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Vesicular dyskinesia remains an unsolved medical issue. A review of the literature

La discinesia vesicular continúa siendo una incógnita a resolver en problemas médicos, revisión de la literatura

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Palabras clave:
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ABSTRACT

Introduction: The purpose of this report is to describe that gallbladder dyskinesia represents the most diffuse gastrointestinal motility disorder of unknown origin. The diagnosis of gallbladder dyskinesia remains a challenge, especially for those physicians who are not well related or familiar with this entity of epigastric or right upper quadrant pain, without any organic alteration that may explain the patient's symptoms. Gallbladder dyskinesia should be suspected in patients with biliary pain, in whom liver enzymes, pancreatic enzymes, hepatobiliary abdominal ultrasound and upper gastrointestinal endoscopy are normal, without alterations that may explain the clinical picture. **Material and methods:** Of 132 patients operated on for cholecystectomy, only 60 patients with gallbladder dyskinesia were registered in this study, of which only 55 patients underwent nuclear medicine imaging for hepatobiliary scintigraphy with Boyden test. All were symptomatic, and their discomfort was not mitigated by antacids, prokinetics, or proton pump inhibitors. **Results:** Of the 60 patients who underwent cholecystectomy by laparoscopic surgery, all have reported good to excellent functional and symptomatic results, with minimal morbidity, no deaths, and no recurrence of symptomatology at six months of follow-up. **Conclusions:** Since the first description by Krukenberg in 1903, the approach to gallbladder dyskinesia has not gained worldwide popularity among surgeons. We believe that when there is a comprehensive clinical history, a complete physical examination showing no visceromegaly and in the absence of gallstones or other structural pathology on abdominal ultrasound, gallbladder dyskinesia should be considered until proven otherwise.

RESUMEN

Introducción: *El propósito de este informe es describir que la discinesia de la vesícula biliar representa el trastorno más difuso de la motilidad gastrointestinal sin conocer el origen, el diagnóstico de la discinesia vesicular continúa siendo un desafío, especialmente para los médicos que no están bien relacionados o familiarizados con esta entidad de dolor en epigastrio o cuadrante superior derecho, sin ninguna alteración orgánica que pueda explicar los síntomas del paciente. La discinesia de la vesícula biliar debe sospecharse en pacientes con dolor biliar, en quienes las enzimas hepáticas, pancreáticas, la ecografía abdominal hepatobiliar y la endoscopia digestiva alta son normales, sin alteraciones que expliquen el cuadro clínico.* ***Material y métodos:*** *De 132 pacientes operados de colecistectomía, sólo 60 pacientes con discinesia vesicular se registraron en este estudio, de los cuales, sólo 55 pacientes se sometieron a medicina nuclear para gammagrafía hepatobiliar con prueba de Boyden. Todos estaban sintomáticos, y no se mitigaban sus molestias con antiácidos, procinéticos o inhibidores de la bomba de protones.* ***Resultados:*** *De los 60 pacientes que se sometieron a colecistectomía por cirugía laparoscópica, todos ellos han reportado de buenos a excelentes resultados tanto funcionales como sintomáticos, con una morbilidad mínima, sin muertes y sin recurrencia de la sintomatología a seis meses de seguimiento.* ***Conclusiones:*** *Desde la primera descripción por Krukenberg en 1903, el enfoque de la discinesia vesicular no ha ganado popularidad mundial entre los cirujanos. Creemos que cuando hay una historia clínica precisa, un examen físico completo que concluya que no hay visceromegalias y en ausencia de cálculos biliares u otra patología estructural en el ultrasonido abdominal, se debe considerar una discinesia vesicular hasta que no se demuestre lo contrario.*

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HISTORICAL BACKGROUND

After a systematic review of PubMed/Medline and Scopus databases from 1980 to 2016 to identify relevant literature, a search was done using terms related to the same disease such as: gallbladder dyskinesia, biliary dyskinesia, functional gallbladder, biliary spasm or acalculous cholecystitis and HIDA or scintigraphy (cholecystography), ejection fraction and cholecystectomy.[1,2]

In this search, 29 studies were retrieved that included 2,891 patients contrasting cholecystectomy with medical treatment, where it was observed that patients with a normal gallbladder ejection fraction (GEF) or above 35% did not benefit from cholecystectomy; while those with low GEF < 35% were more likely (relative risk [RR] = 2.37) to have symptomatic improvement after surgery.

