheral enhancement after contrast administration and restriction in diffusion-weighted magnetic resonance imaging (DWI) sequence and an apparent diffusion coefficient (ADC) map.

She was evaluated by the endocrinology group, considering a neuroendocrine tumor in the pancreas; paraclinical and tumor markers were requested that showed positivity for chromogranin A of 412 ng/ml *(Table 1)*; due to recurrence and persistence of hypoglycemia despite high metabolic flows, it was decided to perform a distal pancreatectomy by laparoscopy through which a single 1.5 cm tumor located in the body of the pancreas was found and removed. The specimen was subsequently sent to the pathology department.

Pathology analysis revealed the presence of a pancreatic neuroendocrine tumor compatible with insulinoma from the submitted clinical history; the diagnosis was confirmed after performing the relevant immunohistochemistry studies *(Figure 2)*.

After the procedure, the patient evolved asymptomatic, without new episodes of hypoglycemia after several days of observation, the reason for which she was discharged with the indication for outpatient follow-up. She

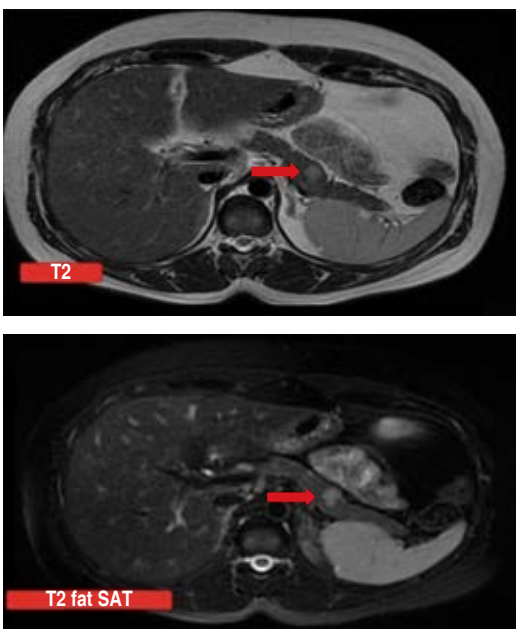

Figure 1: *Gadolinium-contrast MRI in a T2 and fat T2 sequence showing a 12.2 × 9.7 mm tumor lesion in the pancreatic body.*

Table 1: Paraclinical tests performed during the patient's hospital stay, among which the elevated value of the tumor marker chromogranin A stands out.

09/13/2021	
Gastrin, pg/ml	10.3*
Free insulin, ImU/l	14.8*
PTH intact molecule, pg/ml	35*
Total triiodothyronine, ng/ml	0.7*
Total thyroxine, ug/dl	7.2*
TSH, IU/ml	2.83*
HB AC1, %	5.2*
White blood cells, %	8,300
Neutrophils	75
Lymphocytes	15
Hemoglobin, g/dl	10.6
Hematocrit, %	38
VCM, um^3	70
HCM, pg	19
MHC, g/dl	28
EDI, %	19
Platelets, mm^3	363,000
09/15/2021	
Chromogranin A, ng/dl (range)	412 (0-100)
Natriuretic peptide B, pg/dl	2.25*

PTH = parathyroid hormone. TSH = thyroid stimulating hormone. HB = hemoglobin. MCV = mean corpuscular volume. MCH = mean corpuscular hemoglobin. MCHC = mean corpuscular hemoglobin concentration. EDI = erythrocyte dispersion index.
* Normal values.

was evaluated at an outpatient clinic one month after her discharge without evidence of new episodes of hypoglycemia. She was asymptomatic, and the contrasted abdominal tomography follow-up scan showed no residual lesions, with changes secondary to her surgical intervention; she was indicated to continue medical follow-up every three months.

DISCUSSION

The diagnostic and therapeutic approach to this entity should be multidisciplinary. The initial approach should be performed by a specialist in endocrinology who knows the management guidelines, added to a good radiology service that establishes the presence of the lesion and its delimitation so that in case there is the possibility of resection, the procedure is performed by a highly qualified surgical group that guarantees good oncological results.[5]

In patients with insulinoma, episodes of hyperinsulinemic hypoglycemia cause various autonomic and neuroglycopenic symptoms, usually appearing on an empty stomach. Documentation of the so-called Whipple's triad, i.e., symptoms consistent with hypoglycemia, low plasma glucose measured at the time of symptoms, and immediate relief of symptoms after glucose administration, is the cornerstone of the diagnosis of insulinoma.[6]

Demonstration of concomitant low plasma glucose with inappropriately high serum insulin and C-peptide levels in a symptomatic patient forms the basis for biochemical diagnosis, with the exclusion of other causes of hyperinsulinemic hypoglycemia; B-hydroxybutyrate levels of 2.7 mmol/l or less, an increase in plasma glucose of at least 1.4 mmol/l after intravenous glucagon administration, and a negative detection of oral hypoglycemic agents distinguish endogenous hyperinsulinemic hypoglycemia from that caused by other mechanisms.[7]

A 72-hour fasting test with plasma glucose, insulin, and C-peptide measurements is considered the gold standard for the biochemical diagnosis of insulinoma.[6]

Dynamic gadolinium-enhanced magnetic resonance imaging (MRI), three-phase computed tomography (CT) scan, and endoscopic ultrasonography (EUS) have been considered the most useful imaging modalities for the evaluation of insulinomas; in experienced hands, the sensitivity of EUS is 70-95% and in combination with three-phase CT, sensitivities of up to 100% have been reported.[8]

The cornerstone for complete resection of the lesion is surgical treatment. Laparoscopic surgery with intraoperative ultrasound confirmation of the location of the lesion is preferred, considering the inherent advantages of minimally invasive surgery (less postoperative pain, shorter hospital stay, better cosmetic results, reduced morbidity), but

open surgery can also be used without being contraindicated.[9]

Despite the characteristic clinical behavior of insulinomas, diagnostic confirmation should always be made by anatomic pathology; these lesions are characterized at the macroscopic level as solitary, well-demarcated lesions with a homogeneous, tan-yellow cut surface with or without hemorrhage.[5] Microscopically, monotonous cells showing round nuclei with salt/pepper chromatin and abundant cytoplasm arranged in a trabecular, nested, cribiform, or solid architecture are characteristic.[10]

