ISSN 1405-0099

VOLUME 44, No. 2 APRIL-JUNE 2022

Cirujano General

2022

Internet: http://www.amcg.org.mx
www.medigraphic.com/cirujanogeneral

Official Scientific Publication of the
ASOCIACIÓN MEXICANA DE CIRUGÍA GENERAL, A.C.
E-mail: revista@amcg.org.mx

Indexed at LILACS, BIREME-OPS

Cirujano General

Official Scientific Organ of the Mexican Association of
Mexican Association of General Surgery, A.C.

Cirujano General is the scientific communication organ of the Mexican Association of General Surgery, published quarterly.

Postage paid. Periodical publication; Registration No. 0540593. Characteristics 220251118.

Cirujano General is indexed in **Medigraphic Literatura Biomédica, PERIODICA, Índice de Revistas Latinoamericanas en Ciencias y Salud: Ciencia (Sociedad Iberoamericana de Información Científica).**

Designed, produced, and printed in Mexico by: graphimedic S.A. de C.V. Tels: 55-85-89-85-27 to 32. E-mail: emyc@medigraphic.com Printed in Mexico.

Available in full version at:: www.medigraphic.com/cirujanogeneral

Contents

Cirujano General

Mexican Association of General Surgery, A.C.

Directive Board 2021-2022

Contents

Cirujano General
April-June 2022
Vol. 44, no. 2

EDITORIAL

Importance of reading and following authors' instructions 65
Abilene Cirenia Escamilla-Ortiz

ORIGINAL ARTICLE

Subcutaneous endoscopic approach for ventral hernia repair with concurrent plication of rectus diastasis 67
Diego A Díaz, Alberto C Gordillo, Diego F Viteri, Jhony A Delgado

CLINICAL CASES

Multiple abscesses in the thorax and abdomen due to *Streptococcus anginosus:* A case report and literature review 73
Alberto Valdés-Castañeda, Óscar Cervantes-Gutiérrez, Marcos Jafif-Cojab, Ana de la Cajiga-León, Juan Pablo Arribas-Martin, Sebastián Guadarrama-Sistos Vázquez, Carlos Mancera-Steiner

Amebic liver abscess of rare localization 77
Roberto Antonio Alvarado-Hernández, Gerardo de Jesús Mayorga-Lira

Complications and long-term effects in a patient with atypical COVID-19 pneumonia: A case report 83
Edgardo Jiménez-Fuentes, Érika Barlandas-Quintana, Karla del Carmen Piña-Moreno, Asya Zubillaga, Christian Marcelo Carrión-Astudillo

Emphysematous gastritis secondary to gastric mucormycosis in a patient with COVID-19. A case report 87
Martín Islas-Torres, Ana Laura Castillo-Luna, José Juan Rodríguez-Moreno, Valeria Priscilla Rendón-Muñoz, José Gerardo Zamora-Inzuna, Albert Antonio Ibarra-Trejo

Mesenteric ischemia as a presentation of primary antiphospholipid syndrome in a male patient 93
Erick Zúñiga-Garza, Francisco Franco-López

Cirujano General
Abril-Junio 2022
Vol. 44, núm. 2

EDITORIAL

Importancia de leer y seguir instrucciones para autores 65
Abilene Cirenia Escamilla-Ortiz

ARTÍCULO ORIGINAL

Enfoque endoscópico subcutáneo para la reparación de hernias ventrales con plicatura concurrente de diástasis de rectos 67
Diego A Díaz, Alberto C Gordillo, Diego F Viteri, Jhony A Delgado

CASOS CLÍNICOS

Abscesos múltiples en tórax y abdomen por Streptococcus anginosus: reporte de caso y revisión de literatura 73
Alberto Valdés-Castañeda, Óscar Cervantes-Gutiérrez, Marcos Jafif-Cojab, Ana de la Cajiga-León, Juan Pablo Arribas-Martin, Sebastián Guadarrama-Sistos Vázquez, Carlos Mancera-Steiner

Absceso hepático amebiano de localización poco frecuente 77
Roberto Antonio Alvarado-Hernández, Gerardo de Jesús Mayorga-Lira

Complicaciones y efectos a largo plazo en paciente con neumonía atípica por COVID-19: reporte de caso clínico 83
Edgardo Jiménez-Fuentes, Érika Barlandas-Quintana, Karla del Carmen Piña-Moreno, Asya Zubillaga, Christian Marcelo Carrión-Astudillo

Gastritis enfisematosa secundaria a mucormicosis gástrica en paciente con COVID-19. Reporte de caso 87
Martín Islas-Torres, Ana Laura Castillo-Luna, José Juan Rodríguez-Moreno, Valeria Priscilla Rendón-Muñoz, José Gerardo Zamora-Inzuna, Albert Antonio Ibarra-Trejo

Isquemia mesentérica como presentación de síndrome antifosfolípido primario en paciente masculino 93
Erick Zúñiga-Garza, Francisco Franco-López

EDITORIAL
doi: 10.35366/109713

Cirujano General
April-June 2022
Vol. 44, no. 2 / p. 65-66

Importance of reading and following authors' instructions

Importancia de leer y seguir instrucciones para autores

Abilene Cirenia Escamilla-Ortiz*

The authors should know that publishing a manuscript takes a long way, from revising the article to its publication; an important part is to read and follow the authors' instructions of the journal in which they want to publish their manuscript, and at the same time see what kind of manuscripts the journal receives, as this can be a reason for rejection.[1]

It is suggested that they review and read manuscripts from the journal they want to publish to see the format.[1]

The editor(s) should not feel free pressured by the author since they want their manuscript to be published quickly, even if they send letters explaining that they are about to obtain an academic degree, promotion, qualification, etc.[1]

The author should be familiar with the manuscript submission platform since suggestions and corrections made by the reviewers, or the editor will be sent to the author in case of being accepted for review.[1]

In recent years, some authors, impatient to publish without waiting for the times that can take up to eight months, have opted to fall prey to predatory journals that offer to publish in less time and at a lower cost,[2] and should understand that there is no shortcut.

Nathan Efron compares the instructions for authors to the safety instructions on a flight; most do not listen or pay attention to them because they already know them or have heard them many times,[3] so he recommends adhering to the authors' instructions of the journal to which the manuscript is intended to be sent, this makes it more likely to be published, out of respect for the journal and the editor and finally out of pride.[3]

Some authors need help to read and adhere to the instructions for authors, probably because they trust that the editor will do his or her job, but in most cases, this is not the case, as their manuscript may be rejected from the start.

Editors or reviewers verify that authors have adhered to the instructions. Otherwise, it may be thought that the author is not a reader of that journal, or another journal has already rejected the manuscript.[4]

They should follow the instructions to know how many authors should be included according to the type of manuscript, format, typeface, figures, or images. The latter should follow the journal's requirements since they may not be original or may have been manipulated.[4]

Some journals, including Surgeon General, provide authors *with* a checklist to verify that the requested information has been fulfilled before submitting the article for review.

* Editor, Surgeon General. ORCID: orcid.org/0000-0001-5635-5845

How to cite: Escamilla-Ortiz AC. Importance of reading and following authors' instructions. Cir Gen. 2022; 44 (2): 65-66. https://dx.doi.org/10.35366/109713

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Correspondence:
Abilene Cirenia Escamilla-Ortiz
E-mail: escamillaoa@amcg.org.mx

ORIGINAL ARTICLE
doi: 10.35366/109714

Cirujano General
April-June 2022
Vol. 44, no. 2 / p. 67-72

Subcutaneous endoscopic approach for ventral hernia repair with concurrent plication of rectus diastasis

Enfoque endoscópico subcutáneo para la reparación de hernias ventrales con plicatura concurrente de diástasis de rectos

Diego A Díaz,[*] Alberto C Gordillo,[‡] Diego F Viteri,[‡] Jhony A Delgado[*]

Keywords:
rectus diastasis, ventral hernia, diastasis plication, endoscopic.

Palabras clave:
diástasis de los rectos, hernia ventral, plicatura de la diástasis, endoscópica.

ABSTRACT

Introduction: diastasis recti is a weakness of the alba line due to laxity in the aponeurosis that may be associated with abdominal wall hernias. When both are present, they must be surgically resolved. **Objectives:** to describe a minimally invasive subcutaneous technique for repairing ventral hernias with concurrent plication of rectus diastasis. **Material and methods:** this is a case series of seven patients between January 2019 and June 2020 who underwent endoscopic subcutaneous ventral hernia repair with concurrent diastasis recti plication. **Results:** the procedure was performed on seven patients, five men, and two women, with a mean age of 57.6 (38-70) years and a mean body mass index (BMI) of 26.7. The mean operative time was 174 (110-190) minutes. Hospital stays were two days. The mean defect size was 1.8 (1-2) cm, and the mean diastasis size was 3.5 (3-4) cm. After two weeks no postoperative complications (seroma, infection, hematoma). No recurrences have been reported in the medium-term follow-up (15 months). **Conclusion:** the subcutaneous endoscopic technique is a safe, reproducible, and effective alternative for patients with ventral hernias associated with rectus diastasis.

RESUMEN

Introducción: *la diástasis de los rectos es una debilidad de la línea alba debido a una laxitud en la aponeurosis que podría asociarse a hernias de la pared abdominal. Cuando ambas están presentes deben resolverse quirúrgicamente.* ***Objetivos:*** *describir una técnica mínimamente invasiva subcutánea para la reparación de hernias ventrales con plicatura concurrente de diástasis de los rectos.* ***Material y métodos:*** *ésta es una serie de casos de siete pacientes entre enero de 2019 y junio de 2020 que se sometieron a una reparación de hernias ventrales subcutáneas endoscópicas con plicatura concurrente de diástasis de los rectos.* ***Resultados:*** *el procedimiento se realizó en siete pacientes, cinco hombres y dos mujeres, edad media de 57.6 (38-70) años, índice de masa corporal (IMC) medio de 26.7. Media de tiempo operatorio de 174 (110-190) minutos. Estancia hospitalaria de dos días. Media del tamaño de los defectos 1.8 (1-2) cm, la media del tamaño de la diástasis 3.5 (3-4) cm. A las dos semanas no se reportaron complicaciones postoperatorias (seroma, infección, hematoma). Al seguimiento a mediano plazo (15 meses) no se han reportado recidivas.* ***Conclusión:*** *la técnica endoscópica subcutánea es una alternativa segura, reproducible y efectiva para pacientes con hernias ventrales asociadas con diástasis de los rectos.*

[*] Postgraduate Doctor of General Surgery R4, Pontificia Universidad Católica del Ecuador (PUCE).
[‡] General Surgery Attending Physician.

Hospital Quito No. 1 of the National Police, Quito, Ecuador.

Received: 05/16/2020
Accepted: 12/23/2022

INTRODUCTION

Diastasis recti (DR) is caused by the reduction of the cross-linked fibers that form the alba line of the abdominal wall with an increase in their length, generating a separation of both aponeuroses of the rectus abdominis muscles. This separation may present a laxity in the aponeurosis associated with abdominal wall hernias.[1,2] It is defined as the separation of the muscular borders of the midline greater than 2.2 cm;[2] it is not an

How to cite: Díaz DA, Gordillo AC, Viteri DF, Delgado JA. Subcutaneous endoscopic approach for ventral hernia repair with concurrent plication of rectus diastasis. Cir Gen. 2022; 44 (2): 67-72. https://dx.doi.org/10.35366/109714

uncommon condition and occurs more often in women.[3] Diastasis per se is not associated with symptoms such as pain, discomfort, or any complication; the main complaint is aesthetic. There is currently no consensus on the type of approach for surgical repair of DR; however, since it is associated with midline hernias, correction of both pathologies could be the best indication.[2]

Different surgical approaches have been described for treating DR with or without midline hernias for patients with excess skin or excessive weight loss; open surgery, laparoplasty, or laparoabdominoplasty with dermo lipectomy are the best options.[4] However, a scar from a large incision may give an unfavorable aesthetic result for patients without excess skin.[5] Minimally invasive techniques with rectus plication and midline hernia repair with mesh placement have been described.[6]

This study is aimed to describe a minimally invasive subcutaneous technique for repairing ventral hernias with concurrent plication of rectus diastasis.