These authors concluded that it is possible that a low GEF may provide some guidance to identify individuals with gallbladder dyskinesia who may benefit from cholecystectomy. However, available data were inconsistent and are based on low-quality studies that are often subject to patient bias and the impact of confounding factors. For these reasons, these authors speculated that the role of scintigraphy and cholecystectomy in the definition and treatment of this disorder remains unclear pending a definitive study.[1-5]

Krukenberg, in 1903, was the first physician to mention the term gallbladder dyskinesia. Later, pathologists Aschoff and Bacmieister described it in 1909 as a gallbladder stasis occurring without inflammation or biliary lithiasis.[1-5]

The recognition of biliary pain in the absence of gallstones was first described in the 1920s. In 1924, Blalock[6] described 139 patients with acalculous cholecystitis out of 735 patients with lithiasis biliary disease. As early as 1926, Whipple,[7] in agreement with Blalock's article, questioned to perform a cholecystectomy without having a definite pathology to justify it. In that same year, according to the surgical criteria for cholecystectomy, Whipple recommended leaving *in situ* a normal appearing acalculous gallbladder, based in what he observed in the 47 patients undergoing cholecystectomy without evidence of gallstones, of which 76.6% were asymptomatic at follow-up compared to almost 90% of the cases with gallbladder stones.[6] Cholecystography was first described in 1924 by Graham and Cole,[8] who used tetra bromo-phenolphthalein, a substance excreted in the biliary tree to allow radiological imaging of the gallbladder and biliary tree. In the following decades, several patients underwent cholecystectomy in the absence of gallstones. Similarly, in 1956, other physicians such as Gleen and Mannix[9] studied the results in patients who underwent cholecystectomy for gallbladders without stones and showing no inflammation, reporting that only 65% of patients had an improvement in their symptoms, 11% reported some improvement, while 25% reported no improvement at all.

With the increasing use of cholecystokinin during cholecystography, in 1975 Freeman et al[10] and others reported on the use of cholecystokinin injection to identify patients with acalculous gallbladder disease and who may benefit from cholecystectomy. In Freeman's study of 22 patients with decreased ejection fraction or reproduction of symptoms with cholecystokinin injection, 95% reported relief or improvement of symptoms after cholecystectomy.

Over the decades since the Freeman's study,[10] the diagnosis has improved, and subsequent studies have found similarities with high success rates compared to those of the early 20th century literature.

PATHOGENESIS

The exact pathogenesis of gallbladder dysfunction is unknown, but it is presumed that the pain associated with gallbladder dysfunction could be related to a very complex functional signaling that causes functional obstruction of bile flow from the gallbladder due perhaps to non-occlusive narrowing of the cystic duct. Another hypothesis is an abnormality found in the smooth muscle layer of the gallbladder causing

impaired gallbladder emptying, which was proposed by Merg,[11,12] who showed a higher incidence of chronic cholecystitis in patients with gallbladder dysfunction compared to normal subjects. Gallbladder dysfunction has been associated with altered motility in other gastrointestinal organs, for example, impaired gallbladder emptying has been observed more frequently in adults suffering from slow transit constipation,[13] or diarrhea in intestinal hypersensitivity, gastroparesis,[13] and achalasia.[13] This extends the reason if functional motility disorders are often concomitant in separate areas of the gastrointestinal tract, such as gastroesophageal reflux (hiatal hernia), gastroparesis (ulcerative peptic disease), constipation (irritable bowel syndrome), diarrhea (gastroenteritis), postprandial abdominal distention (irritable bowel syndrome) or pancreatic or hepatic insufficiency.

Cholecystokinin (CCK) is a neurotransmitter peptide that was originally discovered in the intestine (Ivy and Oldberg in 1928) but is widely distributed in the central nervous system (Van der Haegen, 1975).[14-16]

Cholecystokinin (CCK) is a neurotransmitter peptide that is secreted mainly in two forms, CCK33 and CCK8. They are condensed in the I cells of the duodenal mucosa and jejunum. They are also synthesized in the central nervous system, mainly in the forms of CCK8 and CCK4.

The two receptors that mediate the effects of CCK are CCKA and CCKB.

The CCKA receptor is found mainly in the gastrointestinal tract, while the CCKB receptor is found mainly in the brain.[11,14-16]

CCK exerts multiple gastrointestinal effects and is released during meals, causing delayed gastric emptying, gallbladder contraction, regulation of intestinal motility, and secretion of pancreatic enzymes in communication with the brain through the vagus nerve, while in the brain it causes an anorexigenic effect, postprandial somnolence and is speculated to have a role in emotions.[11,13-16]

Gallbladder function involves very complex signaling cycles. One hypothesis regarding the cause of pain in gallbladder dyskinesia is the increased pressure of the gallbladder which, by not contracting properly, accumulates bile in the gallbladder.

In 1997 it was demonstrated the formation of crystals in the bile[17] forming gallstones in patients undergoing cholecystectomy due to gallbladder dyskinesia; therefore, inflammation of the gallbladder wall has been proposed as a cause of pain, since even 94% of patients undergoing surgery show chronic and acute changes of cholecystitis in the histological study.[17-20]

Another hypothesis proposed for gallbladder dyskinesia is the existence of generalized hypersensitivity in the neural pathways connecting the brain and thalamus with the intestine.