Immunohistochemical labeling is extremely useful. Stains to be used include neuroendocrine markers such as synaptophysin, chromogranin A, insulin gene enhancer protein ISL-1, proinsulin, amylin, and islet amyloid polypeptide to confirm the suspicion, in addition to the Ki-67 cell proliferation index and epithelial markers to rule out lesions such as cytokeratin cocktails.[11]

In addition to the conventional study, genetic analysis should be performed in patients with early onset of these lesions, mainly recommended in patients under 30 years of age, to rule out the involvement of hereditary clinical syndromes such as multiple endocrine neoplasias (MEN) type 1, Von Hippel Lindau syndrome (VHL) and neurofibromatosis type 1 (NF-1) and to help screen the patient for synchronous lesions of another nature.[12]

The prognosis of these patients in the postoperative period is generally good; however, some factors have been described that are related to a less favorable evolution, among which tumor size greater than 2 cm, high Ki-67 labeling index, and high mitotic count stand out. Despite these, malignant

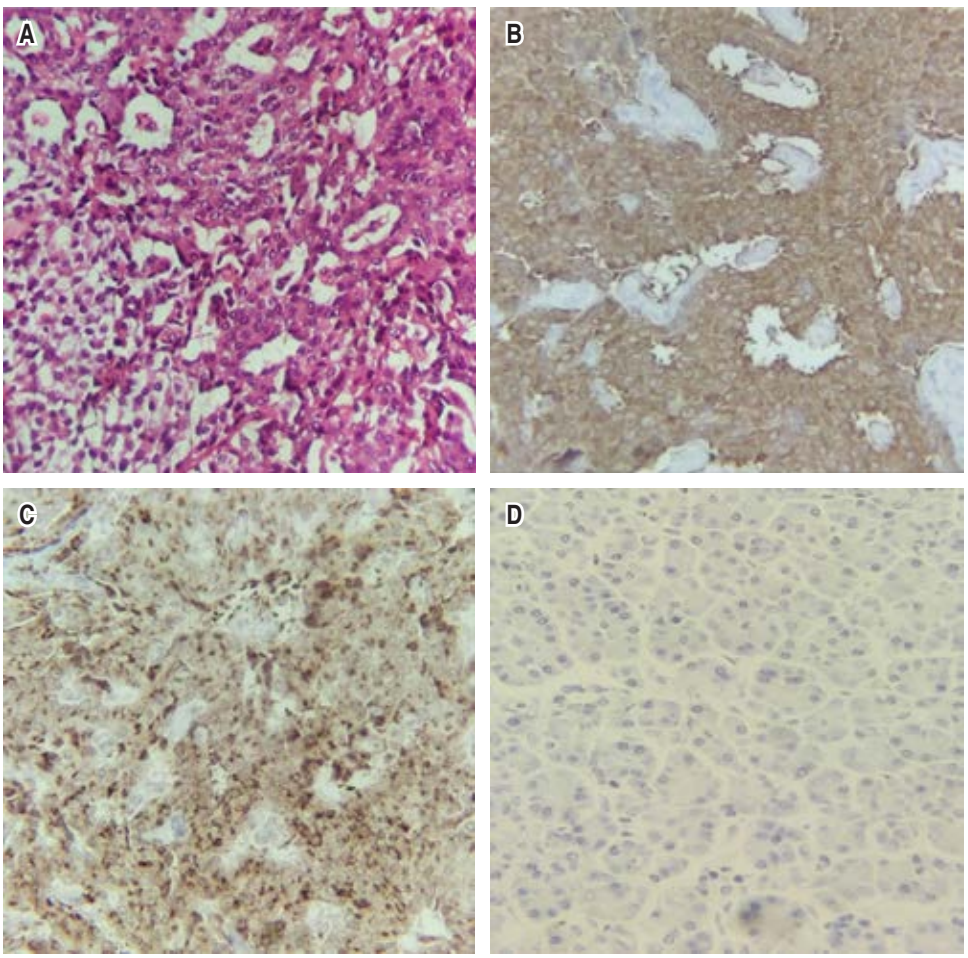

Figure 2:

A) *Tumor lesion consisting of small to medium-sized cells with eosinophilic to amphophilic and firmly granular cytoplasm, uniform, central, oval nuclei, with chromatin arranged in a "salt and pepper" pattern, which are arranged in a cribriform pattern, with a rich vascular network; in other areas, cells with clear and vacuolated cytoplasm are seen.* ***B-C)*** *Cytoplasmic positivity for immunohistochemical markers synaptophysin and chromogranin.* ***D)*** *Low Ki-67 proliferation index, less than 5% of the tumor volume.*

insulinomas are frequently grade 2 according to the World Health Organization classification.[13]

CONCLUSION

Despite its low incidence, insulinoma is the most frequent neuroendocrine neoplasm of the pancreas susceptible to curative surgical treatment, so its recognition and timely management is of vital importance; for the patient's approach, the multidisciplinary approach will allow better management decisions and, therefore, more favorable oncologic results with improvement in the patient's quality of life.

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Hepatic abscess secondary to intrahepatic pyocholecyst perforation

Absceso hepático secundario a ruptura intrahepática de piocolecisto

Mizael Orlando Sevilla-Santoyo,[*] Luis Enrique Bolaños-Badillo,[‡] Julián Felipe Frías-Almaraz[§]

Keywords:
pyocholecyst, bilioma, gallbladder perforation, liver abscess, laparoscopy.

Palabras clave:
piocolecisto, bilioma, perforación vesícula biliar, absceso hepático, laparoscopia.

ABSTRACT

Introduction: spontaneous gallbladder perforation is a rare complication of gallbladder pathologies; in acute cholecystitis, it occurs in approximately 2% of cases and is associated with mortality between 12 and 42%. If it occurs with a concomitant hepatic abscess, it is even rarer. **Material and methods:** a 55-year-old female patient attended the emergency department for abdominal pain in the epigastrium and right hypochondrium, radiating to the interscapular region of one month of evolution. Leukocytosis, elevated alkaline phosphatase (ALP), and gamma-glutamyl transferase (GGT) were found; contrasted abdominal computed axial tomography (CT) scan showed gallbladder with impacted lithos in its interior, intrahepatic rupture, and intrahepatic abscess. **Results:** we performed cholecystectomy plus abscess drainage and placement of drains by laparoscopy. We placed four trocars; we found a Parkland 5 gallbladder and a hepatic abscess in segments IV and V and extracted the piece with an extractor bag. Finally, we placed 19 Fr drains to Winslow's hiatus and the abscess cavity. **Conclusions:** vesicular rupture with liver abscess formation is a rare complication. It represents a diagnostic challenge for the surgeon; however, the diagnosis can be made promptly thanks to new diagnostic tools.