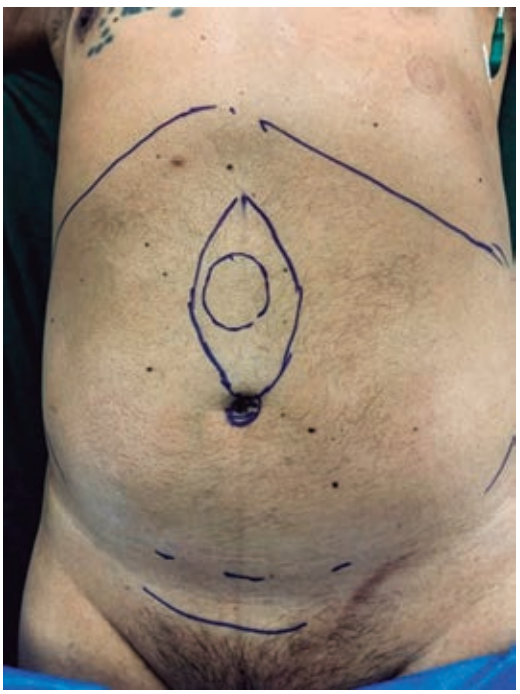

Figure 1: *Image of the marking of the hernial defects, the size of the diastasis recti, and the position of the endoscopic ports.*

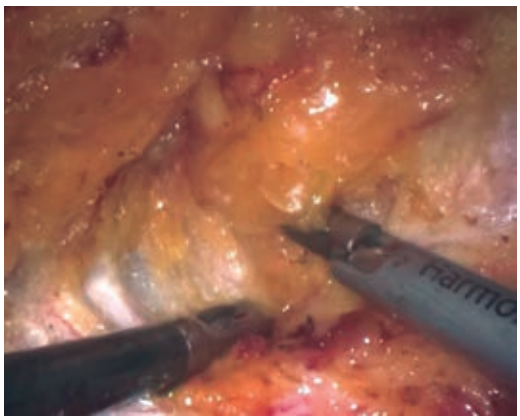

Figure 2: *Dissection of the subcutaneous cellular tissue separating it from the anterior aponeurosis of the rectus with harmonic scissors.*

MATERIAL AND METHODS

This is a case series at Hospital Quito No. 1 of the National Police general surgery service in Ecuador of seven patients between January 2019 and June 2020 who underwent endoscopic subcutaneous ventral hernia repair with a polypropylene mesh placement in the preperitoneal space and concurrent plication of diastasis recti. The procedure was indicated for patients with primary ventral hernias with concomitant diastasis recti. Patients with obesity, excess skin, a history of abdominoplasty, and coagulopathies were excluded.

Surgical technique

Under general anesthesia, the patient is placed in dorsal decubitus in the extended lithotomy position. The limits of the extent of the dissection are marked *(Figure 1)*. A transverse incision is made 1.5 to 2 cm above the pubis. Then, the subcutaneous tissue is dissected up to the anterior aponeurosis of the rectus abdominis; using digital dissection, the subcutaneous cellular tissue is separated from the aponeurosis superiorly and laterally to create an ideal space for the placement

of an 11 mm port for the optics through the suprapubic incision and two 5 mm working ports in both lower quadrants *(Figure 2)*. CO insufflation pressure$_2$ is maintained at 8-10 mmHg. Using harmonic scissors, subcutaneous dissection is performed from the suprapubic incision. The umbilicus is disinserted from the aponeurosis, and the dissection continued up to the xiphoid process and laterally to the semilunar line *(Figure 3)*. The defects are quickly identified, the hernial sac is dissected, and the contents are reduced to the abdominal cavity *(Figure 4)*. Once the dissection is completed, the diastasis of the rectus abdominis muscles is easily identified *(Figure 5)*. The mesh continues to be made according to the size of the defects and considers the principles of hernia repair. The

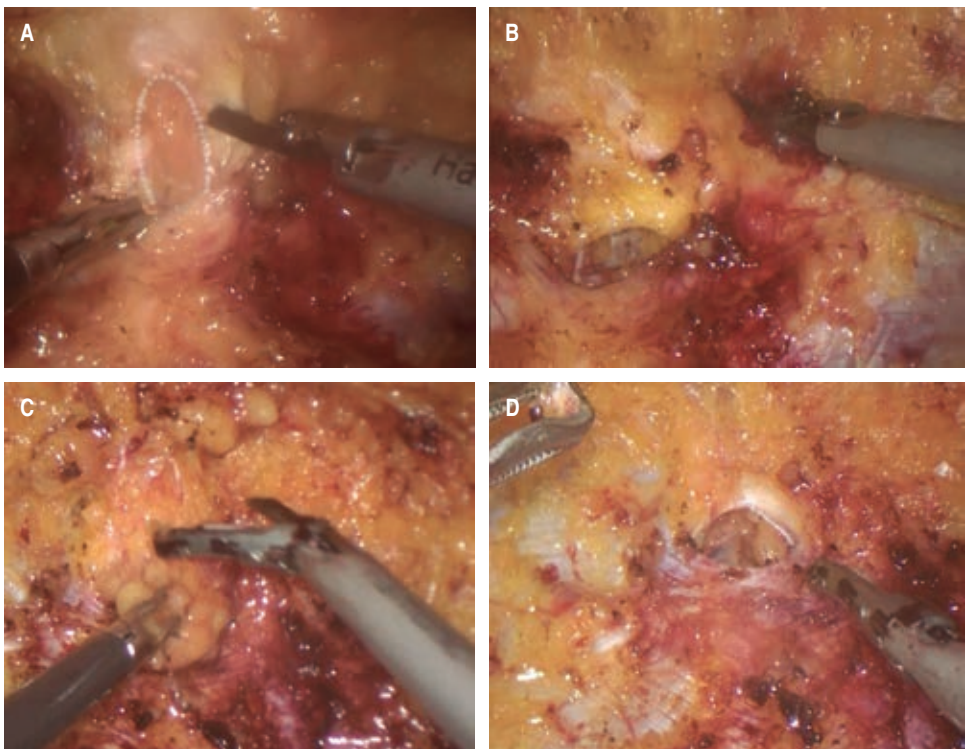

Figure 3:

A) *Umbilical hernial sac (white lines).* ***B)*** *Umbilical hernial defect.* ***C)*** *Epigastric hernial sac.* ***D)*** *Epigastric hernial defect.*

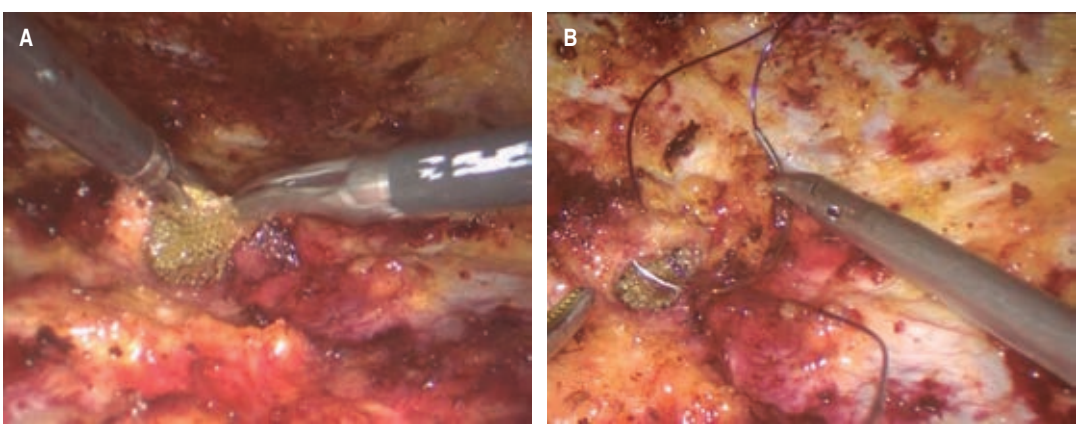

Figure 4: ***A)*** *Placement of the synthetic mesh in the preperitoneal space.* ***B)*** *Closure of the defect with a non-absorbable suture.*

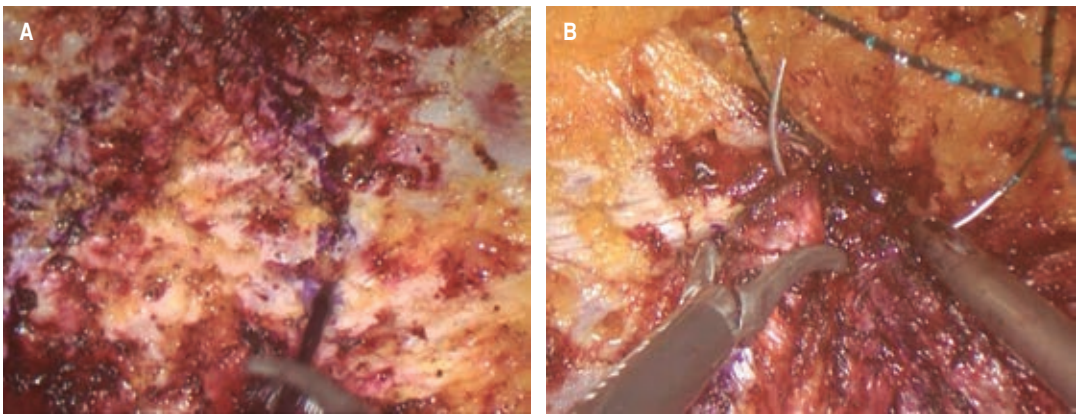

***Figure 5:** A) Diastasis marking. B) Plication of the rectus abdominis muscles.*

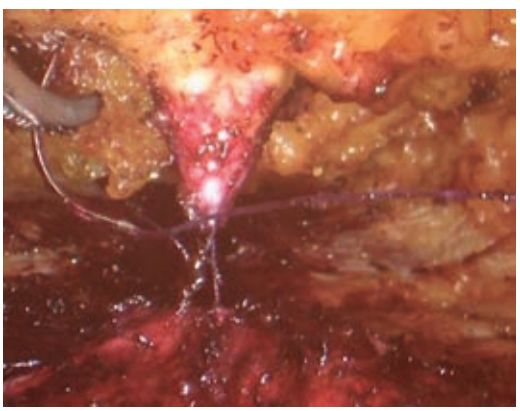

***Figure 6:** Reinsertion of the umbilicus.*

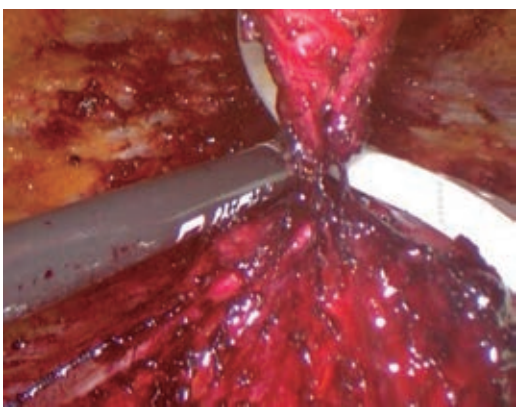

***Figure 7:** Placement of drainage.*

mesh is then placed in the preperitoneal space, and the defect is closed with a non-absorbable suture *(Figure 4)*. The marking of the diastasis is continued, and the plication proceeds, which should extend from the xiphoid process to 2-3 cm below the umbilicus. In this case, barbed sutures were used and are recommended to facilitate plication without losing traction *(Figure 5)*. The umbilicus is reattached to the muscular aponeurotic plane with one or two simple sutures *(Figure 6)*. An aspiration drain is introduced to prevent seroma formation and is externalized through a 5 mm incision *(Figure 7)*.

RESULTS

The procedure was performed on seven patients, five men and two women, with a mean age of 57.6 (38-70) years and a mean BMI of 26.7. The mean operative time was 174 (110-190) minutes. The hospital stay was two days. The mean defect size was 1.8 (1-2) cm, and the mean diastasis size 3.5 (3-4) cm. A postoperative girdle was placed after the procedure and maintained for 60 days. The drain was removed at 14 days with secretions < 20 ml/day. No postoperative complications (seroma, infection, hematoma) were reported at two weeks. During the medium-term follow-up (15 months), no recurrences were reported.