Visceral hypersensitivity has been demonstrated in patients with other functional biliary disorders such as the Oddi sphincter dysfunction.[20]

It has also been shown that patients with irritable bowel syndrome show altered gallbladder contraction in response to cholecystokinin. Therefore, gallbladder dyskinesia may be the result of defects in cholecystokinin nerve signaling causing changes in bile composition and chronic cholecystitis.[16,17,20]

Gallbladder dyskinesia represents the most diffuse gastrointestinal motility disorder.

Esophageal gastric reflux disease and colonic inactivity affect many patients with gallbladder dyskinesia and are therefore commonly confused and diagnosed as irritable bowel syndrome, suggesting a relationship between the two disorders.[19,21] Cholecystokinin receptors are expressed throughout the gastrointestinal tract and, among other things, affect colonic motility and sensory function.

Based on these properties, cholecystokinin antagonists have been developed to treat functional disorders such as the irritable bowel syndrome.[17,19,21,22]

There are multiple active forms of cholecystokinin, and it is recognized as the most widely distributed neuropeptide in the brain with high concentrations of it and its receptors in the cerebral cortex, olfactory bulb, hypothalamus, amygdalae, hippocampus,

striatum, periaqueductal gray matter, and spinal cord.

This neuroanatomical distribution has generated speculation about its role in anxiety disorders, leading to multiple studies that have used cholecystokinin antagonists for these psychiatry conditions.[15,16]

Gallbladder dysmotility is believed to play a central role in the pathogenesis.

Gallbladder dysmotility may be the result of an initial metabolic disorder (i.e., bile supersaturated with cholesterol), which increases biliary viscosity, or a primary motility disorder in the absence, at least initially, of any abnormality in bile composition.[17,21] Functional gallbladder disorder has been associated with abnormal gastric emptying and abnormal colonic transit, suggesting a possible generalized gastrointestinal motility disorder.[11,12,17,19,21]

DIAGNOSIS

In 1923 Westphal described gallbladder dyskinesia as a "dysfunction of the autonomic nervous system of the gallbladder".[1,3] Gallbladder dyskinesia has been postulated for several decades as an entity that constitutes a motility disorder of the gallbladder, which manifests clinically with biliary pain in the right upper quadrant or epigastrium that in many patients may radiate to the right infra-scapular region. The pain is often associated with diaphoresis, nausea, and vomiting, may also be accompanied by abdominal distention, constipation, or diarrheal evacuations and/or gastroesophageal reflux.[21,23]

The pain stabilizes in less than an hour, ranging from moderate to excruciating intensity and once stabilized, the pain usually lasts at least 30 minutes and then slowly subsides over several hours.[21,23,24]

In a large percentage of patients whose abdominal ultrasound is negative for cholelithiasis, a number of erroneous diagnoses such as irritable bowel, peptic acid disease, hepatic or pancreatic pathology are wrongly made, resulting in the request of inappropriate tests, unnecessary costs and inadequate prescriptions. This results in higher costs of care and what is more serious, the persistence of symptoms that do not improve with the intake of antacids, proton pump inhibitors or prokinetics, or the reappearance of these symptoms after discontinuation of the drugs.[21,23,24]

The prevalence of gallbladder dyskinesia is estimated at 8% in men and 22% in women.[21,25] Definitely, the diagnosis of gallbladder dyskinesia is a challenge, particularly for physicians who are not well related or familiar with this entity characterized by pain in the right upper quadrant, accompanied by diaphoresis, nausea, and gastro-biliary or alimentary vomiting and practically always with a hepatobiliary abdominal ultrasound without evidence of lithiasis, sludge or inflammation of the gallbladder, normal liver function tests, pancreatic and a normal esophageal and gastroduodenal endoscopy.[21,23,24]

The Rome IV criteria include[26] "low gallbladder ejection fraction" following dietary stimulation as a "supportive" criterion for making this diagnosis.[5,14,27,28]

Gallbladder dyskinesia constitutes approximately 80% of patients with "unspecified gallbladder disease" in the United States, where hospital admissions for this disease have tripled in recent years, with a 700% increase in the pediatric population, constituting 5 to 20% of cholecystectomies in adult patients and 10 to 50% in the pediatric population in that country.[21,25]

The Rome III criteria for the diagnosis of functional gallbladder disorders defined it as biliary-type pain with normal liver and pancreatic enzymes, along with exclusion by abdominal US of other structural diseases, including gallstones and normal upper endoscopy.

The Rome III criteria define vesicular dyskinesia as the existence of various symptoms with variations during periods such as:

1. An episode of pain lasting at least 30 minutes.
2. Recurrent symptoms at different intervals (not daily).
3. Gradually increasing pain.

4. Moderate to severe pain that sometimes disrupts daily activities and may require evaluation in the emergency department.
5. The pain is not relieved by bowel movements.
6. The pain does not decrease with changes in position.
7. Pain is not relieved using antacids, proton pump inhibitors or prokinetics.
8. Exclusion of other structural diseases that may explain the symptoms.