RESUMEN

Introducción: *la perforación espontánea de la vesícula biliar es una complicación poco frecuente de las patologías vesiculares, en la colecistitis aguda se presenta aproximadamente 2% de los casos, se asocia con una mortalidad entre 12 y 42%. Si se presenta con un absceso hepático concomitante es aún más rara.* ***Material y métodos:*** *paciente femenino de 55 años, quien acudió al Servicio de Urgencias por dolor abdominal en epigastrio e hipocondrio derecho, irradiado a región interescapular de un mes de evolución. Se encontró leucocitosis, elevación de fosfatasa alcalina (FA) y gamma-glutamil transferasa (GGT); la tomografía axial computarizada (TAC) abdominal contrastada mostró vesícula biliar con litos impactados en su interior, ruptura y absceso intrahepáticos.* ***Resultados:*** *realizamos colecistectomía más drenaje de absceso y colocación de drenajes por laparoscopia. Colocamos cuatro trocares, encontramos vesicular biliar Parkland 5 y absceso hepático en segmentos IV y V, se extrajo pieza con bolsa extractora y finalmente se colocaron drenajes de 19 Fr en el hiato de Winslow y a cavidad del absceso.* ***Conclusiones:*** *la ruptura vesicular con formación de absceso hepático es una complicación poco frecuente. Representa un reto diagnóstico para el cirujano; sin embargo, gracias a las nuevas herramientas diagnósticas se puede realizar el diagnóstico de manera oportuna.*

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INTRODUCTION

Spontaneous perforation of the gallbladder is a rare and severe complication of gallbladder pathologies that can put the patient's life at risk.[1-3] Its presentation with concomitant hepatic abscess is an even rarer complication. In the case of patients with acute cholecystitis, it is known that approximately 0.8 to 3.2% of cases present perforation of the gallbladder into the abdominal cavity;[2,4,5] however, the incidence of perforation with the formation of a secondary intrahepatic abscess is unknown, and there are only a few case reports in the literature.[2] This is associated with high morbimortality, with reported mortality ranging

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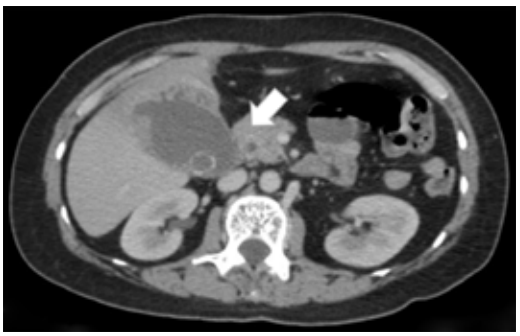
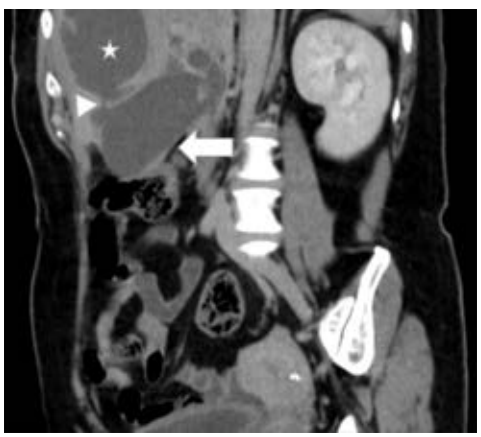


Figure 1: *An axial plane computed tomography showing the intravenous contrast in the venous phase. The gallbladder (arrow) is visualized as expanded with localized lithos towards the gallbladder neck, and the fundus wall is visualized discontinuously towards the hepatic parenchyma with associated edema.*

Figure 2:

Computed axial tomography with sagittal reconstruction in the venous phase image. The gallbladder (arrow) with a 2 mm communication (arrowhead) towards the collection (star) of biliary-type liquid content located in hepatic segments IV and V is visualized.



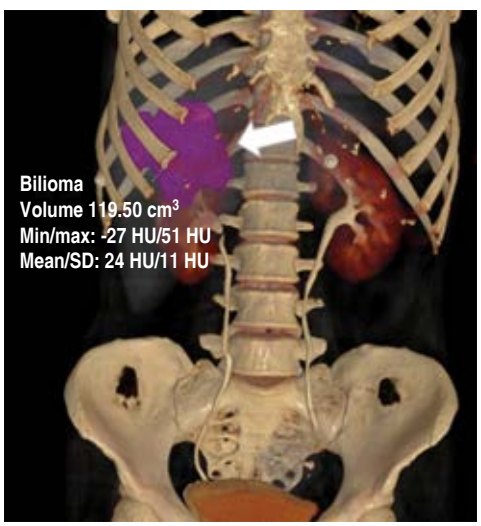


Figure 3:

VR3D reconstruction with the siemens Syngo via platform in which bilioma is quantified (arrow) with a volume of 119.5 ml.

between 12 and 42% of cases,[5] even with aggressive medical management and multiple interventions.[2]

Perforation of the gallbladder develops from a series of events triggered by obstruction of the cystic duct, usually by a litho, followed by bile stasis and gallbladder distension. Subsequently, it causes increased intraluminal pressure leading to vascular and lymphatic compromise, resulting in vesicular necrosis and, finally, vesicular perforation. The gastric fundus is the most frequent location of vesicular perforations because it is the most distal area and contains the most precarious blood supply.[1,2,4]

In the approach to this pathology, abdominal ultrasound can provide vital information for the diagnosis; however, a computerized tomography scan is a more sensitive tool to reach an accurate diagnosis and plan the resolution of the condition.[2]

PRESENTATION OF THE CASE

A 55-year-old female patient came to the emergency department with a picture of a month of evolution characterized by abdominal pain located in the epigastrium and right hypochondrium, radiating to the interscapular region, which was increasing, so she decided to go to the doctor who started homeopathic treatment; however, she did not improve, so she went to the emergency department of our hospital unit where the following vital signs were recorded: BP (blood pressure) 114/76 mmHg, HR (heart rate) 124 bpm, RR (respiratory rate) 16/min, and temperature of 36 ºC. On questioning, the patient denied the presence of fever, nausea, vomiting, or the presence of bowel movements of decreased consistency. For her approach, general laboratories were requested, highlighting the presence of leukocytosis (12,300 mm^3), elevated alkaline phosphatase, and GGT (315 and 268 U/l respectively); a CT scan of the abdomen with intravenous contrast was requested *(Figures 1 to 3)* which showed the presence of gallbladder with impacted lithos in its interior, and an intrahepatic rupture, in addition to an intrahepatic abscess in segments IV and V *(Figure 2)*.

