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MIRIZZI SYNDROME, DESCRIPTION, ETIOLOGY, EPIDEMIOLOGY, PATHOPHYSIOLOGY, CLASSIFICATION, HISTOPATHOLOGY, PRESENTATION, DIAGNOSIS, TREATMENT, PROGNOSIS AND COMPLICATIONS

Bryam Esteban Coello García [1], Cynthia Carolina Cañar Santos [2], Carolina Elizabeth Mosquera Rojas [3], Tatiana Carolina Espinoza Coyago [4], Christian Paul Flores Tapia [5], Paola Fernanda Murillo Pineda [6], David Alejandro Guacho Escudero [7], Gabriela Patricia Murillo Araujo [8]

[1]Postgraduate Doctor in Orthopedics and Traumatology at Faculdade de Ciências Médicas Minas Gerais. Belo Horizonte - Brasil. ORCID https://orcid.org/0000-0003-2497-0274
[2]General Practitioner at "Hospital básico de Paute" Azuay-Ecuador. ORCID https://orcid.org/0009-0000-5025-5376
[3]General Practitioner in Independent Practice, Faculty of Medical Sciences, Universidad de Cuenca. ORCID https://orcid.org/0009-0001-4952-9082
[4]General Practitioner in Independent Practice, Faculty of Medical Sciences, Universidad de Cuenca. ORCID https://orcid.org/0000-0001-9725-5760
[5]General Practitioner in Independent Practice, Faculty of Medical Sciences, Universidad Católica de Cuenca. Azuay- Ecuador ORCID https://orcid.org/0000-0002-7580-2919
[6]General Practitioner at "Centro de Salud Yunganza-Ministerio de Salud Pública" Limón-Ecuador. ORCID https://orcid.org/0009-0006-7735-4132
[7]General Practitioner in Independent Practice, Faculty of Medical Sciences, Universidad Central del Ecuador. Quito- Ecuador ORCID https://orcid.org/0009-0006-6669-2646
[8]General Practitioner at "Centro de Salud San Salvador-Ministerio de Salud Pública". Faculty of Medical Sciences, Universidad Central del Ecuador. ORCID https://orcid.org/0009-0004-6973-1589
Corresponding Author : *Bryam Esteban Coello García* ***Address:*** *Rua Tiradentes 266.Campo Belo. Minas Gerais. Brasil* ***Postal Code:*** *37270-000*

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ABSTRACT

Introduction: *Mirizzi's syndrome, named after the Argentine surgeon Pablo Luis Mirizzi who first reported the syndrome in 1948, when he showed a history of a patient who presented a calculus of considerable size impacted in the infundibulum of the gallbladder, which generated jaundice due to extrinsic compression of the common bile duct. We speak of Mirizzi syndrome when there is an impaction of a calculus in the infundibulum of the gallbladder or the cystic duct that crushes the common hepatic duct, weakening it and forming a cholecystocholedochal fistula.*
Objective: *to detail the current information related to Mirizzi syndrome, description, etiology, epidemiology, pathophysiology, classification, histopathology, presentation, diagnosis, treatment, prognosis and complications.*
Methodology: *a total of 45 articles were analyzed in this review, including review and original articles, as well as clinical cases, of which 33 bibliographies were used because the other articles were not relevant to this study. The sources of information were PubMed, Google Scholar and Cochrane; the terms used to search for information in Spanish, Portuguese and English were: Mirizzi Syndrome, obstruction, common bile duct, gallstone, hepatic duct.*
Results: *Mirizzi syndrome is relatively infrequent, occurring in only 0.1% of patients with gallstones with findings in 0.7% to 25% of patients who have undergone cholecystectomy. Between 5% to 28% of individuals with Mirizzi syndrome had gallbladder cancer following cholecystectomy. The modification of the anatomy and the cholecystocoledocian fistula increase the risk of damage to the biliary tract in the surgical act of cholecystectomy. Regarding classification type I was present in 40% of cases, type II, type III and type IV in 20% each.*
Conclusions: *Mirizzi syndrome, also called extrinsic biliary compression syndrome, is an infrequent complication of chronic cholecystitis and cholelithiasis secondary to obliteration of the cystic duct or gallbladder infundibulum (Hartmann's pouch) generated due to the impact of one or more calculi. The impacted stone plus the inflammatory response produces the obstruction of the external biliary tract, with which the mucosa will erode and form a cholecystohepatic or cholecystocoledocian fistula. The risk of presenting gallbladder cancer increases with Mirizzi syndrome. The most*