In the Rome IV version of the criteria for the diagnosis of functional disorders, "low ejection fraction" of the gallbladder has been included as a supportive criterion for diagnosis of gallbladder dyskinesia.[5,14,28]

The Rome IV criteria for functional gallbladder disorder require[26] biliary pain, which is defined as pain in the epigastrium and/or right upper quadrant that meets all the following criteria:

1. Progressive pain to a constant level and lasts at least 30 minutes.
2. Pain occurring at different intervals (usually not daily).
3. Pain severe enough to disrupt daily activities or to go to the emergency department (< 20%) with bowel movements or relieved by postural change or acid suppression.

Criteria that support but are not required for biliary pain include pain associated with nausea and vomiting, radiation of pain in the right infra-scapular region, and pain that awakens the patient. Absence of gallstones, inflammation, or other structural pathology.

Criteria supporting functional gallbladder disorder, but not mandatory, include a low gallbladder ejection fraction (GEF) on hepatobiliary scintigraphy less than 35% performed in the nuclear medicine department that depends on an intravenous infusion of CCK, or an oral fatty meal and a time of 5, 30 or 40 minutes. In our hospital we use oral stimulation with 40 minutes as total post stimulation time to read the ejection fraction of the gallbladder.

A cut-off of 35% was recommended to define the lower normal limit of FSG[5,14,28] to diagnose gallbladder dyskinesia and predict a good response to cholecystectomy.[5,14,28]

Clinical presentation

Postprandial pain or abdominal distention accompanied by nausea related to intolerance to fatty foods, which is not relieved by antacids, proton pump inhibitors or prokinetics.

Following symptoms occurring one or more times in a 12-month period are nausea 52%, vomiting 43%, abdominal distention 21%, early satiety 21%, constipation 21%, and diarrhea 13%, with no evidence of pathology or structural abnormality to explain the origin of the symptoms.[21,23,24,29]

The first entity one should think of is a patient with epigastric pain or in the right upper quadrant of the abdomen, abdominal distention and gastro-biliary vomiting 15-20 minutes after food intake, particularly when associated with normal liver and pancreatic function tests (aminotransferases, gamma-glutamyl transpeptidase, alkaline phosphatase, total and conjugated bilirubin, amylase, lipase, hematic cytology, and normal erythrocyte sedimentation rate [ESR] and C-reactive protein [CRP] values) with normal esophago-gastroduodenal endoscopy and with an abdominal USG without evidence of gallbladder lithiasis or inflammation, should be considered a gallbladder dyskinesia until proven otherwise. The protocol to be followed to diagnose gallbladder dyskinesia includes performing a study of the gallbladder in the nuclear medicine department *(Figure 1)* with HIDA, also known as hepatobiliary scintigraphy cholecystography with 99_m of technetium (Tc) labeled with hepatic iminodiacetic acid (HIDA), administered as an intravenous bolus of the radio-labeled marker, which is absorbed by the liver and concentrated in the gallbladder as bile. The gallbladder is usually seen by minute 20 and after 60 minutes the patient is given an injection of a drug called cholecystokinin (CCK) or is allowed to eat a fatty meal to be performed at that time, since both CCK administered IV and oral fatty meal (Boyden's test) are signals for the gallbladder to contract, and if the gallbladder ejection fraction

(GEF) is less than 35% after 40 minutes of measurement, the test is considered universally positive for gallbladder dyskinesia (Krishnamurthy described it in 1981).[5,14,28]

The utility of nuclear medicine examination of gallbladder dyskinesia with HIDA focuses on its ability to indirectly assess gallbladder contractility in response to stimulation (nowadays a fatty food intake), where the results are expressed in terms of the percentage of radioactive tracer voided in the gallbladder ejection fraction (GEF) *(Figures 2 and 3)*. Dating back to 1970s,[28] 17 earlier studies using oral cholecystography and stimulation with IV CCK reported that a group of patients with biliary pain and no evidence of gallstones had a poorly contractile gallbladder. After 40 years cholecystography techniques in nuclear medicine *(Figure 1)* have improved considerably, and cholecystectomy has been used for quite some time to treat patients with low GEF less than 35%, obtaining excellent results despite the somewhat vague nature of this ill-defined disorder and the limitations of the proposed diagnostic tests. However, the incidence of cholecystectomy as a treatment