DISCUSSION

Diastasis recti (DR) is defined as the excessive separation between the two bellies of the rectus

abdominis, which can occur at any point of the alba line, from the xiphoid process to the pubic symphysis.[7] DR is frequent and predominant in women, usually in pregnancy, and returns to its normal condition after delivery; however, after 12 months, one-third of these patients, still present it.[7] Hormonal changes cause changes in the tissues, producing tissue laxity at the level of the alba line, this hormonal effect persists up to three months after delivery, so if this condition persists at this time, it can be said that a DR exists.[8]

The association of midline hernias and DR is not uncommon; DR has been diagnosed in 45% of patients with small midline hernias (> 2 cm); of these patients, 31% who underwent suture repair had a higher recurrence compared to patients who had no association with a DR at a 30-month follow-up.[9]

The surgical indication remains controversial, and there is still no standard method for repairing midline hernias associated with DR. Several options for the joint treatment of midline hernias associated with DR have been described, ranging from open, laparoscopic, hybrid, or endoscopic techniques.[5] For obese patients and patients with excess skin, dermo lipectomy is the indicated method;[10] However, there are patients without excess skin in whom a pronounced scar may be an unfavorable aesthetic outcome. In these cases, the endoscopic option is a good alternative, as it presents several advantages, such as minimizing incisions, better esthetic results, less postoperative pain, and less wound infection.[11]

One of the main complications reported in some series is seroma, which has occurred in up to 27% of cases; in this series, no seroma has been reported. Several authors indicate that most seromas are spontaneously reabsorbed in > 50%, and drainage by puncture is the recommended technique in the seroma is not reabsorbed.[2,3] There is no indication of the drainage removal time and its production; however, in this series, the indication for removing the drainage was 14 days with secretions of less than 20 ml/day. In the 15-month follow-up, no recurrences have been reported.

CONCLUSION

The endoscopic subcutaneous technique is a safe, reproducible, and effective alternative for patients with ventral hernias associated with rectus diastasis.

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Ethical considerations and responsibility: ethical considerations were taken into account with the patients, and we have their authorization to carry out this study.

Financing: no financial support was received for the realization of this work.

Disclosure: the authors declare no conflict of interest.

Correspondence:
Diego Fernando Viteri-Cevallos
E-mail: diegoviteri83@hotmail.com

CLINICAL CASE
doi: 10.35366/109715

Cirujano General
April-June 2022
Vol. 44, no. 2 / p. 73-76

Multiple abscesses in the thorax and abdomen due to *Streptococcus anginosus:* A case report and literature review

Abscesos múltiples en tórax y abdomen por Streptococcus anginosus: *reporte de caso y revisión de literatura*

Alberto Valdés-Castañeda,[*] Óscar Cervantes-Gutiérrez,[*] Marcos Jafif-Cojab,[*] Ana de la Cajiga-León,[‡] Juan Pablo Arribas-Martin,[*] Sebastián Guadarrama-Sistos Vázquez,[§] Carlos Mancera-Steiner[*]

Keywords: thoracic abscess, abdominal abscess, *Streptococcus anginosus*, infection, a case report.

Palabras clave: *absceso torácico, absceso abdominal,* Streptococcus anginosus, *infección, reporte de caso.*

ABSTRACT

Introduction: intra-abdominal abscesses occur mainly secondary to a surgical intervention that, for some reason, becomes contaminated, whether because of appendicitis, perforations, or trauma. **Case report:** we present a case report of abscesses associated with *Streptococcus anginosus* in an adult male patient who underwent fundoplication by laparoscopy. **Results:** six days after laparoscopic surgery, the patient presented data of infection of abdominal origin with systemic involvement, and the presence of abscesses in the abdomen and thorax were identified and satisfactorily treated. **Conclusions:** new agents capable of dissemination by different routes have been identified; *Streptococcus anginosus* is becoming increasingly relevant when establishing a diagnosis and line of treatment.

RESUMEN

Introducción: *los abscesos intraabdominales se presentan principalmente de manera secundaria a una intervención quirúrgica que por alguna razón se contamina, puede ser como resultado de una apendicitis, perforaciones o trauma.* ***Reporte de caso:*** *se presenta un reporte de caso de abscesos asociados a* Streptococcus anginosus *en paciente masculino adulto que fue sometido a funduplicatura por laparoscopia.* ***Resultados:*** *seis días posteriores a intervención quirúrgica laparoscópica se presenta paciente con datos de infección de origen abdominal con compromiso sistémico, identificándose y tratándose de manera satisfactoria la presencia de abscesos en abdomen y tórax.* ***Conclusión:*** *se han identificado nuevos agentes capaces de diseminación por diferentes vías, el* Streptococcus anginosus *se vuelve cada vez más relevante al establecer un diagnóstico y línea de tratamiento.*

[*] Department of General Surgery, Hospital Ángeles Lomas.
[‡] Department of Anesthesiology, Hospital Ángeles Lomas.
[§] Mexican School of Medicine, Universidad La Salle.

Received: 07/27/2021
Accepted: 12/23/2022

INTRODUCTION

Intraabdominal abscesses are mainly generated from contaminated or dirty surgeries such as diverticulitis, appendicitis, intestinal perforations, and penetrating abdominal trauma, among others.[1] Other causes of abdominal abscesses are those caused by bacterial translocation from contiguous foci, or hematogenous or lymphatic dissemination from a distant focus.[2]

The *Streptococcus anginosus* group (SAG) has three species (*S. intermedius, S. constellatus, and S. anginosus*). These microorganisms have low bacterial virulence and are generally found as oral and genitourinary bacterial flora.[3] However, multiple reports in the literature demonstrate these organisms' ability

How to cite: Valdés-Castañeda A, Cervantes-Gutiérrez Ó, Jafif-Cojab M, de la Cajiga-León A, Arribas-Martin JP, Guadarrama-Sistos VS, et al. Multiple abscesses in the thorax and abdomen due to *Streptococcus anginosus:* A case report and literature review. Cir Gen. 2022; 44 (2): 73-76. https://dx.doi.org/10.35366/109715

to form distant abscesses by hematogenous dissemination.[1]

In this work, we report through the presentation of a clinical case in a private hospital in the State of Mexico, Mexico, in which a patient who had undergone laparoscopic fundoplication showed abscesses in the thorax and abdomen, where *S. anginosus* was identified. It also emphasizes the importance of detecting this pathogen, which has increased its incidence in recent years.

CLINICAL CASE

We present the case of a 51-year-old male patient diagnosed with gastroesophageal reflux disease, whose only comorbidity was obesity, scheduled for fundoplication by laparoscopy. Preclinical examinations within normal parameters revealed complex airway data reported by anesthesiology. A prophylactic antibiotic was administered consistent of ceftriaxone 1 g in a single dose. The surgery was successfully performed, and only three attempts for intubation were required without incident. On the first postoperative day, an esophagogram with a water-soluble contrast medium was performed, which showed the integrity of the digestive tract without leakage of the contrast medium into the abdominal cavity.

On the second postoperative day, the patient was discharged. Two days later, while receiving analgesic therapy with nonsteroidal anti-inflammatory drugs, he presented to the emergency department with a temperature of 39.5 ºC and tachycardia of 115 beats per minute. Physical examination revealed a soft, depressible, non-painful abdomen, with no evidence of peritoneal irritation. The rest of the examination was normal. A thoracoabdominal computed tomography (CT) scan showed a basal consolidation in the left lung field and the expectoration culture reported *Streptococcus anginosus*. Treatment with broad-spectrum antibiotics, such as ceftriaxone, and metronidazole was initiated. On the third day, he started with anorexia, and it was decided to take a new CT scan identifying a 300 ml subhepatic abscess. It was decided to drain the abscess and wash the abdominal cavity by laparoscopy without incident. Five days later, he presented fever of 38.5 ºC, so it was decided to perform a new imaging study identifying multiple abscesses in the thorax and abdomen, which were drained by CT-guided puncture without incident.

With favorable evolution, the patient was discharged five days later with improved symptoms, without fever or alarm data. Antibiotic treatment with imipenem and linezolid was continued, and the patient evolved satisfactorily.

DISCUSSION

Streptococcus anginosus (SAG), first described by Andrewes and Horder in 1906 as a variant of *Streptococcus pyogenes,* constitutes the *S. anginosus* family along with *Streptococcus intermedius* and *Streptococcus constellatus*, also known as the *Streptococcus milleri* group.[4] Infections by these bacteria have reported significant variability in their clinical presentations.[5] These organisms are microaerophilic, catalase-negative, gram-positive cocci that form tiny colonies and a characteristic caramel odor due to the production of the metabolite diacetyl when cultured on blood agar.[4]

These microorganisms are low-virulence bacteria that exist as commensals in the oronasal flora, gingival sulci, gastrointestinal tract, and urogenital tract of humans.[6] *S. anginosus* can spread to the blood in individuals with poor oral hygiene in cases of oral infections such as gingivitis and dental abscesses that may develop after the loss of the mucosal unit. This can lead to infections manifesting mainly as brain and liver abscesses or peritonitis.[1]

Intra-abdominal abscesses usually develop due to abdominal surgery for pathologies such as diverticulitis and appendicitis or for biliary disorders, pancreatitis, or organ perforations. Penetrating abdominal trauma can also cause *S. anginosus* abscesses. Abscesses caused by infectious bacteremia reaching the abdomen from a distant source are rare.[1]

Several authors have reported cases of *S. anginosus* infection. J. Tomas describes the association of *S. anginosus* as the causative germ of a pancreatic abscess in a patient with poor oral hygiene after multiple dental extractions.[2] J. Cooper describes a case of purulent pericarditis due to *S. anginosus* with contiguous subdiaphragmatic and hepatic collections, which resolved with initial subxiphoid pericardial drainage.[4] G. Simone et al. report a single case of disseminated infection of the *S. anginosus* group with multiple pyogenic cerebral, hepatic, and pulmonary lesions.[7]

A retrospective systematic review of 52 episodes of infection in patients aged 0-18 years by M. Furuichi et al. describes the sites of infection associated with each SAG species among pediatric patients: skin and soft tissue (35%), gastrointestinal tract (21%), genitourinary tract (21%), head and neck (19%), and central nervous system (2%).[8] *S. anginosus* is most frequently isolated from the genitourinary tract and in blood cultures.[9] *S. constellatus* is responsible for most skin and soft tissue infections and abscesses, and *S. intermedius* is isolated mainly in head and neck infections and brain abscesses.[8,9] Polymicrobial infections are the most common, and more than 70% of patients infected with *S. anginosus* and *S. constellatus* are co-infected with obligate anaerobes *(Bacteroides* spp*)*.[9]

Susceptibility of *S. anginosus* to penicillin, ampicillin, cefotaxime, erythromycin, clindamycin, levofloxacin, and vancomycin has been reported.[9] O. Kobo et al. analyzed the association between SAG species and the presence of pyogenic infection through a retrospective, observational cohort study between the years 2009 and 2015, concluding that *S. intermedius* has the most potential to cause infections involving abscess formation or other deep infections. *S. constellatus* and *S. anginosus* caused bacteremia without an associated pyogenic infection more frequently than *S. intermedius*.[3]

The acidic environment is one of the most common stressors for bacteria in infected tissues, so they have mechanisms to thrive even in these environments.[10]

S. anginosus has aciduric properties like those of *S. mutans* or *S. pyogenes*, so the acid tolerance of this microorganism can facilitate the infection of the oral cavity or gastrointestinal organs, causing chronic inflammation and consequently causing infective endocarditis and abscesses in various sites of the body including the thorax and abdomen as observed in the case.[10]

CONCLUSIONS

Species of the SAG group have been identified more frequently and recognized as emerging pathogens in the formation of disseminated infections by the hematogenous route.

However, although these species have been shown to exhibit various factors that promote their virulence, they have yet to acquire much interest from the medical and scientific community.

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Ethical considerations and responsibility: the authors declare that they followed the protocols of their work center on the publication of patient data, safeguarding their right to privacy through the confidentiality of their data.

Funding: no financial support was received for this work.

Disclosure: the authors declare no conflict of interest in carrying out the work.

Correspondence:
Sebastián Guadarrama-Sistos Vázquez
E-mail: sebastian.guadarramasistos.v@gmail.com

CLINICAL CASE
doi: 10.35366/109716

Cirujano General
April-June 2022
Vol. 44, no. 2 / p. 77-82

Amebic liver abscess of rare localization

Absceso hepático amebiano de localización poco frecuente

Roberto Antonio Alvarado-Hernández,* Gerardo de Jesús Mayorga-Lira‡

Keywords:
liver abscess, drainage, laparotomy.

Palabras clave:
absceso hepático, drenaje, laparotomía.