Antibiotic therapy based on carbapenems and imidazole was started with subsequent

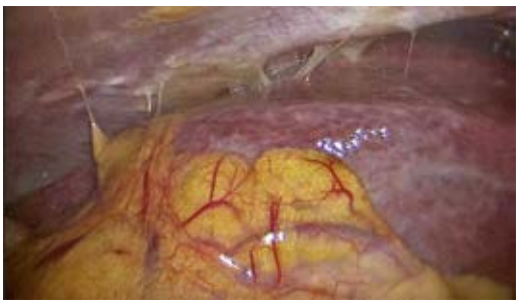


Figure 4: *Gallbladder completely covered by omentum (Parkland 5) and hepatic abscess.*

surgical management. Cholecystectomy plus abscess drainage and placement of drains by laparoscopy were performed. Four trocars of 12 mm were placed in the umbilical and subxiphoid region; in the hypochondrium and right flank, a 5 mm trocar was placed. We found a gallbladder covered entirely by omentum corresponding to Parkland 5 *(Figure 4)* with edematous walls and a hepatic abscess in segments IV and V adhered to the abdominal wall. Approximately 200 cm^3 of purulent material (pyococcus and abscess) were drained *(Figure 5)*, which were sent to culture, where growing of *Streptococcus anginosus* and *Enterobacter cloacae* was reported. The surgical piece was removed with an extractor bag, and finally, 19 Fr Blake-type closed drains were placed into the Winslow's hiatus and the abscess cavity, the patient was discharged home on the fourth postoperative day. The antimicrobial scheme was adjusted at discharge with ampicillin/sulbactam and metronidazole to complete 14 days of antibiotic therapy. In the follow-up consultation, on the seventh postoperative day, the patient was pain-free at the surgical site, afebrile, with good tolerance to oral administration and regular bowel movements. It was decided to remove both drains.

DISCUSSION

Gallbladder rupture is an infrequent complication[1] of biliary pathologies. It is more prevalent in the female sex in acute cholecystitis; however, gallbladder perforation is more common in men.[1] It occurs in approximately 2-10% of patients with acute cholecystitis,[4,5] with a mortality of 12-42%.[6]

Gallbladder perforation secondary to lithiasis occurs in patients who delay surgical treatment or who do not improve with conservative management;[4] on the other hand, in the context of an alithiasic cholecystitis, patients are more susceptible to gallbladder perforation.[7]

The clinical presentation is usually insidious, with abdominal pain predominantly in the right upper quadrant, and may be associated with fever and jaundice. Elevation of liver enzymes, predominantly alkaline phosphatase (ALP), is expected.[2,7]

Gallbladder perforation begins due to obstruction of the cystic duct, preventing the outflow of bile to the main biliary tract, which is secondary to a litho causing stasis inside the gallbladder and subsequent distension. This increases intravesicular pressure, compromising venous and lymphatic drainage, resulting in ischemia and, finally, gallbladder perforation occurs.[7,8] The leading perforation site is the fundus; since it is the most distal portion of the organ, it has the least blood supply and therefore is more susceptible to ischemia and necrosis.[6] The development of biliary peritonitis is more common in this location; however, perforations in other portions of the gallbladder are usually sealed by the omentum or intestinal loops, which limits damage to the right hypochondrium and abdominal cavity.[6]

In 1934 Niemeier proposed a classification for gallbladder perforation *(Table 1)*:[6] type 1 perforations represent 16% and are considered chronic because they have a fistulous tract;

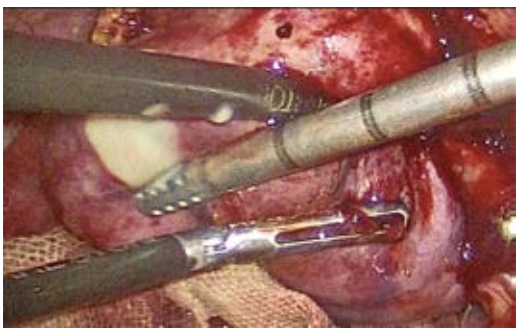


Figure 5: *Drainage of purulent material.*

Table 1: Niemeier classification: types of gallbladder perforation.

Type	Features
1	Chronic perforations: the fistulous tract is seen between the gallbladder and some other viscera
2	Subacute perforations: the gallbladder is surrounded by an abscess and adhesions in the peritoneal cavity
3	Acute perforation: free perforation into the abdominal cavity

type 2 perforations are the most common (60%); and type 3 perforations represent 16% and are associated with more significant mortality;[4,7] however, this classification does not include complications such as intrahepatic rupture with the formation of a secondary hepatic abscess.[1] Nevertheless, any vesicular perforation is considered a surgical emergency.[1] Niemeier perforations types 1 and 3 usually require urgent surgical management, while type 2 perforations can be managed initially with drainage or cholecystostomy and later cholecystectomy.[4,6]

In an article published by Hussain in 2016,[1] it is reported that most cases of hepatic abscess secondary to vesicular rupture are resolved with open cholecystectomy. In our case, we opted for an initial laparoscopic approach obtaining good postoperative results like those published by Quiroga,[6] where the mean in-hospital stay was five days. This same study reports that almost half (42%) of the patients submitted to laparoscopic cholecystectomy required a second intervention; in our case, no additional procedure was necessary.