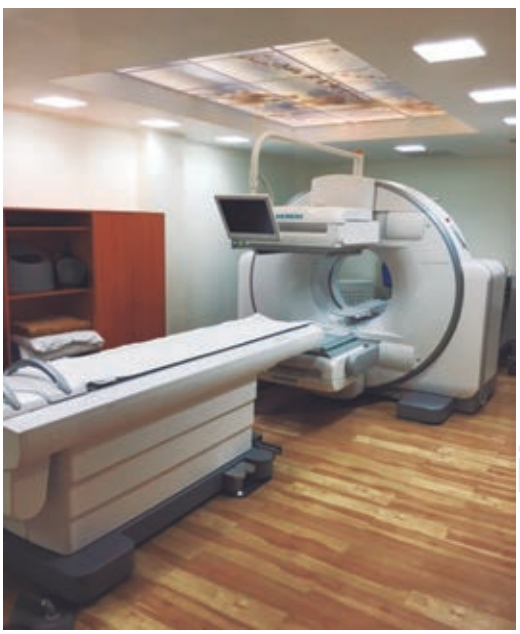

Figure 1: *SIEMENS SYMBIA INTEVO nuclear medicine equipment plus CT scan.*

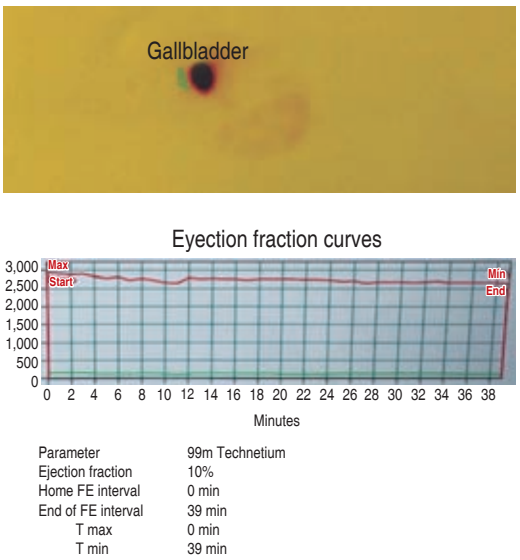

Figure 2: *Hepatobiliary iminodiacetic acid (HIDA) showing gallbladder EF (ejection fraction) 10% positive for gallbladder dyskinesia.*

for gallbladder dyskinesia (i.e., individuals without gallstones or other organic gallbladder pathology) has increased rapidly in the United States compared with other parts of the world, particularly since the advent of the laparoscopic approach.[5,14,19,20,28]

Therefore, despite years of research and debate, controversies still exist regarding the diagnosis and prognosis of gallbladder dyskinesia as well as the usefulness of cholecystectomy for gallbladder dyskinesia. Our objective, therefore, was to analyze through a systematic review the performance of gallbladder ejection fraction less than 35% in predicting response to cholecystectomy in patients with gallbladder dyskinesia.

It should be considered and recorded in the clinical history that several medical conditions can decrease the GEF such as diabetes mellitus, pregnancy, celiac disease, and irritable bowel syndrome as well as the administration of drugs such as anticholinergics, opioids, NSAIDs, calcium channel blockers, oral contraceptives, histamine receptor antagonists[2] and benzodiazepines, which should be discontinued at least 24 hours before the study.[5,14,18,28]

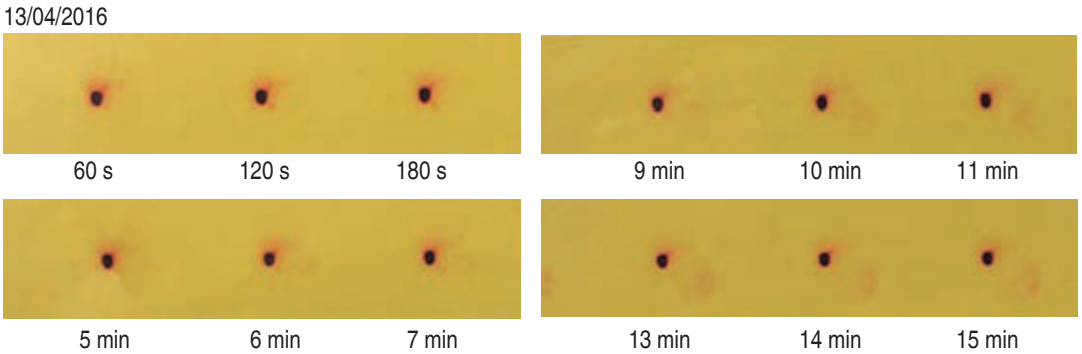

Figure 3:

Hepatobiliary iminodiacetic acid (HIDA) showing an EF of 10% of gallbladder compatible with gallbladder dyskinesia.

MATERIAL AND METHODS

From June 2015 to June 2019, we operated by laparoscopic surgery on 132 patients. They were 38 males (28.7%) and 94 females (71.2%), with a mean age of 28 years (range, 14-58 years). Only patients with gallbladder dyskinesia were included in this study. Patients with gallbladder stones were excluded from the study.

All patients underwent cholecystectomy by laparoscopic surgery.

Of 60 patients diagnosed with vesicular dyskinesia, 42 were female (70%) and 18 were male (30%) *(Table 1)*.

All patients underwent abdominal ultrasonography, where no stones, sludge, or gallbladder inflammation were observed, followed by 55 patients with hepatobiliary scintigraphy with hepatic iminodiacetic acid (HIDA) labeled with technetium (Tc) 99_{mm} and stimulated by a standard fatty oral feeding, showing a gallbladder ejection fraction (GEF) less than 35%. There were five patients who did not received HIDA, due to previous gallbladder abnormalities seen in an abdominal ultrasonography (US) scan, such as cap or phrygian cap. or due to the presence of a septum in neck or gallbladder fundus without lithiasis, sludge or inflammation.

