

SELF ASSESSMENT ANSWERS

Treatment options for common bile duct stones

Q1: What are the treatment options for this patient?

These are summarised in fig 1. Endoscopic extraction of common bile duct stones after sphincterotomy and mechanical lithotripsy has a success rate of up to 95% and is considered the treatment of choice.^{1,2} The reason for failure in this case was the large size of the bile duct calculus. Other reasons include bile duct strictures, unusual anatomy, and calculi beyond reach of the wire basket.¹⁻³

Traditionally such patients have been referred for surgical exploration of the common bile duct but this procedure is not without risk, particularly in elderly patients or those with major medical comorbidities.⁴

Extracorporeal shock wave lithotripsy (ESWL) was investigated initially for treatment of gallbladder stones, but a high stone recurrence rate has limited its use in this condition.⁵ In recent years high energy ESWL has been used with more promising results in high risk patients with common bile duct stones.

In this case, given the patient's age and comorbidities it was decided that this was the treatment of choice. Biliary drainage was achieved during initial ERCP using a pigtail stent.

The patient then underwent one session of high energy ESWL, during which the calculus was targeted by ultrasonography.^{1,2} Studies have shown that between 20% and 50% of patients will require more than one treatment

session.^{3,6,7} The success rate of this procedure, with complete clearance of the common bile duct is between 80% and 90%.^{1-3,6}

The main complication is cholangitis (1%-8%) and this is reduced by use of prophylactic antibiotics.^{1,6} Procedure related mortality has not been reported.

Q2: What does the post-treatment ERCP film (fig 1 in questions; see p 178) show?

At repeat ERCP the pigtail stent was removed and the cholangiogram shows no evidence of calculi with satisfactory drainage from the common bile duct.

Spontaneous passage of calculi occurs in up to 10% of patients, with 80% requiring removal of stone fragments during repeat ERCP.¹ Although recurrence of bile duct calculi is estimated at 14% after one year, most of these are amenable to endoscopic treatment.²

ESWL is an effective non-invasive treatment modality that can be performed safely on an outpatient basis, without use of general anaesthesia. For this reason it is a useful treatment option in patients with difficult common bile duct calculi who are considered to be poor candidates for surgery.

Final diagnosis

Extracorporeal shock wave lithotripsy as a treatment option for common bile duct calculi.

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clearance of bile duct stones resistant to endoscopic extraction. *Gastrointest Endosc* 2001;53:27-32.

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An unusual cause of persistent vomiting

Q1: What abnormality is shown on the barium meal (see p 178)?

An abnormal filling defect in close association with a thin C-shaped radio-opaque strip can be seen in the gastric remnant (grey arrow). Radio-opaque clips can also be seen around the cardia (white arrow). These appearances are in keeping with an Angelchik prosthesis which has become detached and eroded into the stomach.

Q2: What are the options for dealing with this complication?

Gastroscopy should be performed not only to confirm the diagnosis but also in an attempt to remove the prosthesis endoscopically.¹ At gastroscopy the stomach mucosa appeared normal and the silicon prosthesis, although covered with debris, was easily visualised. However, despite several attempts, it was not possible to retrieve the prosthesis endoscopically. The only other option was to remove the prosthesis surgically and this was performed in this case by carrying out a laparotomy and gastrotomy. The patient made an uncomplicated recovery and was free of symptoms at review three months later.

Q3: What other complications have been reported after insertion of this prosthesis?

The Angelchik prosthesis is no longer widely used because of the high incidence of complications. Intractable dysphagia was common and often required removal of the prosthesis. Free extraluminal migration into the abdominal cavity can occur. The prosthesis usually comes to rest in the pelvis and is usually manifested by chronic lower abdominal pain or urinary symptoms. Migration into the mediastinum and distal slippage has also been reported. Erosion of the prosthesis into the oesophagus can lead to abscess formation and intraluminal erosion may even progress to cause small bowel obstruction.

Discussion

In 1979, Angelchik and Cohen reported a series of 46 patients who had reflux oesophagitis treated surgically with the insertion of an incomplete "doughnut" shaped ring of silicon around the gastro-oesophageal junction: the