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common presentation is obstructive jaundice and right upper quadrant pain accompanied by epigastric pain, dark urine, nausea, tachycardia, vomiting, anorexia, fever and chills. The beginning of the diagnosis should be made with the usual tests for cholecystitis.Right upper quadrant abdominal ultrasound is currently the first-line study for the diagnosis of gallstones and acute cholecystitis. If a gallstone in the common bile duct is suspected by ultrasound, magnetic resonance cholangiopancreatography should be performed. This is followed by endoscopic retrograde cholangiopancreatography (ERCP) performed by a trained gastroenterologist. Conventionally the treatment of Mirizzi syndrome is surgical. Cholecystectomy is the first-line treatment; in case of fistula, open cholecystectomy with bilioenteric anastomosis, possibly with a Roux-en-Y is effective. In individuals without fistula, the prognosis is usually positive. In elderly patients with multiple comorbidities and high risk of surgical complications, non-surgical methods should be considered to minimize the morbidity associated with surgery.

KEY WORDS: *Mirizzi, syndrome, obstruction, duct, calculus, common bile duct, hepatic.*

INTRODUCTION

Mirizzi's syndrome, named after the Argentine surgeon Pablo Luis Mirizzi, graduated from the Faculty of Medical Sciences of the National University of Cordoba in 1915, who is recognized for performing the first intraoperative cholangiography in 1931, generating a strong impact in the field of biliary surgery in the twentieth century. Pablo Luis Mirizzi reported the syndrome for the first time in 1948, showing a history of a patient who presented a calculus of considerable size impacted in the infundibulum of the gallbladder, which generated jaundice due to extrinsic compression of the common bile duct (CBD) accompanied by inflammation directed from the gallbladder to the CBD. This syndrome occurs in about 1% of individuals with cholelithiasis and is an infrequent complication. We speak of Mirizzi syndrome when there is an impaction of a calculus in the gallbladder infundibulum or the cystic duct that crushes the common hepatic duct, weakening it and forming a cholecystocholedochal fistula. This disease is closely related to gallbladder cancer, in addition to presenting obstructive jaundice. The preoperative diagnosis of this syndrome is complex and sometimes goes unnoticed, however there are tools that facilitate the diagnosis and confirmation of the same as abdominal ultrasound, endoscopic retrograde cholangiopancreatography, percutaneous cholangiography or cholangio-resonance. Treatment is surgical, using a laparoscopic or open approach depending on its stage(1-3).

METHODOLOGY

A total of 45 articles were analyzed in this review, including review and original articles, as well as cases and clinical trials, of which 33 bibliographies were used because the information collected was not important enough to be included in this study. The sources of information were Cochrane, PubMed and Google Scholar; the terms used to search for information in Spanish, Portuguese and English were: Mirizzi Syndrome, obstruction, common bile duct, gallstone, hepatic duct.

The choice of bibliography exposes elements related to Mirizzi syndrome: description, etiology, epidemiology, pathophysiology, classification, histopathology, presentation, diagnosis, treatment, prognosis and complications of the disease.