The histopathological diagnoses of the 60 operated patients were compatible with acute chronic cholecystitis and coincided with adhesions of the greater omentum, duodenum, transverse colon to Hartman's pouch, or body, or gallbladder fundus or that the greater omentum completely covered the entire gallbladder as shown in *Figures 4 to 6*.

On histopathology review, 10 had sludge and cholesterolosis of the biliary mucosa *(Figures 7 and 8)*, two had polyps and three had gallbladder segmentation, one in the fundus and two in Hartmann's pouch that were not detected by abdominal US.

Of the total patients in this study, 100% presented with symptoms of upper abdominal pain, accompanied by diaphoresis, nausea, vomiting, abdominal distention, constipation, and postprandial gastroesophageal reflux in the absence of other attributable causes and associated with low gallbladder ejection fractions (GEF) less than 35%, with no evidence of hiatal hernia by endoscopy in patients who reported gastroesophageal reflux.

Abdominal pain, nausea, vomiting, constipation, and abdominal distention were the most common symptoms, and diaphoresis when the pain was so severe that they had to go to the emergency room, or the pain subsided within 30 minutes.

The average GEF was 26.0%.

All patients were followed for six months. There were no losses. All the questions asked were regarding their main symptoms before surgery. They were all retrieved taken from the clinical record and the satisfaction level after surgery. All patients revealed that their symptoms were completely relieved, including gastroesophageal reflux symptoms after cholecystectomy.

RESULTS

Of the 60 patients who underwent cholecystectomy by laparoscopic surgery

Table 1: Cases of vesicular dyskinesia, pathology and accompanying symptoms.

Sex	Age	Pathology	FE vesicular (%)	Associated symptoms
1. F	32	Mud	20	Postprandial epigastric pain, nausea, vomiting, abdominal distention, constipation
2. F	32	Mud	35	Postprandial epigastric pain, nausea, vomiting, abdominal distention
3. M	39	Mud	28	Postprandial epigastric pain, nausea, vomiting, abdominal distention, constipation
4. F	34	Cholecystitis	2	Postprandial epigastric pain, nausea, vomiting, abdominal distention, constipation
5. F	52	Cholecystitis	15	Postprandial epigastric pain, nausea, vomiting, abdominal distention
6. F	57	Cholecystitis	20	Postprandial epigastric pain, nausea, vomiting, abdominal distention
7. F	27	Cholecystitis	10	Epigastric pain, nausea, vomiting, abdominal distention, constipation, gastroesophageal reflux
8. M	32	Cholecystitis	3	Postprandial epigastric pain, nausea, vomiting, abdominal distention
9. F	20	Cholecystitis	7	Epigastric pain, nausea, vomiting, abdominal distention, constipation, gastroesophageal reflux
10. F	25	Cholecystitis	35	Postprandial epigastric pain, nausea, vomiting, abdominal distention
11. F	19	Cholecystitis	8	Epigastric pain, nausea, vomiting, abdominal distention, constipation, gastroesophageal reflux
12. F	28	Cholecystitis	35	Postprandial epigastric pain, nausea, vomiting, abdominal distention
13. F	53	Cholecystitis	35	Postprandial epigastric pain, nausea, vomiting, abdominal distention
14. F	22	Cholecystitis	20	Postprandial epigastric pain, nausea, vomiting, abdominal distension, gastroesophageal reflux
15. F	18	Cholecystitis	1.8	Postprandial epigastric pain, nausea, vomiting, abdominal distension, gastroesophageal reflux
16. F	57	Mud and polyp	30	Postprandial RUQ pain, nausea, vomiting, abdominal distention, constipation
17. F	26	Cholecystitis	16	Postprandial epigastric pain, nausea, vomiting, abdominal distension, gastroesophageal reflux
18. F	40	Cholecystitis	24	Postprandial epigastric pain, nausea, vomiting, abdominal distention
19. F	19	Cholecystitis	15	Epigastric pain, nausea, vomiting, abdominal distention, gastroesophageal reflux, constipation
20. F	25	Cholecystitis	24	Postprandial epigastric pain, nausea, vomiting, abdominal distention
21. M	17	Cholecystitis	35	Postprandial epigastric pain, nausea, vomiting, abdominal distention
22. M	42	Cholecystitis	35	Postprandial epigastric pain, nausea, vomiting, abdominal distention
23. F	54	Phrygian cap	0	Postprandial epigastric pain, nausea, vomiting, abdominal distention, constipation
24. F	30	Cholecystitis	24	Postprandial epigastric pain, nausea, vomiting, abdominal distention
25. M	49	Mud	2	Postprandial epigastric pain, nausea, vomiting, abdominal distention
26. F	18	Phrygian cap	3	Postprandial epigastric pain, nausea, vomiting, abdominal distension, gastroesophageal reflux
27. F	36	Cholecystitis	25	Postprandial epigastric pain, nausea, vomiting, abdominal distension, gastroesophageal reflux
28. M	37	Cholecystitis	28	Postprandial epigastric pain, nausea, vomiting, abdominal distention
29. F	55	Phrygian cap	3	Postprandial epigastric pain, nausea, vomiting, abdominal distension, gastroesophageal reflux
30. F	51	Mud	0	Continuous pain in epigastrium and RUQ, right subscapular irradiation, constipation.
31. M	25	Cholecystitis	13	Postprandial epigastric pain, nausea, vomiting, abdominal distension, gastroesophageal reflux