CONCLUSIONS

Vesicular rupture with subsequent liver abscess formation is a rare complication. It represents a diagnostic challenge for the surgeon; however, early diagnosis is possible thanks to new diagnostic tools.

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Cholecystogastric fistula: A case report

Fístula colecistogástrica: reporte de caso

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Keywords: cholecystogastric fistula, interval cholecystectomy, open surgery.

Palabras clave: *fístula colecistogástrica, colecistectomía de intervalo, cirugía abierta.*

ABSTRACT

Introduction: cholecystoenteric fistulas are rare, the most common being cholecystoduodenal. They are due to erosion of the gallbladder with subsequent formation of the fistulous tract. **Case report:** a 59-year-old female patient with a history of biliary colic of six months of evolution. Abdominal tomography showed pneumobilia, gastric distension, and pyloric lithotripsy. Conservative surgery was performed, with the removal of the litho and placement of a nasojejunal tube. **Conclusion:** interval cholecystectomy is a surgical option.

RESUMEN

Introducción: *las fístulas colecistoentéricas son poco frecuentes, la más común es la colecistoduodenal. Se deben a la erosión de la vesícula con ulterior formación del trayecto fistuloso.* ***Caso clínico:*** *paciente femenino de 59 años, con antecedente de cólico biliar de seis meses de evolución. Con tomografía de abdomen se observó pneumobilia, distensión gástrica y lito en píloro. Se realizó cirugía conservadora, con remoción del lito y colocación de sonda nasoyeyunal.* ***Conclusión:*** *la colecistectomía de intervalo es una opción quirúrgica.*

INTRODUCTION

Cholecystogastric fistulas are a rare complication of cholecystitis and cholelithiasis; the reported incidence is 1-3%. The pathogenesis of fistula formation is due to erosion and necrosis of the gallbladder wall, which in turn encounters the adjacent hollow viscera.[1] The local inflammation induces the formation of adhesions and, finally, the formation of the fistula. The gallstone can reach the stomach through the cholecystogastric or cholecystopyloric fistula and indirectly through a cholecystoduodenal or choledochoduodenal fistula.[2] The diagnosis of cholecystogastric fistula is made trans operatively in up to 90% of cases since most patients present with non-specific symptoms; however, up to 7-10% will develop biliary ileus.[3]

PRESENTATION OF THE CASE

The patient was a 59-year-old female with a history of type 2 diabetes mellitus of long evolution. She came to the emergency department with colicky pain in the right hypochondrium after six months of evolution, intensity 6/10 on the visual analog scale, without irradiation, associated with the ingestion of cholecystokinetic foods, without mitigating factors. She denied fever, jaundice, choluria, or acholia. On physical examination, the Murphy sign was absent, with pain on deep palpation in the right hypochondrium, with no evidence of peritoneal irritation. She has the following laboratories: hemoglobin 12.9 g/dl, hematocrit 37%, leukocytes 9.4 × 10^3/μl, platelets 360 × 10^3/μl, glucose 178 mg/dl, creatinine 0.7 mg/dl, sodium 145 mEq/l,

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potassium 3.7 mEq/l, chlorine 105 mEq/l, total bilirubin 0.7 mg/dl, alkaline phosphatase 55 U/l. Ultrasound of the liver and biliary tract was performed, which revealed an increased echogenicity of the gallbladder throughout its extension, causing a wall-echo-shadow (WES) phenomenon and measuring 52.7 × 29.4 × 18.7 mm. The wall was non-evaluable; the common bile duct measured 5.8 mm, with no evidence of any litho inside *(Figure 1)*. An abdominal tomographic scan showed a heterogeneous gallbladder with a volume of 13.2 cm^3, with the presence of air density and a regular thickened wall with the presence of calcifications, a distended stomach, and the presence of a hyperdense image inside resembling a probable litho *(Figure 2)*.

Due to imaging findings, a cholecystogastric fistula was suspected, and the patient was scheduled for open cholecystectomy. Other findings were important adhesive process, a scleroatrophic gallbladder, and a cholecystogastric fistula are evidenced. We proceeded to perform gastrorrhaphy and extraction of a 2 × 3 cm litho with primary closure of the fistula. It was decided to place a nasojejunal tube to keep the pylorus bypassed. A cholecystostomy tube was placed *(Figure 3)*.

After surgery, parenteral nutrition was started. On the fifth postoperative day, a computed axial tomography (CT) scan with oral contrast showed no evidence of contrast medium leakage *(Figure 4)*, and it was decided to restart the oral route.

DISCUSSION

Cholecystogastric fistula is a rare complication of cholecystitis and is more prevalent in females between the seventh and eighth decade of life. Litho migration into the stomach can lead to pyloric stenosis and gastric mucosal hemorrhage. When the migration of the litho causes occlusion of the pylorus, it is called Bouveret's syndrome.[4] If migration occurs caudally, it causes biliary ileus.

The signs and symptoms of patients with cholecystogastric fistulas are diffuse and unspecific, hence the difficulty of making a preoperative diagnosis. It is helpful to perform imaging studies. Ultrasound is performed initially but is not specific and operator dependent. Pneumobilia, thickened gallbladder wall, and enclaved lithos can be observed. A CT scan is the study of choice, which may reveal pneumobilia (present in 30-70% of