DEVELOPMENT

Description

Mirizzi syndrome, also called extrinsic biliary compression syndrome, is a rare complication of chronic cholecystitis and cholelithiasis secondary to obliteration of the cystic duct or infundibulum of the gallbladder (Hartmann's pouch) caused by the impact of one or more stones. This causes crushing of the adjacent bile duct, which ends in a total or partial obstruction of the common hepatic duct to subsequently cause an alteration of hepatic functionality. Extrinsic biliary compression goes hand in hand with inflammation of the gallbladder, sometimes presenting a cholecystocholedochal fistula giving rise to a Mirizzi syndrome type II, III, IV or in the absence of it a Mirizzi syndrome type I. The origin of Mirizzi syndrome may be due to the inflammatory process following erosion caused by a gallstone impacted in the cystic duct, in the infundibulum of the gallbladder or Hartmann's pouch(4-8).

Etiology

Gallstones are usually generated by bile stasis, when bile is not completely evacuated from the gallbladder, it precipitates as sludge and therefore forms stones. Biliary obstruction can also generate gallstones, including bile duct stenosis and cancers. The most frequent origin of cholelithiasis is cholesterol precipitation. The next form of gallstones are pigmented gallstones, which are due to increased red blood cell devascularization in the intravascular system, resulting in increased concentrations of bilirubin, which then accumulates in the bile, giving a black color. The third form of gallstones are pigmented mixed stones, a mixture of calcium substrates such as calcium phosphate, calcium carbonate, bile and cholesterol. The fourth form consists primarily of calcium and occurs in individuals with hypercalcemia. When a single large gallstone or multiple gallstones impact the lower external gallbladder sac an external compression of the common hepatic duct or common bile duct may occur. This appears to correlate with a flexible Hartman's pouch, causing inflammation and leading to subsequent fistula formation(1,9).

Epidemiology

Gallstones are generally asymptomatic, the prevalence of these increases as age increases, the probability of developing them is higher in women and in obese people, because of the greater biliary secretion of cholesterol, on the other hand people who are fasting or who present a sudden considerable weight loss present a greater predisposition to the formation of gallstones because of biliary stasis. There is a connection between hormones and gallstones; estrogen generates an increase in biliary cholesterol and decreases the contraction of the gallbladder, that is why women who take contraceptive drugs containing estrogen or in

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reproductive age are twice as likely to generate gallstones compared to men. Mirizzi's syndrome is relatively uncommon, occurring in only 0.1% of patients with gallstones, with findings in 0.7% to 25% of patients who have undergone cholecystectomies. This syndrome usually presents a higher incidence in idoses, however the prevalence of cases in males or females does not seem to have a particular preference for either, the same happens with ethnicity, however more studies on the subject are needed. Mirizzi's disease presents a connection with the risk of presenting gallbladder cancer(1-3).

Anatomical variants of the cystic duct are common, and are arranged in advance of the formation of Mirizzi syndrome in 18-23%. The modification of the anatomy and the cholecystochochoediac fistula increases the risk of damage to the bile duct in the surgical act of cholecystectomy(4,10,11).

Pathophysiology

Gallstones form when the components of bile reach the limit of their solubility. As the concentration of bile in the gallbladder increases, it becomes supersaturated with these components forming small crystals. These crystals get stuck in the gallbladder mucosa, generating sediments. These crystals can increase in size and generate large and/or multiple stones. The latter sometimes generate cholecystitis symptoms, and can also cause jaundice when impacted in a flexible Hartman's pouch. With the evolution of this condition, it leads to the creation of internal fistulas from the gallbladder to the common hepatic duct (CHD), the duodenum and the common bile duct. In other words, the impacted stone plus the inflammatory response produces the obstruction of the external bile duct, whereby the mucosa will erode and form a cholecystohepatic or cholecystocholedochal fistula, which show some degree of connection between the gallbladder and the bile duct. (1,5,7,12,13).

Classification

A clinical study showed with respect to classification that type I was present in 40% of cases, type II, type III and type IV in 20% each(14).

Table 1. Algorithm of classification of the various stages of Mirizzi syndrome, assigned by categories.

I: without fistula.

- IA: presence of cystic duct
- IB: obliteration of the cystic duct.

II to IV: with fistula.