ABSTRACT

This is a 38-year-old male patient evaluated in the emergency department for abdominal pain with no relevant history; he starts his current condition with pain in the epigastrium, radiating to the right flank, aggravated by food intake. A computed tomography was performed, which showed a large amount of fluid in the left pleural space that conditioned passive atelectasis of the lower lobe; a lesion with heterogeneous density was detected in the liver, in addition to an increase in the left hepatic lobe. The lesion was drained, and a ruptured liver abscess was found in the peritoneal cavity; about three liters of purulent hematic liquid with the appearance of "anchovy paste" were obtained. A cytological and cytochemical study of the drained liquid was performed and *Entamoeba histolytica* was isolated.

RESUMEN

Se trata de paciente masculino de 38 años de edad, el cual es valorado en el servicio de urgencias por dolor abdominal, sin antecedentes de relevancia, inicia padecimiento actual con dolor en epigastrio, irradiado a flanco derecho, agravado con la ingesta de alimentos. Se realiza una tomografía computarizada, la cual mostró una importante cantidad de líquido en el espacio pleural izquierdo que condicionó una atelectasia pasiva del lóbulo inferior, se detecta en el hígado una lesión con densidad heterogénea, además de incremento del lóbulo hepático izquierdo. Se procede al drenaje de la lesión encontrando un absceso hepático roto a cavidad peritoneal, se obtienen cerca de tres litros de líquido purulento hemático con aspecto de "pasta de anchoas". Se realiza estudio citológico y citoquímico del líquido drenado, aislando Entamoeba histolytica.

INTRODUCTION

The infection by *E. histolytica* is highly endemic and very common in our environment;[1-3] it is usually associated with poor sanitary conditions, and a large percentage of infected people are carriers,[4,5] so it should be suspected in patients who present pain in the right upper quadrant of the abdomen, fever, hepatomegaly, and liver abscess.[1,6,7]

E. histolytica is named for its lytic effect on the surrounding cells.[2,8] Under scanning electron microscopy, it has been demonstrated that the main organelles involved in the pathological response are the lysis of nuclei and cytoplasm.[6] The amoeba binds to host cells through galactose lectin bonds, and in the initial stages, tissue invasion is caused by the action of proteases from the trophozoites, which degrade fibronectin and laminin, components of the extracellular matrix triggering an innate immune response. The lysis of the cell is caused by the formation of amoeba-pores created by the action of phospholipases; this cycle is the one that will end in the formation of an abscess.[2,8,9]

It is common in all age groups, and slightly more frequent in men aged 20-40 years, predominantly in countries with tropical climates.[1,4,5] Gender predominance is thought to be associated with alcohol consumption, as alcohol triggers Kupffer cell dysfunction and inappropriate cellular and humoral responses.[10]

* Fourth-year resident, general surgery.
‡ Chief undergraduate medical intern.

University Hospital "Dr. Joaquín del Valle Sánchez", Torreón, Coahuila.

Received: 03/30/2021
Accepted: 12/23/2022

How to cite: Alvarado-Hernández RA, Mayorga-Lira GJ. Amebic liver abscess of rare localization. Cir Gen. 2022; 44 (2): 77-82. https://dx.doi.org/10.35366/109716

The clinical manifestations usually appear four months after exposure to an endemic area,[4,5] and vary with the symptoms' severity and the abscess's location. In right lobe abscesses it is common to present with sudden onset of fever, accompanied by chills, predominantly night sweats, as well as constant and intense pain in the right hypochondrium radiating to the right scapular and deltoid region,[7] which is aggravated by coughing, while in left lobe abscesses patients frequently report epigastralgia radiating to the left deltoid region.[10] Approximately 30% of patients develop a non-productive cough, anorexia, nausea, vomiting, diarrhea, and weight loss.[1] In both cases, hepatomegaly and pain on palpation of the hypochondrium are very common, whereas jaundice is seen in less than 10%. Left lobe lesions represent only 35%, have a worse prognosis,[1,7,10] and a vast rate of complications due to their extrahepatic extension. The most affected are the peritoneum, great vessels, pericardium, pleura, bronchial tree, and lungs.[10]

Almost all patients present leukocytosis, and it is estimated that approximately 5% of cases developed leukemoid reactions;[1] alkaline phosphatase is elevated in more than 50% of patients and is considered one of the most reliable markers of liver abscess.[9] Mild anemia is present in one in four patients,[6] and hyperbilirubinemia is found in about one-third, with elevation of aminotransferases and hypoalbuminemia being uncommon.[1,6] Anti-ameba antibodies are usually present in 90% of patients, reaching their peak in the third month, and are detectable nine months after abscess resolution.[4] Indirect hemagglutination tests are considered to be the most sensitive and specific, with a cutoff considered to be 1:512, while in non-endemic areas, it is 1:256.[8] The imaging method of choice is ultrasonography, which shows single, space-occupying lesions with well-defined margins, well-delimited hypointense lesions on CT scan, elevation of the right hemidiaphragm, presence of ileus, and an anomalous distribution of gas on chest X-ray.[1,4,5,10]

PRESENTATION OF THE CASE

A 38-year-old male patient was admitted to the emergency department with a diagnosis of abdominal pain under study to rule out intestinal obstruction versus pancreatitis.

He is a merchant selling fruits and vegetables, originally from the city of Lerdo, Durango, Mexico. He had a history of smoking that suspended three months ago at a rate of three cigarettes a day. He denied any chronic degenerative diseases and surgical procedures, hypoxia, and weight loss of two months of evolution, and had no history of dysentery.

The patient began 15 days before admission with burning colicky abdominal pain of 7/10 intensity located in the epigastrium, radiating to the right flank and mesogastrium, exacerbated by food intake, managed with paracetamol and omeprazole with mild attenuation of pain. It was associated with asthenia, abdominal distension, oral intolerance, constipation, and hypoxia three days before admission, without fever.

Physical examination on admission found him conscious, with a Glasgow score of 15 points, oriented, with an antalgic gait on admission, diaphoretic with pallor of skin and integuments. He had an increased respiratory rate and decreased O_2 saturation to 90% on room air requiring supplemental oxygen support. His left hemithorax was hypo ventilated at its base with decreased vocal vibrations and dullness to percussion. Cardiac examination showed a rhythmic heart with increased frequency. His abdomen distended, painful to superficial and deep palpation in a generalized way, with muscular resistance and positive rebound, absent peristalsis, and dullness to percussion. His genitalia showed the presence of a Foley catheter, discharging a concentrated urine. The rectal examination showed an empty rectal ampoule. On admission to the hospital, the patient presented laboratory results taken at another facility that showed leukocytosis of 21,000/mm^3, thrombocytosis of 825,000 mm^3, and glucose of 230 mg/dl. On admission to the emergency department new lab tests were taken that reported hemoglobin (Hb) 11.7

g/dl, hematocrit of 37.6%, white blood cells of 15,900/mm^3, neutrophil count of 79%, bands 2%, platelets of 858,000/mm^3, glucose of 270 g/dl, creatinine of 0.4 mg/dl. His serum electrolytes were Na 130 mEq/l, Ca 7.8 mg/dL, P 4.6 mg/dl, Cl 89 mEq/l, K 4.9 mEq/l. To rule out pancreatitis the pancreatic enzymes were measured and reported amylase 33 and lipase 36. Liver function tests showed a TB 7.3 mg/dl, IB 5.5 mg/dl, DB 1.8 mg/dl, albumin 2.7 g/dl, TGO 43 U/l, TGP 28 U/l, GGT 225 U/l, AP 203 U/l, LDH 288 U/l. His arterial blood gases showed a pH of 7.52, pCO_2 of 41, pO_2 of 72, of HCO_3 27.2 mmol/l, BE +3, O_2 Sat 96%, and lactate of 1.59.

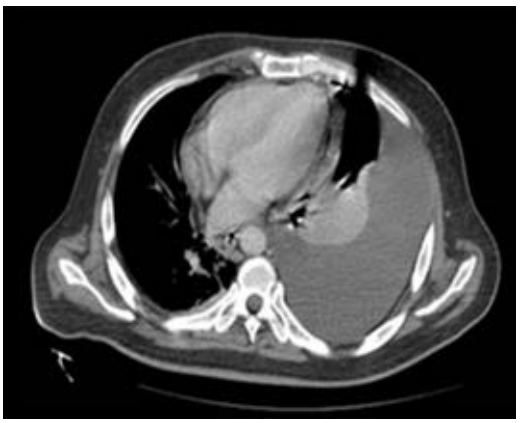

Figure 1: *Abdominal non-contrast computed axial tomography scan showing a significant left pleural effusion.*

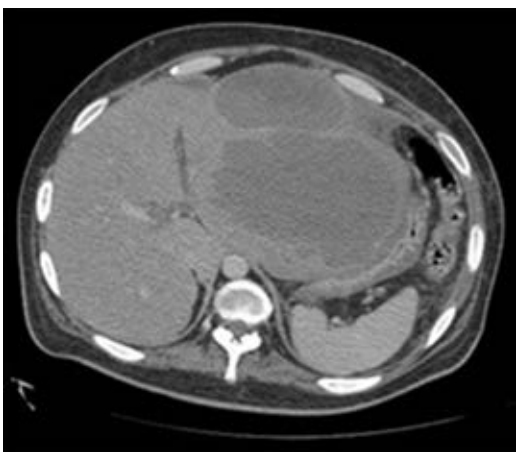

Figure 2: *An abdominal non-contrast computed axial tomography scan at the level of the spleen showing a hypodense, multilobulated image.*

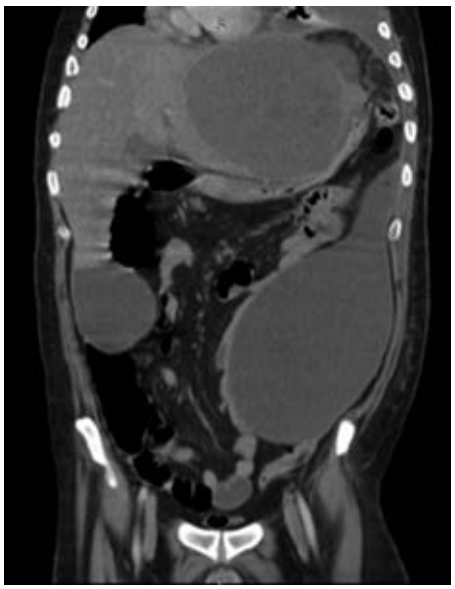

Figure 3: *Coronal section of a computed axial tomography scan showing displacement of structures and a space-occupying lesion in the left flank.*

A cytochemical study of pleural fluid showed a hematic color, cloudy appearance, pH 6.9, glucose 95, protein 3.2, albumin 1.7, LDH 2,098; the sediment had countless erythrocytes, moderate epithelial cells, abundant bacteria, white blood cells 87.3, polymorphonuclear cells 36, mononuclear cells 64, and abundant bacteria.

An anteroposterior (AP) chest X-ray was performed, in which a left pleural effusion covering more than 50% was visualized. A computed tomography scan showed a significant amount of fluid in the left pleural space that conditioned a passive atelectasis of the lower lobe *(Figure 1)*; the liver was found with heterogeneous density with an increase of the left hepatic lobe secondary to the presence of a heterogeneous hypodense lesion with lobulated borders, showing peripheral reinforcement and some internal septa covering segments II, III, IVa, and IVb measuring approximately 20 × 14.5 × 12.5 cm *(Figure 2)*, conditioning posterior displacement and compression of the stomach

and pancreas and caudal displacement of the intestinal loops *(Figures 3 and 4)*. In the lower pole of the right hepatic lobe adjacent to segment IV, a homogeneous cystic lesion with peripheral enhancement was identified after intravenous contrast administration, measuring 25 × 27 × 13 cm in its major axes. A Foley tube and nasogastric tube were placed as initial management draining a liquid of gastric characteristics (30 ml). A central catheter was placed and verified by radiography. Imaging studies were requested. Fasting was ordered and fluid therapy, antibiotic therapy with ceftriaxone and metronidazole, pain management, glycemic control, and gastric protection were started. When visualizing the image of the left pleural effusion, it was decided to perform a thoracentesis, obtaining a thick exudate with a chocolate appearance. When the tomography was available, urgent surgical treatment was performed.