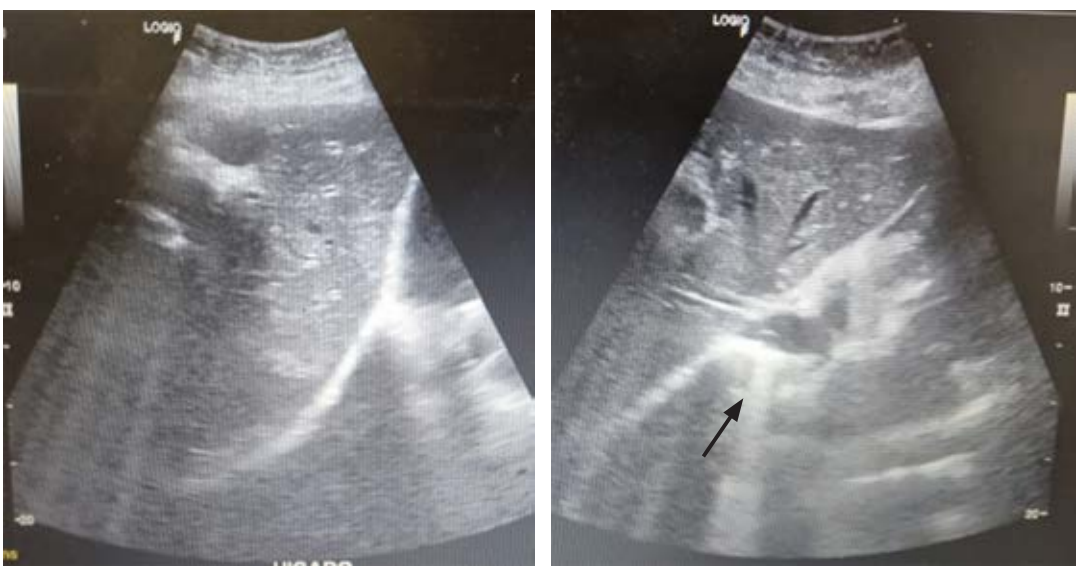

Figure 1: *Ultrasound scan of liver and biliary tract. The arrow indicates the vesicular and scleroatrophic wall thickening.*

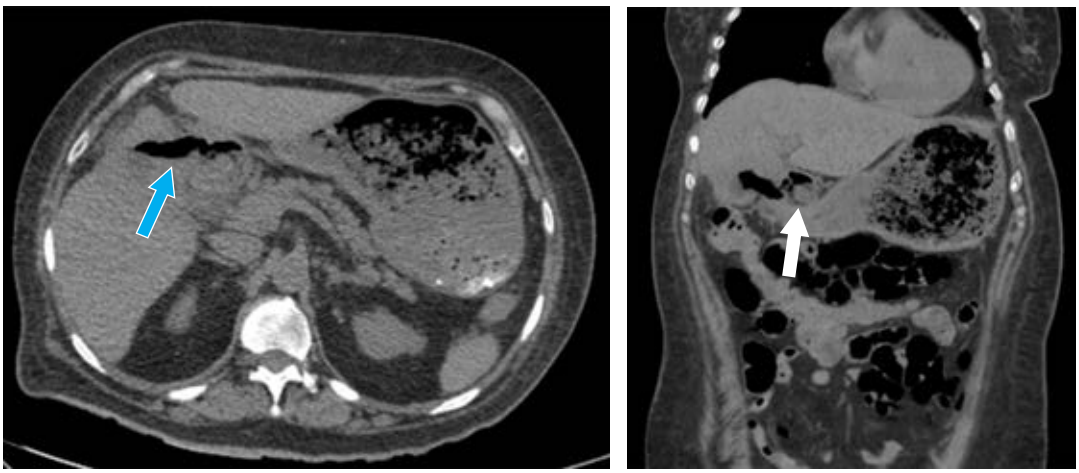

Figure 2: *An abdominal simple computed axial tomography scan showing evidence of pneumobilia (blue arrow). The white arrow shows an image suggestive of a litho.*

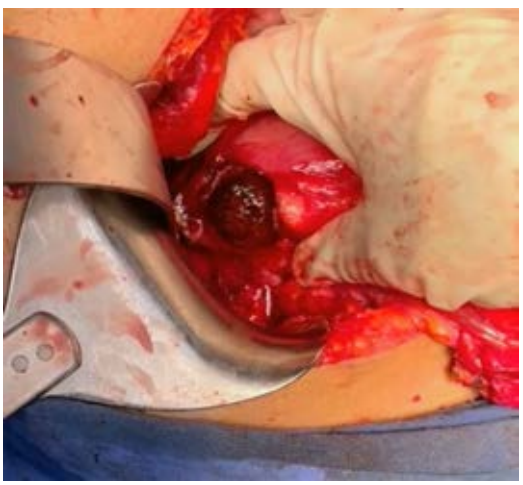

Figure 3: *Pyloric enterotomy with litho removal.*

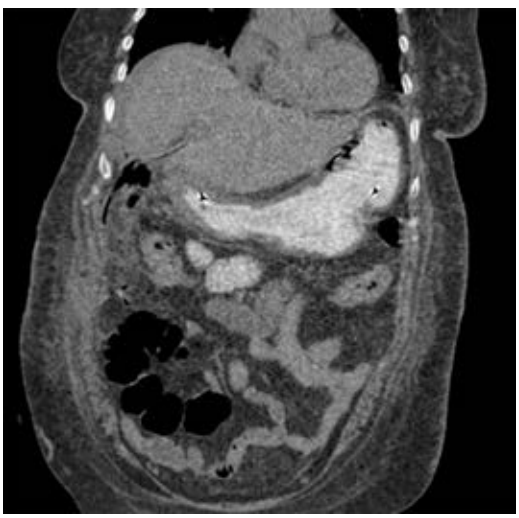

Figure 4: *Computed axial tomography scan with oral contrast showing no evidence of contrast medium leakage.*

cases), cholecystitis, and gastric distension. When biliary ileus is suspected, the Rigler's triad -pneumobilia, intestinal distension, and ectopic cyst- may be found.[5]

Surgical treatment has been the treatment of choice in patients with fistulas; open surgery is the preferred modality for surgeons due to the intense inflammatory process that characterizes these clinical conditions, which makes tissue dissection difficult. The management of fistulas has been described laparoscopically, and in specialized centers, the rate of conversion to open surgery is reported to be 6%.[6] The decision to choose laparoscopic surgery will depend on the patient's comorbidities and the surgeon's experience.

Another essential aspect to consider is managing a single surgical procedure, which includes removing the litho, closing the fistula, and performing a cholecystectomy. The removal of the litho is achieved through an enterotomy, and a gastroduodenostomy

or a Billroth II anastomosis can be performed to allow the pylorus to heal. However, this procedure increases the risk of bile duct injury. Due to the complications of the one-stage approach, a two-stage surgical procedure has been proposed, consisting of interval cholecystectomy and repair of the fistula.[7] In some cases, spontaneous fistula closure is reported without the need for cholecystectomy when the cystic duct remains intact.