- II: defecto menor al 33% del diámetro del conducto hepático común.
- III: defecto del 33 % al 66 % del diámetro del conducto hepático común.
- IV: defecto de más del 66 % del diámetro del conducto hepático común.

Source: Jones MW, Ferguson T. Mirizzi Syndrome (1).

Histopathology

In terms of histology, findings of acute or chronic cholecystitis may be found. The gallbladder wall thickens variably and sometimes presents adhesions to the serosal surface. Smooth muscle hypertrophy is also found, mainly in prolonged chronic indoles. It is more usual to observe calcium bilirubinate or cholesterol stones, these modify their size and presentation sometimes even showing in the totality of the light of the gallbladder or becoming multiple. Disease without stones may show sludge or extremely viscous bile, which may suggest that they are precursors to gallstones and are generated from increased bile salts or stasis; normal appearing bile may also be found. Multiple bacteria appear with a frequency of 11% to 30%. In cholecystitis specimens, the so-called Rokitansky-Aschoff sinuses are present in 90%, which is practically a hernia of the intraluminal sinuses due to increased pressure probably related to the ducts of Luschka. The mucosa presents inflammation in different categories. The risk of presenting gallbladder cancer increases with Mirizzi's syndrome, the exact reason is not yet expressly established, however, it is thought to be due to the continuous and repetitive irritation of the area and also due to chronic biliary stasis. Some studies show that between 5% to 28% of individuals with Mirizzi syndrome had gallbladder cancer after cholecystectomy and all of these diagnoses were made with pathological examination of the specimens after surgery(15,16).

Presentation

The most common presentation is obstructive jaundice and right upper quadrant pain, both varying from 50 to 100% of affected individuals. Epigastric pain, dark urine, nausea, tachycardia, vomiting, anorexia, fever and chills may also be present. And it is usually associated with those who present acute cholecystitis, choledocholithiasis and pancreatitis (4,5,7,17,18).

Diagnosis

Diagnosis should begin with the usual tests for cholecystitis. Right upper quadrant abdominal ultrasound is currently the first-line study for the diagnosis of gallstones and acute cholecystitis. It has a specificity rate of 90% although it is operator dependent. Ultrasound can show stones as small as 2 mm, sludge and gallbladder polyps. The specific ultrasound findings that point to acute cholecystitis as opposed to cholelithiasis are thickening of the gallbladder wall greater than 3 mm, positive ultrasound Murphy's sign and pericholecystic fluid. Other studies such as tomography and nuclear magnetic resonance could be used, however the sensitivity is not very high for cholecystitis; in radiographs gallstones can be seen in 10% due to the fact that some of them have high calcium content, air can also be seen in the biliary tree when accompanied by an enteric fistula. In the hypothesis of calculi in the common bile duct by ultrasound, a magnetic resonance cholangiopancreatography (MRCP) should be performed. If visualized in this study, it is followed by endoscopic retrograde cholangiopancreatography (ERCP) performed by a trained gastroenterologist. Percutaneous transhepatic cholangiography (PTHC) may be helpful in the diagnosis to identify common bile duct stones if ERCP is not possible. Usually, the diagnosis of Mirizzi's syndrome is mistaken for a common bile duct stone or missed entirely postoperatively(1,19).

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Preoperative diagnosis of the syndrome is essential to avoid complications such as cholecystobiliary or cholecystoenteric fistula, as well as iatrogenic biliary damage caused by the chronic inflammatory process. Safety in the correct diagnosis is essential to choose the surgical approach technique to be used(20).