Through a left endo-pleural probe one liter of serous fluid with serous characteristics was obtained. During the surgery, a ruptured hepatic abscess was found in the peritoneal cavity, obtaining about three liters of purulent hematic liquid with the appearance of "anchovy paste" *(Figure 5)*. The abscess capsule located in the left hepatic lobe segments 2, 3, and 4, approximately 10 cm in diameter, was visualized as extending towards the diaphragm without invading it *(Figure 6)*. Exhaustive lavage of the

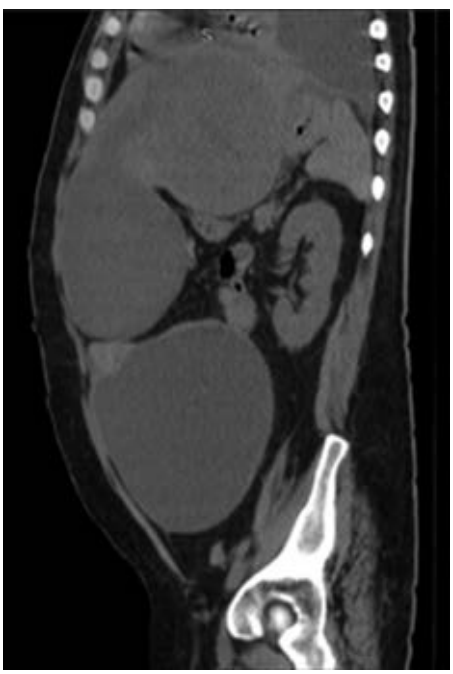

Figure 4: *Sagittal section of a computed axial tomography scan showing a space-occupying lesion and displacement of retroperitoneal structures.*

Figure 5: *Total aspirated contents from the abdominal cavity.*

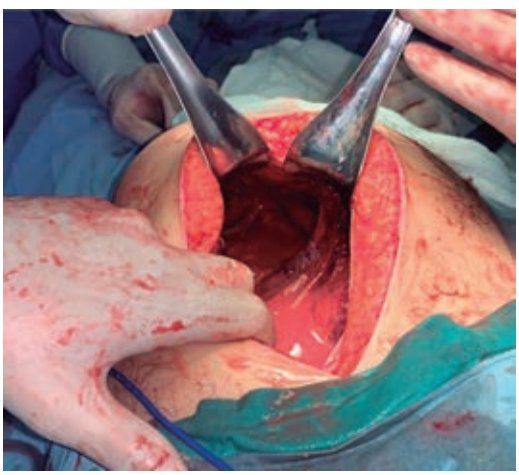

Figure 6: *Image of the abdominal cavity showing the capsule of the ruptured abscess and the diaphragmatic dome.*

peritoneal cavity was performed without finding any involvement of neighboring organs. A Saratoga drainage directed to the abscess capsule and Penrose drainage directed to the pelvic cavity *(Figures 5, 7 and 8)* was placed.

CONCLUSIONS

Although intestinal amebiasis continues to be an endemic disease, its prevalence has not been well clarified in our country[3] because of the low reporting of cases and a large number of asymptomatic carriers.[5] The complication rate of this disease with extraintestinal manifestations is very low.[1] The evolution of this patient is interesting because he presented a large abscess in an uncommon location,[4] which represents a surgical challenge due to many anatomical structures involved.

A satisfactory result was achieved after one month of medical treatment, follow-up in consultation, and subsequent rehabilitation with the patient's full recovery.

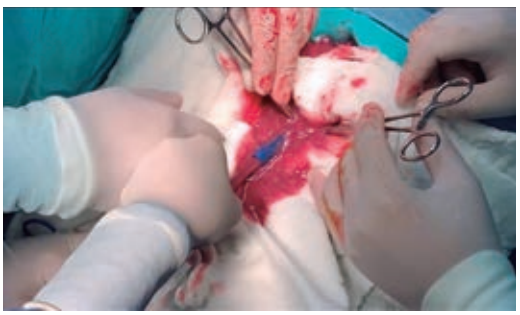

Figure 7: *Outflow of purulent material on opening the peritoneal cavity.*

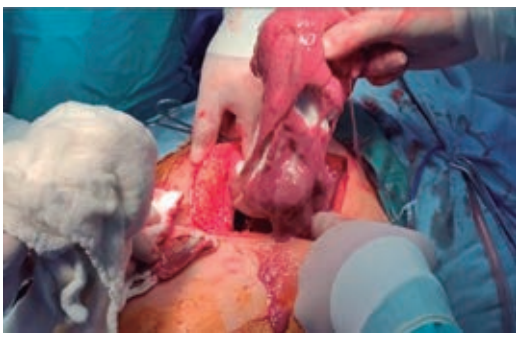

Figure 8: *Removal of purulent material from the abdominal cavity.*

ACKNOWLEDGMENTS

Special thanks to the General Surgery Service of the Hospital Universitario Torreón "Dr. Joaquín del Valle Sánchez"; to its residents for involving undergraduate interns in research and continuing education, showing the virtues of teamwork; to the outpatient, operating room, and auxiliary staff; as well as to the chief of the General Surgery Service of this hospital for his dedication to the academic training of his residents and interns.

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Ethical considerations and responsibility: the authors declare that they followed the protocols of their work center on the publication of patient data, safeguarding their right to privacy through the confidentiality of their data.

Funding: no financial support was received for this work.

Disclosure: the authors declare no conflict of interest in carrying out the work.

Correspondence:
Roberto Antonio Alvarado-Hernández, MD
E-mail: ra_ah17@hotmail.com

CLINICAL CASE
doi: 10.35366/109717

Cirujano General
April-June 2022
Vol. 44, no. 2 / p. 83-86

Complications and long-term effects in a patient with atypical COVID-19 pneumonia: A case report

Complicaciones y efectos a largo plazo en paciente con neumonía atípica por COVID-19: reporte de caso clínico

Edgardo Jiménez-Fuentes,[*] Érika Barlandas-Quintana,[‡] Karla del Carmen Piña-Moreno,[‡] Asya Zubillaga,[‡] Christian Marcelo Carrión-Astudillo[‡]

Keywords: COVID-19, SARS-CoV-2, pulmonary complications.

Palabras clave: *COVID-19, SARS-CoV-2, complicaciones pulmonares.*

ABSTRACT

Introduction: the type 2 coronavirus infection causing severe acute respiratory syndrome (SARS-CoV-2) has spread rapidly, resulting in a pandemic causing severe disease in thousands of patients worldwide. Although the number of articles on this pathology is growing exponentially, few published clinical cases describe long-term pulmonary complications. **Case report:** a 57-year-old male had right pleural effusion secondary to coronavirus disease of 2019 (COVID-19); an endo pleural tube was placed without achieving complete lung expansion. He underwent an exploratory thoracotomy that revealed necrotizing pneumonia, bronchopleural fistula of the right middle lobe, and pachypleuritis as late pulmonary complications of SARS-CoV-2 infection. **Conclusion:** further research is required regarding the significant variability of complications due to COVID-19 and to consider surgical treatment for selected patients.

RESUMEN

Introducción: *la infección por coronavirus de tipo 2 causante del síndrome respiratorio agudo severo (SARS-CoV-2) se ha propagado rápidamente resultando en una pandemia, condicionando afección grave en miles de pacientes alrededor del mundo. Aunque el número de artículos sobre esta patología crece exponencialmente, hay pocos casos clínicos publicados que describen complicaciones pulmonares a largo plazo.* ***Reporte de caso:*** *masculino de 57 años con derrame pleural derecho secundario a enfermedad por coronavirus de 2019 (COVID-19), se coloca sonda endopleural sin lograr expansión pulmonar completa. Se presenta a toracotomía exploradora que revela neumonía necrosante, fístula broncopleural del lóbulo medio derecho y paquipleuritis como complicaciones pulmonares tardías de infección por SARS-CoV-2.* ***Conclusión:*** *se requiere continuar una ardua investigación con respecto a la gran variabilidad de complicaciones por COVID-19 y plantear tratamiento quirúrgico para pacientes seleccionados.*

[*] General Surgery Resident. Department of General and Endoscopic Surgery.
[‡] Attending Physician. Chief of the Pneumology and Thoracic Surgery Service, Instituto Nacional de Cancerología.

General Hospital "Dr. Manuel Gea González", Ministry of Health (SSA), Mexico City.

Received: 03/30/2021
Accepted: 12/23/2022

INTRODUCTION

In late 2019 a new coronavirus was identified as a cause of atypical pneumonia in a group of patients in Wuhan, China. This microorganism has since spread rapidly, resulting in a pandemic. Such infection was designated by the World Health Organization with the term COVID-19 disease (i.e., coronavirus disease 2019).[1] The virus that causes COVID-19 is called severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The increased morbidity and mortality from COVID-19 are mainly due to acute viral pneumonitis that progresses to acute respiratory distress syndrome (ARDS). The case of a patient with prolonged hospitalization and intubation secondary to COVID-19 disease is presented, as well as the pulmonary complications observed and their surgical resolution.

How to cite: Jiménez-Fuentes E, Barlandas-Quintana É, Piña-Moreno KC, Zubillaga A, Carrión-Astudillo CM. Complications and long-term effects in a patient with atypical COVID-19 pneumonia: A case report. Cir Gen. 2022; 44 (2): 83-86. https://dx.doi.org/10.35366/109717

CLINICAL CASE PRESENTATION

A 57-year-old male with a history of long-standing systemic arterial hypertension is treated with losartan 50 mg every 12 hours. However, he started suffering from odynophagia, asthenia, and adynamic, so he went to a private physician who diagnosed pharyngotonsillitis and indicated unspecified antibiotic treatment. Five days later, he began to have non-quantified fever, dry cough in accesses, and medium effort dyspnea, so he went to the emergency room for evaluation.

The patient was assessed at the respiratory triage of our special hospital unit. It was noticed that his oxygen saturation was at 45%. Accordingly, he was given supplemental oxygen, which increased his oxygen saturation to 55%. Due to his critical condition, he was immediately admitted to the shock service as a priority type I. Due to respiratory failure, airway management was started, performing sedation and neuromuscular blockade with midazolam and rocuronium; then an orotracheal tube number 7.5 was placed, and introduced 21 cm from the dental arch. At the same time, it was decided to place a central venous catheter with right subclavian approach, without complications.

Invasive mechanical ventilation was started with the following parameters: total volume 30 ml, inspired oxygen fraction (FiO_2) 100%, respiratory rate (RR) 24 rpm, positive end-expiratory pressure (PEEP) 14, inspiration:expiration ratio (I:E) 1:1.5, peak pressure (Ppeak) 36 cmH_2O, while maintaining a oxygen saturation ($SatO_2$) > 90%. Management with norepinephrine at 0.7 μg/kg/min and dobutamine at 2.5 μg/kg was also started. In addition, ceftriaxone, oseltamivir, and clarithromycin were administered. The following diagnoses were integrated: acute respiratory failure type 1, mixed acid-base imbalance (acute respiratory acidosis, lactic acidosis), probable atypical pneumonia versus severe ARDS (SARS-CoV-2, CURB 65 two points, PSI PORT 127 points, SMART-COP eight points), hydro-electrolyte imbalance (mild hypocalcemia) without electrocardiographic repercussions.

A polymerase chain reaction (PCR) swab was performed for SARS-CoV-2, which was reported positive on 27-04-20. The patient was presented to the intensive care unit on 28-04-20 as a confirmed case of severe acute respiratory failure syndrome (ARDS) and advanced airway management.

He was extubated on 28-05-20, after which the patient presented an 80% right pleural effusion, so he was referred to the general surgery service on 05-06-20 for placement of an endo pleural probe *(Figure 1)*.

However, the patient evolved in a torpid manner without achieving pulmonary re-expansion, so he was scheduled for right posterolateral thoracotomy that was performed on 10-06-20, observing necrotizing pneumonia, bronchopleural fistula of the right middle lobe, and pachypleuritis as transoperative findings *(Figure 2)*. Approximately 200 cm^3 of cloudy material was drained; cultures were sent to the lab, and an endo pleural tube and two Jackson-Pratt drains, one anterior and the other posterior, were left as drains *(Figure 3)*.

The patient progressed towards improvement. it was decided to remove the endo pleural tube on 15-06-20; he was extubated on 18-06-20, and the anterior Jackson Pratt drain was removed on 19-06-20. He was discharged home on 06-23-20 to continue with outpatient follow-up.