On the other hand, when a non-impacted gallstone is identified in the pylorus, a laparoscopic approach can be considered, thus avoiding gastrostomy. Endoscopic treatment is feasible if the gallbladder is identified with no litho in its interior and an intact biliary tract.[8]

CONCLUSION

Cholecystogastric fistula is a rare complication of cholecystitis. The diagnosis is usually made trans operatively. The surgical approach is the treatment of choice with the closure of the fistula, cholecystectomy, and pyloric bypass. A single-stage surgical procedure is associated with more significant complications. In the case of our patient, we decided to perform a less invasive approach by removing the litho, interval cholecystectomy, placement of a nasojejunal tube, and initiation of parenteral nutrition, which presents an adequate postoperative evolution.

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Internal small bowel herniation through peritoneal defect following inguinal hernia repair with the TAPP technique

Hernia interna del intestino delgado a través de defecto peritoneal posterior a reparación de hernia inguinal con técnica TAPP

Meri Yeghiazaryan,[*] Mauricio González-Urquijo,[‡] Román González-Ruvalcaba,[‡] María Luisa Moreno-García,[§] Cristian Santiago Ramírez-López[¶]

Keywords:
internal hernia, peritoneal defect, inguinal hernia, transabdominal preperitoneal technique.

Palabras clave:
hernia interna, defecto peritoneal, hernia inguinal, técnica transabdominal preperitoneal.

ABSTRACT

Small bowel obstruction following inguinal hernia repair by transabdominal preperitoneal technique is rare. As procedures such as the transabdominal preperitoneal technique (TAPP) are performed more frequently, uncommon complications become more frequent and must be considered in perioperative and postoperative management. We present the case of a 26-year-old male patient who presented 10 hours after his discharge from inguinal hernioplasty by laparoscopy with preperitoneal transabdominal technique, with clinical symptoms suggestive of intestinal obstruction; an internal hernia of the small intestine was observed through the peritoneal defect, this being an uncommon complication after this procedure. Diagnostic laparoscopy was performed, and a small bowel loop herniated in the preperitoneal space through a peritoneal defect was identified as the origin of the obstruction without evidence of loop distress, so the hernia was reduced. Subsequently, the defect was closed with polyglactin 910 3-0.

RESUMEN

La obstrucción del intestino delgado posterior a reparación de hernia inguinal por técnica transabdominal preperitoneal es poco frecuente. A medida que se realizan con mayor frecuencia procedimientos como la técnica transabdominal preperitoneal (TAPP), las complicaciones poco comunes se vuelven más frecuentes y se deben tener en cuenta en el manejo perioperatorio y postoperatorio. Presentamos el caso de paciente masculino de 26 años que acude 10 horas posteriores a su egreso de hernioplastía inguinal por laparoscopia con técnica transabdominal preperitoneal, con cuadro clínico sugestivo de una obstrucción intestinal, se observó una hernia interna del intestino delgado a través de defecto peritoneal, siendo ésta una complicación poco común posterior a este procedimiento. Se realizó laparoscopia diagnóstica, se identificó como origen de la obstrucción un asa de intestino delgado herniada en el espacio preperitoneal a través de un defecto peritoneal sin evidencia de sufrimiento de asa, por lo cual se reduce la hernia y posteriormente se cierra el defecto con poliglactina 910 3-0.

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INTRODUCTION

Obstruction of the small bowel following inguinal hernia repair by transabdominal preperitoneal technique (TAPP) is rare, with an incidence of 0.2 to 0.5%. Common causes of this are inadequate closure, port site herniation, and adhesions. Several risk factors that may predispose to this complication's development must be considered to avoid it.[1]

The cause of a hernia in the preperitoneal space can be attributed to insufficient closure of the peritoneal flap with loosening of the suture and subsequent displacement of the small bowel, or it can also herniate through a poorly closed peritoneal defect. Complications

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arising from failure to close peritoneal tears include exposure of the mesh to the bowel with a risk of bowel erosion, adhesion formation, internal herniation through the tear, and bowel obstruction.[2]

A literature review was done, and a few reported cases of this complication were found. The study's objective is to present the case of a 26-year-old patient postoperative of TAPP, who suffered from intestinal obstruction secondary to small bowel loop incarceration in the peritoneal flap of said hernioplasty. Likewise, a literature review was carried out since this complication is infrequent; it is essential to know about it and to take it into account within the differential diagnoses to solve it promptly.

CLINICAL CASE

The case of a 26-year-old male patient with no medical history of importance is presented. He started suffering 10 hours after his discharge from inguinal hernioplasty by laparoscopy with the TAPP technique with nausea and vomiting of gastro alimentary content on three occasions, associated with abdominal pain in the lower quadrants, cramping, and absence of bowel movements.

On physical examination, his vital signs were within normal parameters; the mucous membranes were underhydrated, the abdomen had decreased peristalsis, and was soft, depressible, and with mild pain on mid and deep palpation in a generalized manner, with no evidence of peritoneal irritation.

Labs were requested and revealed hemoglobin 17.2 g/dl, hematocrit 50%, leukocytes 12,800 cells/mm^3, platelets 337,000 cells/mm^3, C-reactive protein 6.51 mg/dl, serum creatinine 1.40 mg/dl, and BUN 23 mg/dl.

The patient was admitted for observation, intravenous hydration with lactated Ringer's solution was administered, and symptomatic medical management was instituted, persisting with symptomatology, for which the placement of a nasogastric tube (NGT) and a computed tomography (CT) scan of the abdomen with intravenous contrast was indicated. The abdominal CT scan demonstrated a small bowel obstruction with a suspected internal hernia *(Figure 1)*. Based on clinical and radiological findings, it was decided to perform an emergency diagnostic laparoscopy. A small bowel loop herniated into the preperitoneal space through a peritoneal defect was identified as the origin of the obstruction *(Figure 2)*. No evidence of loop distress was identified, so the hernia was reduced, and subsequent closure of the peritoneal defect with polyglactin 910 2-0 cross-stitches was done.