The Mirizzi's disease presents a very colorful panel of symptoms among which abdominal pain is the most important presenting an incidence of 65.7-100 %, followed by jaundice with 45-87.5 %, nausea and vomiting with 31-62 %, cholangitis reaching values up to 56 %, fever with 21-42 % and anorexia 11-29.2 %. Murphy's positive sign is evident in the physical examination at approximately 50%. The average duration of symptoms is 3 to 24 months, however some studies show that symptoms in individuals suffering from uncomplicated gallstones remain half as long as in those with Mirizzi's disease. The percentage of asymptomatic individuals is between 3.7-17%. As for laboratory tests in the group of individuals affected with Mirizzi's syndrome the most frequent are white blood cell count (WBC), aspartate aminotransferase (AST), alanine aminotransferase (ALT), bilirubin, alkaline phosphatase (ALP) and gamma-glutamyl transpeptidase (GGT). Laboratory data commonly show elevated levels of alkaline phosphatase (ALP), bilirubin and transaminase. In one study leukocytosis was shown in 73.4% of individuals with cholangitis, acute cholecystitis or pancreatitis along with Mirizzi syndrome, mean WBC levels are usually near the upper limit of normal levels or higher. ALT and AST levels are elevated in 39-98% of tests for ALT and between 37-89% for AST. Some literature shows mean ALT and AST levels many times higher than the normal level reaching 286 and 263 U/L, respectively. However, studies report a significant drop in AST and ALT levels from more than 250 U/L to less than 100 U/L in cholecystobiliary fistula, while other studies show an exponential increase in the levels of the parameters with the progression of the fistula, however neither value exceeds 90 U/L. The ALP test increases in up to 93.8% of patients with average levels of 324-402 U/L, and can reach up to 1236 U/L. Total bilirubin values are elevated in 92.2%. Mean bilirubin levels are commonly reported to range from 2 to 9.9 mg%. The literature is consistent in GGT levels, elevated to mean values of 204-1018 U/L (4,17,21-26).

In recent years, high levels of the tumor marker CA 19-9 with values greater than 20,000 U/mL have been shown in patients with Mirizzi type II or higher categories of the syndrome; however, there are few reported cases and therefore CA 19-9 is not useful as a screening test due to its low sensitivity in early stages. Hyperbilirubinemia could be a confounding factor because it is associated with higher CA 19-9 values (4,5,27).

Among the differential diagnoses there are several conditions, among the most common that present similar symptoms are(1,28):

- Peptic ulcer disease.
- Pulmonary embolism.
- Inflammatory bowel disease.
- Irritable bowel disease.
- Gastroesophageal reflux disease.

The presence of jaundice in combination with the other symptoms that occur in Mirizzi syndrome is usually obfuscated with different etiologies such as:

- Common bile duct stones.
- Pancreatic tumors.
- Ascending cholangitis.
- Biliary cancer.

Other clinical conditions to keep in mind that can pass for Mirizzi's syndrome are:

- Acute hepatitis.
- Drug-induced hepatitis.
- Ischemic liver disease.

Treatment

Conventionally the treatment of Mirizzi syndrome is surgical, being effective in most cases, however some patients between 8-25% may require restoration of the bile duct by fistulization of the main bile duct. Cholecystectomy is the first line treatment in patients with Mirizzi syndrome, and the specific technique will vary according to the classification of the subtype. Several bibliographies contraindicate laparoscopic cholecystectomy in Mirizzi syndrome due to the risk of adhesions and inflammatory tissue in the triangle of Calot, which when dissection is attempted can lead to an unnecessary alteration in the biliary tract. On the other hand, other surgical literature assures that the laparoscopic technique is feasible, however the technique can be challenging. In those individuals with more advanced disease, a partial cholecystectomy is considered, which involves leaving the Hartman's pouch in place and removing the gallstones and the body of the gallbladder. This procedure decreases the incidence of damage to the bile ducts and hepatic portal. There is literature demonstrating that in case of fistula, open cholecystectomy with bilioenteric anastomosis, possibly with a Roux-en-Y is effective. In the surgical procedure it is advisable to methodically dissect the biliary structures, observe the common bile duct, establish the type and location of the fistula, relieve the obstruction, reconstruct the defect and provide appropriate drainage of the biliary tract(4,13,18,28-30).