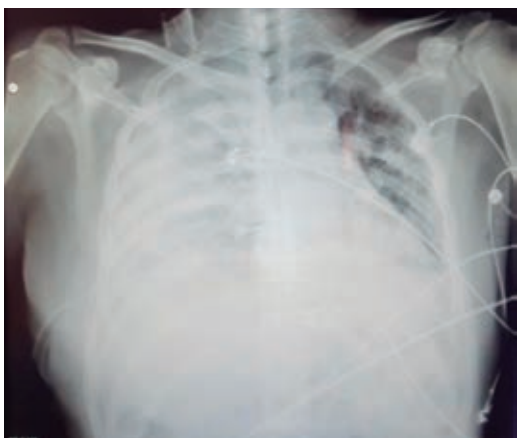

Figure 1: *Chest X-ray after endo pleural probe placement showing a right pleural effusion of approximately 80%.*

Subsequently, a pathology report was collected, which mentioned acute fibrinopurulent and chronic organized pachypleuritis with few foreign body type giant cells. These are histological findings compatible with ulcerated bronchopleural fistula with granulation tissue, chronic inflammation with few foreign body type giant cells, recent and old hemorrhage, with irregular scar-like fibrosis in the adjacent lung parenchyma.

DISCUSSION

Following the acquisition of SARS-CoV-2 infection, multiple associated complications can occur. Age is the leading risk factor for progression to acute respiratory distress syndrome (ARDS).[2-5] Comorbidities, high fever (≥ 39 ºC), smoking history, and certain laboratory features also predict progression and death from COVID-19. The need for mechanical ventilation in critically ill patients ranges from 30 to 100%.[3,5-8] However, lung compliance is high compared to other etiologies of ARDS. The incidence of barotrauma in those requiring mechanical ventilation has been reported in up to 25% of patients despite low tidal volumes and peak pressures.[9] On the other hand, pleural effusions are considered unusual.[10]

There needs to be more data describing the pulmonary pathology of COVID-19 pneumonia in critically ill patients. Most autopsy reports describe mononuclear inflammation,[11,12]

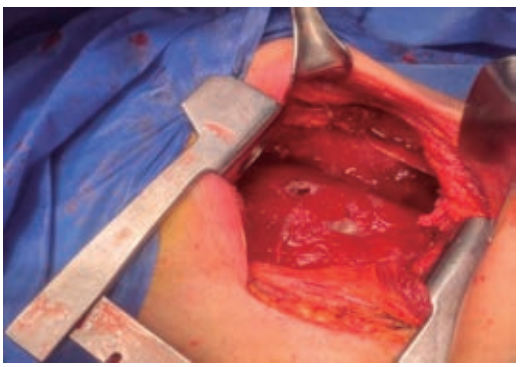

Figure 2: *A bronchopleural fistula in the middle lobe of the right lung measuring approximately 1 × 1.5 cm during posterolateral thoracotomy.*

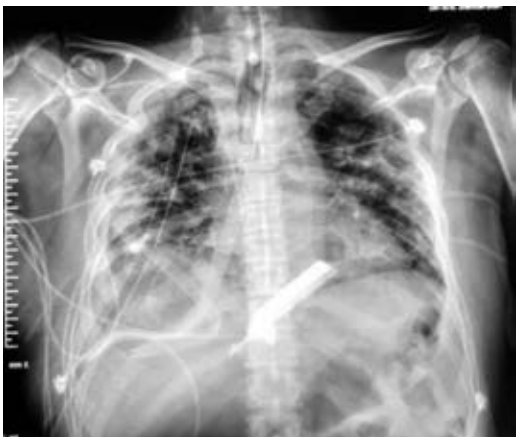

Figure 3: *Chest X-ray taken 24 hours after the procedure, showing adequate lung expansion, presence of two Jackson Pratt drains (anterior and posterior), and an endo pleural tube.*

hyaline membrane changes, and micro vessel thrombosis suggestive of early ARDS (i.e., exudative, and proliferative phases of diffuse alveolar damage [DAD]).[12-14] Other findings include bacterial pneumonia (isolated or superimposed on DAD) and viral pneumonitis.[13,14] Less common findings include acute fibrinous organizing pneumonia (late stages),[15] amyloid deposition, and rarely, alveolar hemorrhage and vasculitis.[14]

In this patient, there were related histopathologic findings, chronic inflammation with rare foreign body-like giant cells, and findings described as "less common" or atypical, including pachypleuritis, and ulcerated bronchopleural fistula, hemorrhage, and fibrosis of adjacent lung parenchyma. The percentage of patients with long-term sequelae is currently unknown; however, a retrospective study of 110 patients with COVID-19 reported persistent pulmonary function abnormalities at discharge in patients with mild and severe pneumonia.[15]

CONCLUSION

This case leads us to consider the significant variability of possible complications secondary to this new virus and the need for further research. Likewise, this case exemplifies the need to consider surgical treatment for

those critical patients with a compromised pulmonary distensibility who do not respond to conventional medical treatment. However, patients should be carefully selected not to cause further aggravation and to offer resolutive treatment to candidates for it.

ACKNOWLEDGMENTS

We thank the healthcare workers (medical staff, nurses, orderlies, housekeeping, and administrative staff) for their unstinting efforts to fight the pandemic and care for our sick.

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Ethical considerations and responsibility: the authors declare that they followed the protocols of the workplace when publishing data of patients, safeguarding their right to privacy through the confidentiality of their data.
Financing: the authors declare that no financing was received for the report's writing.
Disclosure: the authors declare no conflict of interest.

Correspondence:
Érika Barlandas-Quintana
E-mail: barlandas@gmail.com

CLINICAL CASE
doi: 10.35366/109718

Cirujano General
April-June 2022
Vol. 44, no. 2 / p. 87-92

Emphysematous gastritis secondary to gastric mucormycosis in a patient with COVID-19. A case report

Gastritis enfisematosa secundaria a mucormicosis gástrica en paciente con COVID-19. Reporte de caso

Martín Islas-Torres,[*] Ana Laura Castillo-Luna,[‡] José Juan Rodríguez-Moreno,[‡] Valeria Priscilla Rendón-Muñoz,[‡] José Gerardo Zamora-Inzuna,[‡] Albert Antonio Ibarra-Trejo[‡]

Keywords: gastrointestinal mucormycosis, emphysematous gastritis, vertical gastrectomy, COVID, case report.

Palabras clave: *mucormicosis gastrointestinal, gastritis enfisematosa, gastrectomía vertical, COVID, reporte de caso.*

[*] Attending Physician, Department of General Surgery.
[‡] Resident Physician, Department of General Surgery.

Hospital de Especialidades, Centro Médico Nacional de Occidente, Instituto Mexicano del Seguro Social.

Received: 07/27/2021
Accepted: 12/23/2022

ABSTRACT

Introduction: mucormycosis is an invasive fungal infection with high mortality. It is seen mainly in immunocompromised patients. It is characterized by necrotizing vasculitis and the presence of branching right-angled hyphae. The gastrointestinal form is among the rarest; symptoms are nonspecific, and only 25% are diagnosed *antemortem*. Treatment consists of antifungal therapy and urgent surgical debridement. **Case report:** a 69-year-old male patient with a history of type 2 diabetes mellitus presents with dyspnea; the rapid antigen test for SARS-CoV-2 was positive. During his stay, he presented sepsis, gastrointestinal bleeding, and pneumoperitoneum on abdominal computed axial tomography (CT) scan. He underwent exploratory laparotomy, and necrosis of the greater curvature of the stomach was found, and vertical gastrectomy was performed. The histopathological report reveals pan mural necrosis associated with arterial thrombosis secondary to *Mucor sp.*, liposomal amphotericin B was started; however, the patient developed nosocomial urinary and pulmonary infections and died 29 days after admission. **Conclusion:** mucormycosis is an emerging fungal infection that requires high suspicion for its diagnosis. Antifungals and urgent surgical debridement by the general surgeon represent an essential pillar in treating this entity.

RESUMEN

Introducción: *la mucormicosis es una infección micótica invasiva con alta mortalidad, ocurre principalmente en pacientes inmunocomprometidos. Se caracteriza por vasculitis necrosante y la presencia de hifas ramificadas en ángulo recto. La forma gastrointestinal es una de las más raras, los síntomas son inespecíficos y sólo 25% se diagnostica antemortem. El tratamiento consiste en antifúngico y desbridamiento quirúrgico urgente.* ***Caso clínico:*** *paciente masculino de 69 años con antecedente de diabetes mellitus tipo 2 acude con disnea, la prueba rápida de antígeno para SARS-CoV-2 resulta positiva. Durante su estancia presenta sepsis, sangrado gastrointestinal y neumoperitoneo en tomografía axial computarizada (TAC) abdominal. Se somete a laparotomía exploradora, en la que se encuentra necrosis de la curvatura mayor del estómago, se le realiza gastrectomía vertical. El reporte histopatológico revela necrosis panmural asociada a trombosis arterial secundaria a* Mucor sp., *se inicia anfotericina B liposomal; sin embargo, el paciente desarrolla infecciones nosocomiales urinaria, pulmonar y fallece a los 29 días de su ingreso.* ***Conclusión:*** *la mucormicosis es una infección micótica emergente que requiere una alta sospecha para su diagnóstico. Los antifúngicos y el desbridamiento quirúrgico urgente por parte del cirujano general representan un pilar esencial en el tratamiento de esta entidad.*

How to cite: Islas-Torres M, Castillo-Luna AL, Rodríguez-Moreno JJ, Rendón-Muñoz VP, Zamora-Inzuna JG, Ibarra-Trejo AA. Emphysematous gastritis secondary to gastric mucormycosis in a patient with COVID-19. A case report. Cir Gen. 2022; 44 (2): 87-92. https://dx.doi.org/10.35366/109718

INTRODUCTION

Mucormycosis is an emerging infection caused by fungi belonging to the order Mucorales;[1] it occurs mainly in patients with immunosuppression such as hematologic malignancy, hematopoietic stem cell or solid organ transplantation, and diabetes mellitus;[2] In this context, patients with COVID-19 may have an altered cell-mediated immune response and increased susceptibility to fungal co-infections.[3] The disease is characterized by extensive necrotizing vasculitis, resulting in thrombosis and subsequent tissue infarction. Primary gastrointestinal disease is the rarest form, with the stomach being the most common site of infection. It can be acquired by ingesting contaminated food but can also be associated with contaminated healthcare-associated devices. Diagnosis may be suspected by endoscopic findings showing a fungal mass or necrotic lesions overlying an ulcerated area; radiological findings are nonspecific.[3] Early diagnosis helps to accelerate antifungal therapy and improve survival; liposomal amphotericin B is the treatment of choice. Surgical debridement or complete resection of the affected organ is of utmost importance to eliminate necrosis and improve penetration of antifungal agents into the target site; these patients are at high risk of perforation and bleeding, which requires extensive surgical resection.[4] We reviewed the literature in databases such as *Wiley and PubMed* using the keywords *mucormycosis, COVID, and infection*. We found very few case reports of fungal disease associated with COVID-19 and no reports when searching for the association between the words *gastric, mucormycosis, and COVID*.