In the postoperative period, with good evolution, he presented gas channeling and two bowel movements; the nasogastric tube was removed; he tolerated the oral route and was discharged 48 hours after surgery. The

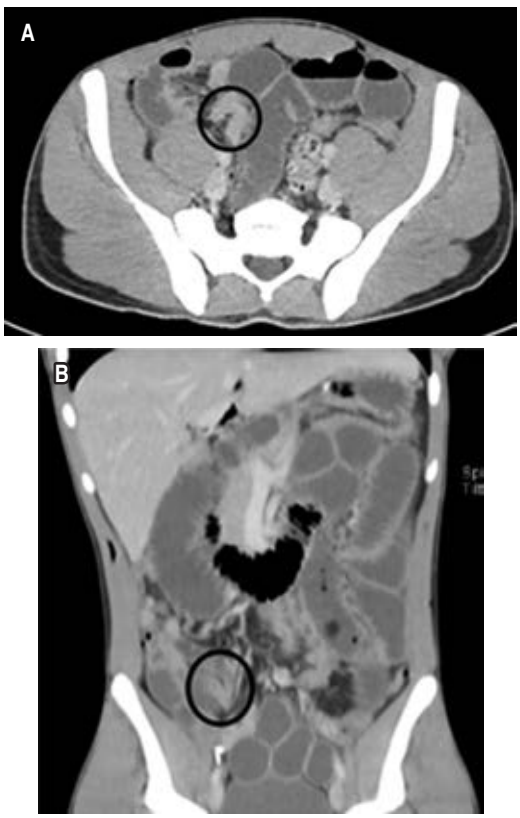

Figure 1: *Computed axial tomography scan of the abdomen with intravenous contrast. Dilatation of intestinal loops up to 3.5 cm in caliber and formation of hydro-aerial levels are seen. Areas of decreased caliber in the distal ileum at the right iliac fossa (circle) level are shown. Distal to this transition zone is a decrease in the caliber of the colonic frame. There is free fluid in the pelvis region.* ***A)*** *Axial section.* ***B)*** *Coronal section.*

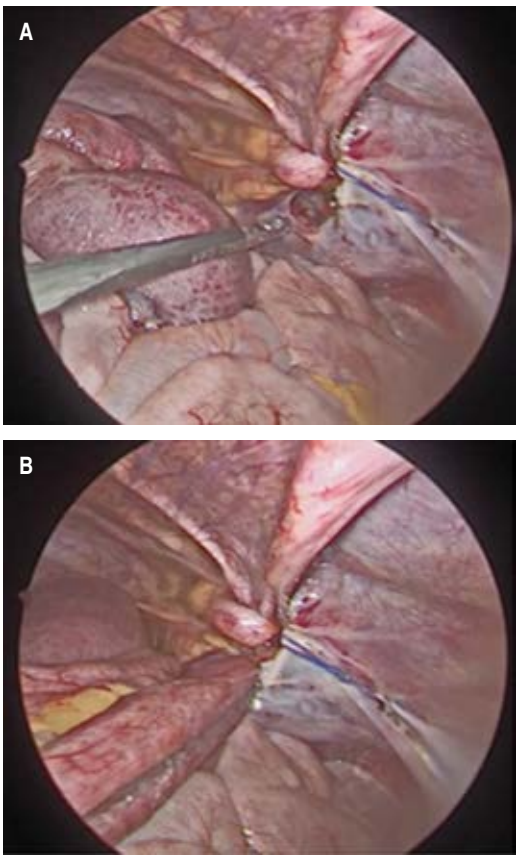

Figure 2: A) *Peritoneal defect is observed after the reduced herniated small bowel loop.* ***B)*** *A herniated small bowel loop in the preperitoneal space through the peritoneal defect is shown.*

patient was subsequently seen seven days after surgery. He had good evolution and tolerated the regular diet; he had no nausea, vomiting, or abdominal pain. Regular bowel movements were present without alarm data, for which he was discharged from the hospital.

DISCUSSION

This case represents a rare complication following inguinal hernia repair by TAPP. This technique is performed more frequently because it is a minimally invasive procedure, with less postoperative pain, better esthetics, and shorter hospital stay.[3]

Common causes of this complication are inadequate closure, port site herniation, and adhesions.[1] The presence of comorbidities and the size of the hernia defect are factors correlated with postoperative complications that often require reoperation.[4]

Risk factors for early surgical management include closed-loop obstruction, CT evidence of intestinal ischemia, recurrent small bowel obstruction, evidence of peritoneal irritation, or systemic inflammatory response syndrome.[1]

Good closure of the peritoneum during the initial procedure with particular attention to detail is essential to avoid preperitoneal hernias following TAPP. The cause of a hernia in the preperitoneal space can be attributed to insufficient closure of the peritoneal flap with loosening of the suture and subsequent displacement of the small bowel, or it can also herniate through a poorly closed peritoneal defect. The resulting small bowel obstruction that follows this causes symptoms of postoperative ileus.[5]

Many methods of closing the peritoneal flap include sutures, tacks, and staples. Fewer cases of a hernia in the preperitoneal space have been reported when using sutures for closure.[6] A bowel obstruction complication of laparoscopic inguinal hernia repair can be divided into adhesive or herniated disease. The one caused by herniation can be subdivided into early, due to peritoneal defects, or late, because of herniation at the port site.[2] In the case presented here, we can see that the cause was an early herniation caused by a peritoneal defect. Complications that could arise from failure to close peritoneal tears include exposure of the mesh to the bowel with a risk of bowel erosion, adhesions, internal hernia through the tear, and bowel obstruction.[2]

In addition, other surgical details related to reducing peritoneal tension become essential. The pneumoperitoneal pressure can be reduced to 8-10 mmHg by closing the peritoneal flap. Also, more peritoneum can be dissected from the cord structures inferiorly to the peritoneal reflection. Finally, carbon dioxide should be released slowly to avoid a sudden pressure difference between the abdominal cavity and the preperitoneal space.[7]

In the literature, it was found that closing the peritoneal flap with sutures reduces the risk of a hernia appearing in the preperitoneal space, which in our case was how the peritoneal

flap was initially closed; even so, had this complication but was also the reason why we closed the peritoneal defect again with sutures and no other material to prevent it from tearing again was used, and with which we were able to have a favorable evolution of the patient and not present this complication again.

CONCLUSIONS

In conclusion, this case shows an infrequent complication, corresponding to a bowel obstruction caused by a preperitoneal hernia during the early postoperative period related to a failure to close the peritoneal defect during the TAPP procedure.

As procedures such as TAPP are developed more frequently worldwide, uncommon complications become more frequent and must be considered in perioperative and postoperative management. This differential diagnosis was considered in our case, so we intervened quickly and had adequate management.

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