Continue Table 1: Cases of vesicular dyskinesia, pathology and accompanying symptoms.

Sex	Age	Pathology	FE vesicular (%)	Associated symptoms
32. F	32	Phrygian cap	3	Epigastric pain, nausea, vomiting, abdominal distention, constipation, gastroesophageal reflux
33. F	37	Phrygian cap	3	Postprandial epigastric pain, nausea, vomiting, abdominal distention
34. F	24	Phrygian cap	3	Postprandial epigastric pain, nausea, vomiting, abdominal distention
35. F	27	Phrygian cap	3	Postprandial epigastric pain, nausea, vomiting, abdominal distension, gastroesophageal reflux
36. M	23	Phrygian cap	3	Epigastric pain, nausea, vomiting, abdominal distention, constipation, gastroesophageal reflux
37. M	29	Cholecystitis	24	Postprandial epigastric pain, nausea, vomiting, abdominal distention
38. F	58	Mud	0	Postprandial epigastric pain, nausea, vomiting, abdominal distention, constipation
39. M	33	Cholecystitis	32	Postprandial epigastric pain, nausea, vomiting, abdominal distention
40. F	24	Cholecystitis	35	Postprandial epigastric pain, nausea, vomiting, abdominal distention, constipation
41. M	23			Phrygial cap postprandial epigastric pain, nausea, vomiting, abdominal distention, gastroesophageal reflux
42. F	25			Phrygial cap, postprandial epigastric pain, nausea, vomiting, abdominal distention, constipation
43. M	40	Cholecystitis	32	Postprandial epigastric pain, nausea, vomiting, abdominal distention, constipation
44. F	34	Mud	1	Epigastric pain, nausea, vomiting, abdominal distention, gastroesophageal reflux, constipation
45. M	57	Mud	0	Postprandial epigastric pain, nausea, vomiting, abdominal distention, constipation
46. M	29	Polyp	25	Postprandial RUQ pain, nausea, abdominal distention, gastroesophageal reflux, constipation
47. F	20	Mud	28	Postprandial RUQ pain, nausea, vomiting, abdominal distention, constipation
48. M	47	Cholecystitis	32	Postprandial epigastric pain, nausea, vomiting, abdominal distention, constipation
49. F	20			Phrygial cap, postprandial epigastric pain, nausea, vomiting, abdominal distention, constipation
50. F	20	Cholecystitis	10	Postprandial epigastric pain, nausea, vomiting, abdominal distention, constipation
51. F	40	Cholecystitis	1	Postprandial epigastric pain, nausea, vomiting, abdominal distention, constipation
52. F	26	Cholecystitis	0	Postprandial epigastric pain, nausea, vomiting, abdominal distension, gastroesophageal reflux
53. F	23	Cholecystitis	35	Postprandial epigastric pain, nausea, vomiting, abdominal distention, constipation
54. F	32	Cholecystitis	27	Postprandial epigastric pain, nausea, diarrhea, abdominal distention, constipation
55. F	36	Cholecystitis	24	Postprandial epigastric pain, nausea, vomiting, abdominal distention, constipation
56. M	26	Cholecystitis	21	Postprandial epigastric pain, nausea, vomiting, abdominal distention, constipation
57. M	29			Phrygial cap, postprandial epigastric pain, nausea, vomiting, abdominal distention, gastroesophageal reflux
58. F	20			Phrygial cap, postprandial epigastric pain, nausea, vomiting, abdominal distention, gastroesophageal reflux
59. F	14	Mud	9	Postprandial epigastric pain, nausea, vomiting, abdominal distension, gastroesophageal reflux
60. F	35	Cholecystitis	34	Postprandial epigastric pain, nausea, vomiting, abdominal distention, constipation

F = female; M = male; EF = ejection fraction; RUQ = right upper quadrant.