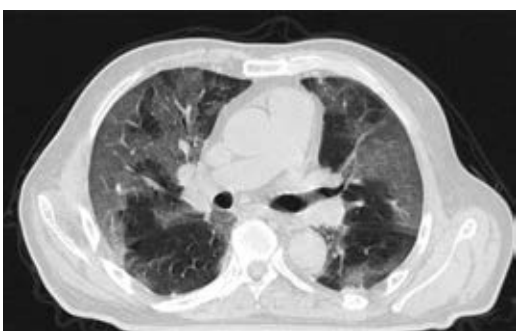

Figure 1: *A chest axial computed tomography (CT) scan of the chest showing radiological data suggestive of SARS-CoV-2 infection.*

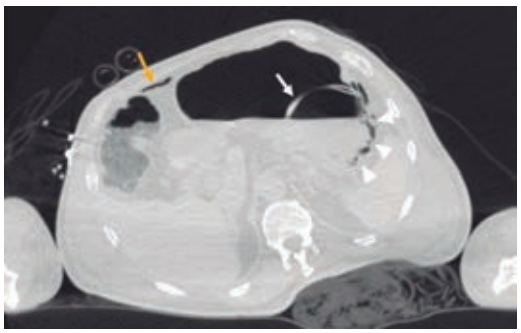

Figure 2: *Simple abdominopelvic axial computed tomography scan showing gastric pneumatosis and pneumoperitoneum.*

CLINICAL CASE

A 69-year-old male patient with a history of type 2 diabetes mellitus was treated with metformin and chlorpropamide. He also had hypertension treated with losartan and amlodipine. He presented to the emergency department with headache, chest pain, cough, myalgias, arthralgias, and dyspnea of one-week evolution. On admission, his heart rate was 114 bpm, respiratory rate 24 rpm, oxygen saturation 80% without supplemental oxygen support; his glucose was 587 mg/dl, white blood cells 25,690 cells/mm^3, neutrophils 85%, lymphocytes 1.2%, hemoglobin 16.3 g/dl, procalcitonin 3.78 ng/dl, arterial blood gas with a pH of 7.41, pCO_2 21 mmHg, PO_2 35 mmHg, HCO_3 13 mmol/l, oxygen saturation 62%. A rapid antigen test for SARS-CoV-2 was performed, and the result was positive; a CT scan of the chest showed radiological data suggestive of SARS-CoV-2 infection *(Figure 1)*, and management with supplemental oxygen, insulin infusion pump, carbapenem antibiotics, antihypertensives, dexamethasone, and antithrombotic prophylaxis with enoxaparin was started. 48 hours after admission, the patient continued with septic shock, so vasopressor amines were started; an orotracheal intubation was performed and support with

invasive mechanical ventilation was initiated. A nasogastric tube (NGT) was placed. After 24 hours of its placement, he presented hematemesis accompanied by abdominal distension, leukocytosis of 40,000 cells/mm^3. A simple abdominopelvic CAT scan was performed, which revealed gastric pneumatosis and pneumoperitoneum *(Figure 2)*. An urgent exploratory laparotomy was performed finding necrosis of the greater curvature of the stomach. A vertical gastrectomy was performed with GIA stapler with purple cartridges of 45 and 60 mm *(Figure 3)*; the stapling line was reinforced with continuous a Prolene suture 00; a feeding jejunostomy tube was placed at 60 cm from the Treitz angle and Saratoga drains were left in place. Twenty-four hours later, the patient had hemodynamic stability, vasopressor amines were suspended, and enteral nutrition with an elemental diet was started. On the fifth postoperative day, the patient showed adequate tolerance to the enteral diet through jejunostomy. The methylene blue test was performed through the nasogastric tube showing no evidence of leaks; the white blood cell count decreased to 24,000 cells/mm^3. Histopathology results were obtained revealing pan mural necrosis associated with arterial thrombosis secondary to microorganisms compatible with *Mucor sp.;* therefore, management with amphotericin B was initiated *(Figures 4 and 5)*. During follow-up he showed good evolution; an abdominopelvic CT scan with contrast by the nasogastric tube was performed, which ruled out leaks and intra-abdominal collections and corroborated the integrity of the gastric wall; drains were removed, and the patient was discharged from general surgery on day 9 post-surgery, continuing to be managed by the internal medicine service. During the following days of hospitalization, the patient presented cardiovascular and pulmonary deterioration; a urine culture was taken, which showed growing of *Candida tropicalis*, and a CAT scan of the skull, thorax, and abdominopelvic with oral contrast was performed *(Figures 6 and 7)*, The patient continued without evidence of abdominal leaks, rhino-cerebral and pulmonary mucormycosis were ruled out. A probable superimposed bacterial pneumonia and interstitial pneumopathy secondary to COVID-19 were reported. He showed persistent clinical deterioration and refractory shock and 29 days after admission.

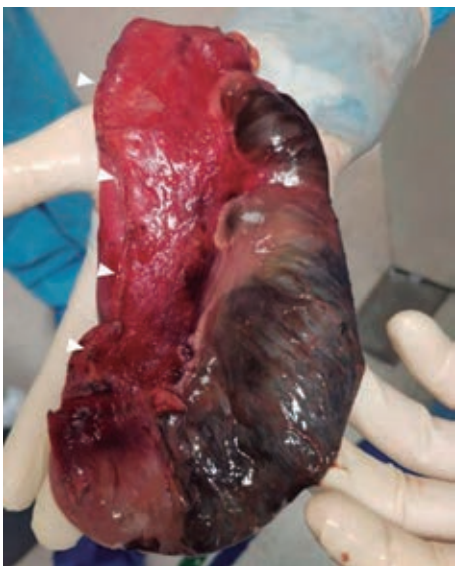

Figure 3: *Product of a vertical gastrectomy due to necrosis of the gastric greater curvature.*

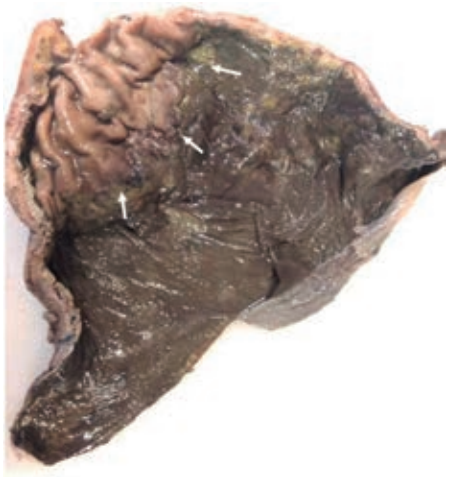

Figure 4: *Pathological specimen with pan mural necrosis associated with arterial thrombosis secondary to microorganisms compatible with* Mucor sp.

DISCUSSION

Mucormycosis is an invasive fungal infection with high mortality; its incidence has

increased in recent years due to the increase in the population at risk of infection and the improvement in diagnostic tools. It was first described at the pulmonary level in 1876 by Furbringer.[5] It occurs mainly in the context of an immunocompromised patient.[1] Mortality varies from 32 to 70%; the localized infection is associated with better survival;[6] It is usually diagnosed *antemortem* in only 25% of cases. In their review of 31 cases, Dioverti et al. report a predominance of male sex (61%) and a mean age of 47 years; 52% of the cases occurred in patients with a solid organ transplant, and 35% were patients undergoing chemotherapy for hematologic malignancy. All the patients had at least one comorbidity, with neutropenia being the most common finding.[2] It has been described in patients with severe malnutrition and the use of corticosteroids, but also in patients without any predisposing factor.[7] In our case, the patient had multiple risk factors already described such as decompensated type 2 diabetes mellitus, acute coronavirus infection, and steroid therapy initiated to manage COVID-19. The only factor that improved the prognosis was the focal infection in the stomach. The disease is characterized by extensive necrotizing vasculitis with arterial thrombosis and tissue infarction[1] and a pathognomonic presence of non-septate right-angled branching hyphae in tissues.[8] The genus Rhizopus is the most frequently isolated.[9] Other known risk factors include hyperglycemia and acidosis, as they cause leukocyte dysfunction of neutrophils and macrophages, affecting their chemotaxis.[10] Six different clinical syndromes can occur, with rhino-orbit-cerebral and pulmonary infection being the most frequent and gastrointestinal infection the least common. Gastrointestinal infection is acquired through ingestion of contaminated food, or in the case of healthcare, it is associated with contaminated devices.[1] The stomach is the most common site of involvement, followed by the colon, small intestine, and esophagus.[11] Most symptoms are nonspecific, which delays diagnosis and increases mortality.[2] Clinical presentation may be with abdominal pain (68%), gastrointestinal bleeding (48%), fever (19%), or defecation changes (10%).[2]

On admission, the patient did not report any abdominal symptoms, so gastrointestinal involvement was not suspected. Subsequently, intravenous sedation and orotracheal intubation were performed, making it impossible for the patient to manifest gastric involvement. The only sign the patient presented was hematemesis 24 hours after the nasogastric tube placement, which ruled out the possibility of healthcare-associated mucormycosis since 24 hours are not enough to achieve the introduction, inoculation, and growing of the fungus. This led to the hypothesis that the patient acquired the fungal infection in his community. The diagnosis may be suspected by endoscopic findings, such as a fungal mass or necrotic lesions covering

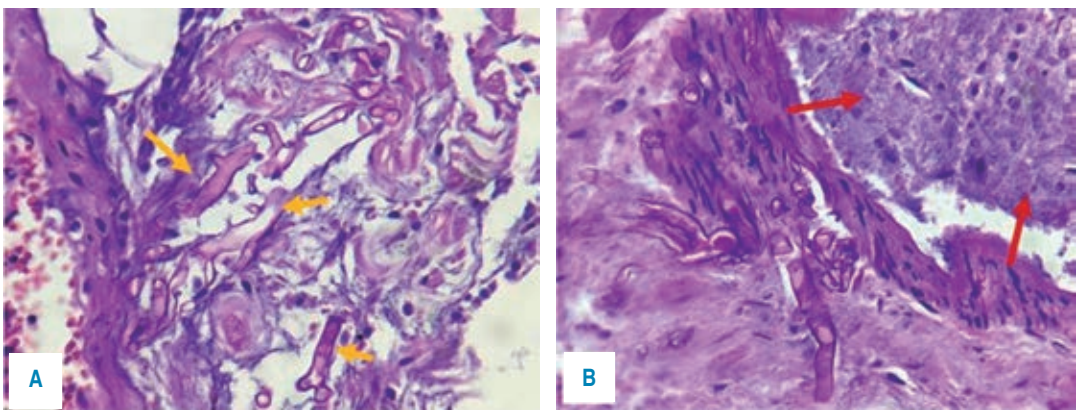

Figure 5: *H-E staining showing pan mural necrosis associated with arterial thrombosis secondary to microorganisms compatible with* Mucor sp.

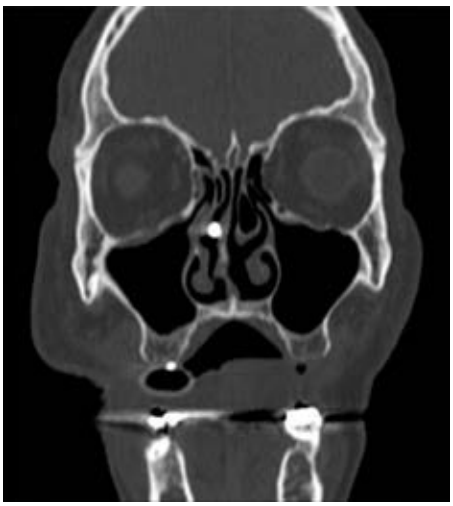

Figure 6: *Cranial computed tomography scan ruling out rhino-cerebral mucormycosis.*

ulcerated areas that may perforate tissues and cause peritonitis.[2] Often, the study protocol is initiated in the presence of an intra-abdominal abscess; the diagnosis can be made by biopsy of the suspicious area during surgery or endoscopy.[7] Few samples are usually sent for culture,[2] and those sent are positive in only 30%, in addition, specialized culture media such as potato dextrose agar are required for fungal growing.[12] Confirmatory molecular tests that may detect surface antigens are also required, but they are not yet available.[2] CT findings may include focal or diffuse thickening of the gastric wall, pneumatosis with decreased wall enhancement on contrast administration (emphysematous gastritis) secondary to ischemia and necrosis, adjacent collections, wall necrosis with focal disruption or perforation, and rarely, pneumoperitoneum.[13] When hematemesis occurs, the next diagnostic step should be an upper endoscopy. However, the abdominal distention presented by the patient suggested a high probability of perforation of the hollow viscera, so initially, a CT scan of the abdomen was performed. A surgical emergency was considered when reporting gastric pneumatosis and pneumoperitoneum, ruling out an endoscopy. Exploratory laparotomy allows direct exploration of the stomach and partial resection of the stomach as a diagnostic and therapeutic measure. Treatment consists of antifungal and urgent surgical debridement since necrotic tissue's presence will affect the antifungal's penetration to the tissues; additionally, aggressive medical support for comorbidities should be provided.[14] Intravenous liposomal amphotericin B is the treatment of choice. It is more effective than conventional amphotericin B.[15] Delaying the initiation of amphotericin for more than six days, doubles mortality.[4] Although most of literature favors aggressive surgical treatment, we opted for a more conservative treatment by preserving a portion of the stomach and performing only a vertical gastrectomy since macroscopic inspection showed a clear demarcation between necrotic and viable tissues. We consider that the surgical treatment we performed was correct since, in the post-surgical follow-up, the patient had a good evolution; the methylene-blue test and the two abdominal CT scans with contrast in the stomach through the nasogastric tube allowed us to verify the integrity of the suture line from the first post-surgical day until the day of his death. The sample sent to pathology allowed to make the diagnosis and thus initiate targeted therapy with amphotericin B, and since no fungal infection by *Mucor sp*. at any other organic level was found, the cause of death was attributed to nosocomial infectious complications at the pulmonary and urinary level.