Prognosis

In those individuals who do not present fistula, the outcome of surgical treatment is known to be positive. Because of the distorted anatomy and the frequent rate of conversion to open cholecystectomy in these patients, some authors advise an open surgical approach for all individuals presenting Mirizzi's syndrome. In those individuals presenting with fistula the prognosis depends on the type of treatment that goes hand in hand which can be:

- Placement of a T-tube in the middle of the fistulas that are small or intermediate.
- Biliary bypass plus choledochochojejunostomy.

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- Roux-en-Y placement for larger fistulas.

It should be taken into account that the longer the surgery and hospitalization time, the higher the risk of complications and at the same time the higher the mortality and morbidity. In individuals with several comorbidities in addition to a high risk of intraoperative complications, non-surgical methods should be chosen to reduce the risk related to the surgical intervention. In elderly patients with multiple comorbidities and high risk of surgical complications, non-surgical methods should be considered to minimize the morbidity associated with surgery. For prognosis, all factors must be taken into account since gallbladder cancer has also been associated with Mirizzi syndrome(1,31,32).

- Complications.
- Among the most frequent complications of gallstone disease are:
- Acute cholecystitis.
- Acute pancreatitis.
- Ascending cholangitis.
- Gangrenous gallbladder.

Among the least frequent within the complications of gallstone disease are:

- Mirizzi's syndrome.
- Cholecystocoledocholedochal fistula.
- Biliary ileus.

Among the most frequent complications of Mirizzi syndrome due to prolonged inflammation are:

- Formation of cholecystobiliary fistulas.
- Formation of cholecystoenteric fistulas.

Among the less frequent complications of Mirizzi's syndrome due to prolonged inflammation are the following:

- Formation of cutaneous fistulas.
- Secondary biliary cirrhosis.
- Biliary stenosis of late onset.

Among the surgical complications in correlation with prolonged procedure time due to dense adhesions:

- Bile duct injury.
- Hemorrhage.
- Massive hemorrhage in Calot's triangle dissection.

All these are complications that can be seen in individuals with Mirizzi's syndrome(1,4,33).

CONCLUSIONS

Mirizzi syndrome, also called extrinsic biliary compression syndrome, is a rare complication of chronic cholecystitis and cholelithiasis secondary to obliteration of the cystic duct or infundibulum of the gallbladder (Hartmann's pouch) generated due to the impact of one or more stones. The impacted stone plus the inflammatory response produces the obstruction of the external biliary tract, with which the mucosa will erode forming a cholecystohepatic or cholecystocholedochal fistula. Mirizzi's syndrome is relatively infrequent, appearing in only 0.1% of patients with gallstones, with findings in 0.7% to 25% of patients who have undergone cholecystectomies. Between 5% to 28% of individuals with Mirizzi syndrome had gallbladder cancer following cholecystectomy. The modification of the anatomy and the cholecystocholedocholedochal fistula increases the risk of damage to the biliary tract in the surgical act of cholecystectomy. In terms of classification, type I was present in 40% of the cases, type II, type III and type IV in 20% each. The most frequent presentation is obstructive jaundice and right upper quadrant pain accompanied by epigastric pain, dark urine, nausea, tachycardia, vomiting, anorexia, fever and chills. The beginning of the diagnosis should be made with the usual tests for cholecystitis. Right upper quadrant abdominal ultrasound is currently the first-line study for the diagnosis of gallstones and acute cholecystitis. If a gallstone in the common bile duct is suspected by ultrasound, magnetic resonance cholangiopancreatography should be performed. This is followed by endoscopic retrograde cholangiopancreatography (ERCP) performed by a trained gastroenterologist. Conventionally the treatment of Mirizzi syndrome is surgical. Cholecystectomy is the first-line treatment; in case of fistula, open cholecystectomy with bilioenteric anastomosis, possibly with a Roux-en-Y is effective. In individuals without fistula, the prognosis is usually positive. In elderly patients with multiple comorbidities and high risk of surgical complications, non-surgical methods should be considered to minimize the morbidity associated with surgery.

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Conflict of Interest Statement

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