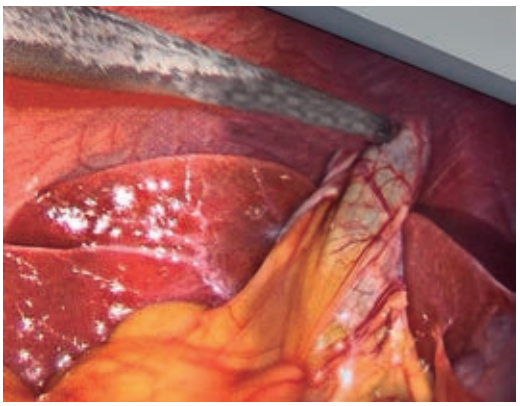

Figure 4: *Note the adhesions of the chronic process. Multiple adhesions, practically no structure of the gallbladder is identified and in most of these cases of severe inflammation the duodenum is attached to the gallbladder.*

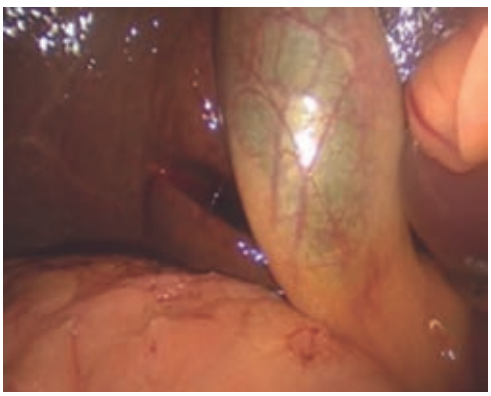

Figure 5: *Note the inflammation in Hartman's pouch of a chronic process.*

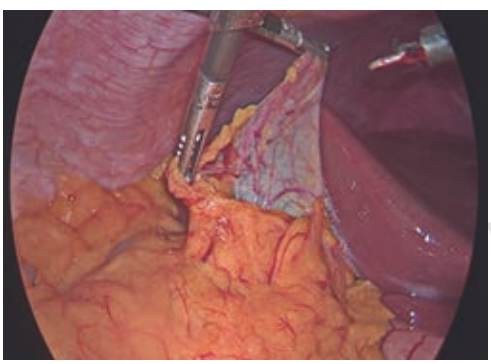

Figure 6: *Note the adhesion of the greater omentum to the entire gallbladder due to a chronic inflammatory process that was not seen on abdominal US scan.*

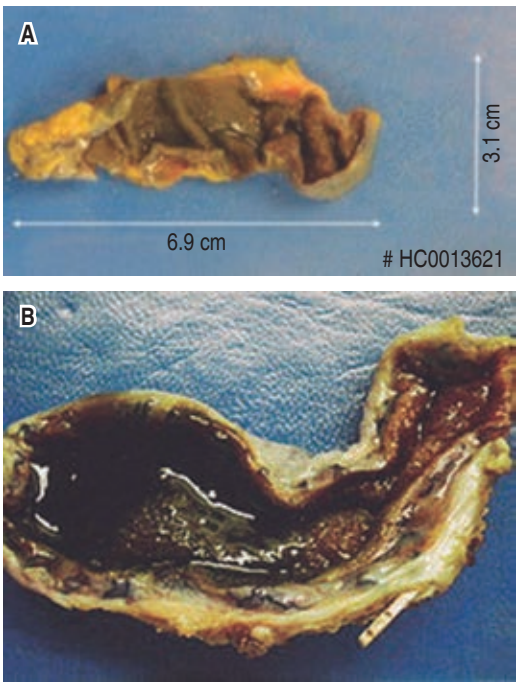

Figure 7: *Note the gallbladder inflammation, congestive mucosa and edematous mucosal cholesterolosis (**A**) without lithiasis and sludge (**B**).*

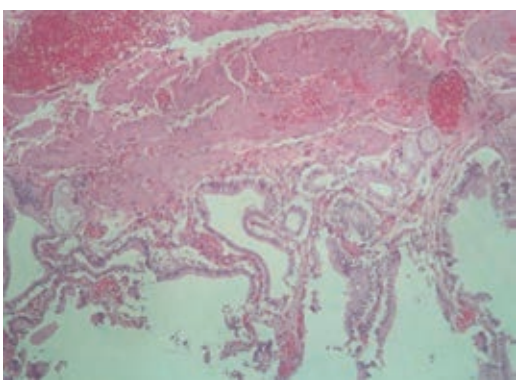

Figure 8: *Edematous and congestive vesicular wall, mixed type inflammatory infiltrate with polymorphonuclear predominance involving up to the muscularis.*

for gallbladder dyskinesia, all reported good to excellent functional and symptomatic results with minimal morbidity, no deaths, no complications, no infections, and no recurrence of symptomatology.

CONCLUSION

Gallbladder dyskinesia continues to be more common in women than in men, in younger people than in older adults, and is characterized clinically by symptoms of biliary-type pain in the absence of gallstones or other structural pathology.

Gallbladder dyskinesia remains as a challenge and a clinical mystery for gastroenterological surgeons.

Cholecystectomy has shown efficacy in curing symptoms in more than 90% of patients.[4]

To avoid late diagnosis, HIDA with feeding stimulation should be used as early as possible in the evaluation of a patient with biliary colic pain and a negative ultrasonography scan.

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