CONCLUSION

Mucormycosis is a life-threatening fungal infection. Therefore, the diagnosis requires

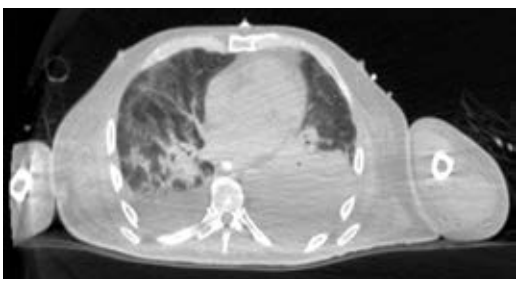

Figure 7: *Chest computed tomography scan in pulmonary phase showing probable superimposed bacterial pneumonia and interstitial pneumopathy secondary to COVID-19.*

a high index of suspicion, especially in cases with intestinal involvement, and should be suspected in all patients with risk factors and imaging studies suggestive of unexplained gastrointestinal ischemia and necrosis. The general surgeon's role in the success of the treatment consists of urgent surgical debridement, who, based on the transoperative surgical findings, should opt for aggressive management or preserve the integrity and functionality of the affected organ as far as possible.

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Declaration of ethical conflicts: the authors have no ethical conflicts to declare.
Funding and disclosure: the authors have no conflicts of interest or funding conflicts to declare.

Correspondence:
José Juan Rodríguez-Moreno
E-mail: rodriguez_medicina@hotmail.com

CLINICAL CASE
doi: 10.35366/109719

Cirujano General
April-June 2022
Vol. 44, no. 2 / p. 93-96

Mesenteric ischemia as a presentation of primary antiphospholipid syndrome in a male patient

Isquemia mesentérica como presentación de síndrome antifosfolípido primario en paciente masculino

Erick Zúñiga-Garza,* Francisco Franco-López‡

Keywords:
mesenteric ischemia, antiphospholipid antibody syndrome, acute abdomen.

Palabras clave:
isquemia mesentérica, síndrome de anticuerpos antifosfolípidos, abdomen agudo.

ABSTRACT

Antiphospholipid syndrome is an autoimmune disease characterized by the thromboembolic formation and maternal morbidity, associated with a persistent increase in antiphospholipid antibody titers; involvement in male patients is rare, and onset with gastrointestinal manifestations is uncommon; for this reason, we present the clinical case of a 19-year-old male patient with no relevant history, who started with antiphospholipid syndrome through mesenteric ischemia.

RESUMEN

El síndrome antifosfolípido es una enfermedad autoinmune caracterizada por formación tromboembólica y/o morbilidad materna, asociada a incremento persistente en los títulos de anticuerpos antifosfolípidos, la afectación en pacientes masculinos es rara, y el inicio con manifestaciones gastrointestinales es algo poco común, por dicha razón presentamos el caso clínico de paciente masculino de 19 años sin antecedentes de importancia, quien inicia con un síndrome antifosfolípido a través de una isquemia mesentérica.

INTRODUCTION

Antiphospholipid syndrome (APS) is a multisystem disease characterized by thrombus formation and emboli associated with maternal morbidity and persistent increase in antiphospholipid antibody titers;[1] 1% of cases present as catastrophic APS, in which multiple vascular occlusive events occur affecting perfusion of various organs.[2] A small percentage of patients present with gastrointestinal involvement.[3]

CASE PRESENTATION

A 19-year-old male with no previous history came to the emergency department for two weeks of intermittent abdominal pain located in the epigastrium with irradiation towards the mesogastrium, described as oppressive, only referred to a change in bowel habits, previously treated with proton pump inhibitor and butylhyoscine/lysine as analgesic without improvement. He went twice to the emergency department with the same symptoms; on each visit he was treated conservatively. On his fourth visit, he mentioned that pain had increased and was accompanied by vomiting. Physical examination revealed dehydrated mucous membranes and generalized pallor, tachycardia (100 bpm), and abdomen with pain on palpation in the epigastrium, with evidence of peritoneal irritation. H was admitted for further diagnostic protocol with the following paraclinical tests:

Urea 40.3 mg/dl, creatinine 1.99 mg/dl, sodium 139 mEq/l, potassium 3.6 mEq/l, chlorine 104 mEq/l, white blood cells 15,070

* Resident of general surgery.
‡ Attending physician, General Surgery Service. Centennial Hospital Miguel Hidalgo.

Received: 03/30/2021
Accepted: 12/23/2022

How to cite: Zúñiga-Garza E, Franco-López F. Mesenteric ischemia as a presentation of primary antiphospholipid syndrome in a male patient. Cir Gen. 2022; 44 (2): 93-96. https://dx.doi.org/10.35366/109719

10^3/l, neutrophils 65%, hemoglobin 15.7 g/dl, hematocrit 47%, platelets 370,000 10^3/l, prothrombin time 15.1 seconds, thromboplastin time 25.2 seconds, INR 1.2.

The abdominal ultrasound showed free fluid in subhepatic, peri splenic, and para vesical recesses *(Figure 1)*.

Due to these findings, an exploratory laparotomy was performed where abundant inflammatory response fluid (900 ml), small bowel loops (120 cm) with thrombosis of mesenteric vessels were seen. An ischemic segment resection with Brooke ileostomy at 150 cm from the angle of Treitz and Hartmann closure of the distal stump *(Figure 2)* were performed. It was decided to place a Bogota bag for a *second look* laparotomy.

Subsequently, he was sent to the general surgery floor, where he presented an adequate stoma and Bogota pouch expense. An evaluation by the internal medicine, hematology, and rheumatology services was requested, who suggested taking anticardiolipin antibodies, anti-Beta-2-glycoprotein antibodies, antinuclear antibodies, and homocysteine levels. Treatment was started with enoxaparin, then with acenocoumarin.

After 48 hours he was reoperated (second look). The Bogota bag ass withdrawn. A few thrombosed vessels on the non-ischemic omentum were found. The small bowel loops were dilated, edematous, and without peristalsis. The mesentery was thickened, cramped, with multiple swollen lymph nodes. The distal segment (stoma) had no signs of ischemia. The terminal ileum stump showed no leak *(Figure 3)*. The abdominal wall was closed with a continuous suture with polydioxanone 1 in two stages, and Blanco Benavides stitches were used with braided polyester 5.

Subsequently, he continued his evolution on the general surgery floor, where the suggested laboratory results were obtained (Ac anticardiolipin IgG 59, IgM 50.2), and a diagnosis of primary antiphospholipid syndrome was made. The patient was discharged with a functional ileostomy. Follow-up by general outpatient surgery and rheumatology continued, and intestinal reconnection was performed electively at six months *(Figure 4)* without complications.

DISCUSSION

Between 1983 and 1986, a clinical syndrome of thrombosis associated with antibodies against phospholipids was identified. Initially it was named anticardiolipin syndrome and currently is known as antiphospholipid syndrome.[3] It is a multisystem disease characterized by thromboembolic formation and maternal morbidity, associated with a persistent increase in antiphospholipid antibody titers.[1] In 2019, an incidence of antiphospholipid syndrome of two cases/100,000 population was reported, with a prevalence of 50 cases/100,000 population;[4] however, no data were found in the Mexican literature search.

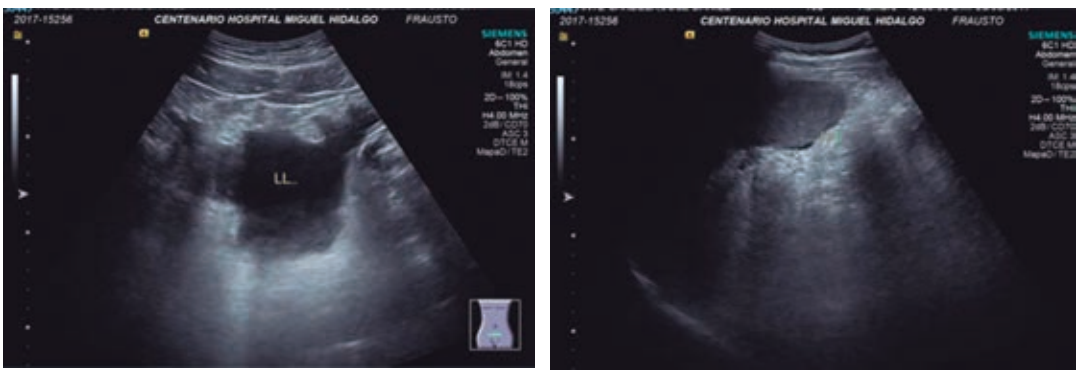

Figure 1: *Ultrasound scan showing free fluid.*

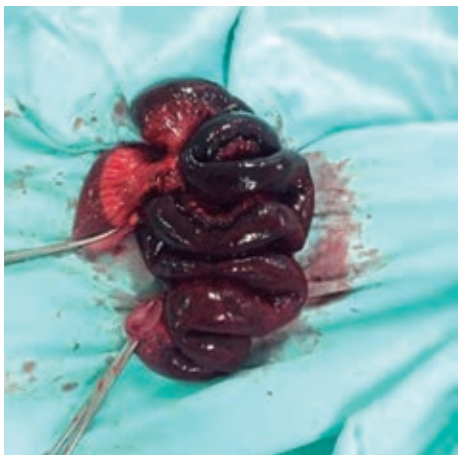

Figure 2: *The macroscopic specimen of the surgical event shows a necrotic small bowel. Histopathology reported a small bowel segment of 112 cm, with transverse dimensions of 3 × 3 cm. Microscopically there were recent multifocal arterial thrombosis and extensive pan mural ischemic necrosis.*

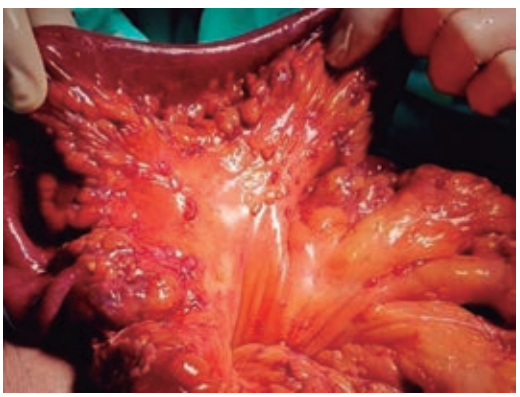

Figure 3: *Surgery (second look) showing few thrombosed vessels in the omentum, without omentum ischemia, and edematous small bowel loops dilated.*

The diagnosis of APS is made through clinical and laboratory criteria (Sapporo criteria).[5] They were updated in 2006;[6] the clinical criteria include an episode of vascular thrombosis or morbidity during pregnancy. In contrast, laboratory criteria include elevated lupus anticoagulant titers, anticardiolipin antibodies, and anti-β_2 glycoprotein antibodies.[6]

Intestinal involvement by APS is rare and is usually associated with a poor outcome.[7] Intestinal involvement may manifest as mesenteric ischemia, characterized by hypoxia of the bowel due to a sharp decrease in blood perfusion caused by embolism or thrombosis.[8]

The clinical presentation is nonspecific. Symptoms and other findings that may suggest mesenteric thrombosis are abdominal pain, diarrhea, vomiting, blood in stool, hyperlactatemia, leukocytosis, and metabolic acidosis.[8]

There is no standardized treatment for catastrophic APS, but anticoagulation is the mainstay therapy, and in some cases, surgery is necessary.[7,9] The prognosis is generally poor due to low clinical suspicion.[1,6-9]

CONCLUSION

The onset of an antiphospholipid syndrome as mesenteric ischemia is an extremely rare pathology. In a previously healthy patient, this disease should always be suspected in the range of differential diagnoses. Early recognition and multidisciplinary treatment can change the outcome of these patients.

Figure 4: *Postoperative status of the patient, showing a healed wound.*

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Ethical considerations and responsibility: the authors declare that they followed the protocols of their work center on the publication of patient data, safeguarding their right to privacy through the confidentiality of their data.

Funding: no financial support was received for this work.

Disclosure: the authors declare no conflict of interest in carrying out the work.

Correspondence:
Erick Zúñiga-Garza
E-mail: erickzunigagarza5@gmail.com

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