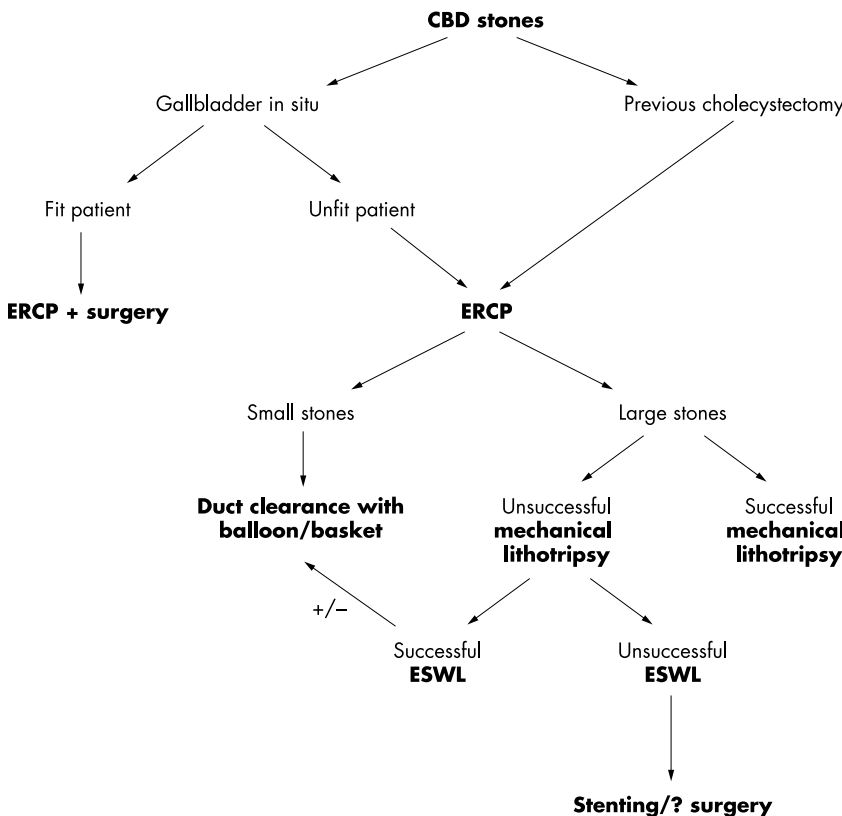


Figure 1 Treatment options for common bile duct stones (CBD, common bile duct; ERCP, endoscopic retrograde cholangiopancreatography; ESWL, extracorporeal shockwave lithotripsy).

Angelchik prosthesis. The C-shaped ring was tied around the lower oesophagus with Dacron straps. Insertion of this prosthesis was quick, simple to perform, and standardised and it was hoped that it would become superior to the other surgical antireflux procedures available at the time which were all technically difficult, time consuming, and had variable results which were operator dependent.²

The early short term results for the prostheses were promising, and objective and subjective outcome measures were similar to other accepted surgical antireflux procedures.³ However the initial enthusiasm has been tempered with experience and when more long term results were analysed up to 20% of prostheses had to be removed for intractable dysphagia and there were other reports of significant problems due to migration and erosion of the prosthesis.^{4,5}

The prosthesis has a tantalum radio-opaque marker encircling its periphery and radio-opaque clips were frequently used to reinforce the knot in the tied Dacron straps making it possible to identify the prosthesis on radiological images as seen in this case.

The continued use of the Angelchik prosthesis was abandoned in most centres over 10 years ago but at least 25 000 have been inserted worldwide. In this case erosion occurred 14 years after insertion, longer than any other case reported. The presentation of new gastrointestinal symptoms should still today arouse suspicion in a patient known to have an Angelchik prosthesis.

Final diagnosis

Eroded Angelchik prosthesis.

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A limping 6 year old child with no history of illness or trauma

Q1: What is the likely diagnosis and how does it typically present?

Legg-Calvé-Perthes (LCP) disease, also known as idiopathic avascular necrosis of the proximal femoral epiphysis, is one of the more insidious causes of a limp in the paediatric population. It commonly presents between the ages of 5 and 7, although it has been reported in children ranging from the ages of 2 to 16. Although its aetiology remains unclear, it is three times more common in boys and has been shown to be associated with protein C and protein S deficiencies as well as various other thrombophilias.

Clinically, LCP disease presents with a slowly developing and gradually worsening limp, that may or may not be painful. Associated pain is usually activity related and

relieved by rest. The pain is usually localised to the groin, inner thigh, or knee region. On examination, these children will have a positive Trendelenburg test and have limited abduction and difficulty medially rotating their affected leg. Often, they also have trouble with hip motion. With disease progression, necrosis of the femoral head leads to further degeneration and immobilisation of the hip, which can then progress to disuse atrophy of the buttocks, thigh, and calf muscles.¹

Q2: What are alternative differential diagnoses in a child with a limp?

The differential diagnosis list for a child with a limp is quite extensive and can include various hormonal, metabolic, orthopaedic, and genetic causes. Frequent causes of limp also vary by age group. In the 4–11 year old age group, the most common causes of a limping gait are: septic arthritis, osteomyelitis, tarsal coalition, transient monoarticular synovitis, juvenile rheumatoid arthritis, LCP, slipped capital femoral epiphysis, and neoplasias such as osteoid osteomas or osteochondromas.² Many of these common causes can be diagnosed by laboratory evaluation such as erythrocyte sedimentation rate for transient monoarticular synovitis, rheumatoid factor and antinuclear antibody testing for juvenile rheumatoid arthritis, and presence of a febrile course for septic arthritis or osteomyelitis.

Discussion

Epidemiologically, LCP disease has been shown to be associated with a myriad of external factors such as lower socioeconomic group, advanced maternal age, later born children, short stature, low birth weight, delayed bone age, and breech birth. LCP also demonstrates a higher incidence in Asian and eastern European populations and a decreased incidence in African-Americans and native Americans. Trauma can be related to the onset of symptoms but is not implicated as a direct cause.

LCP disease is diagnosed and staged by radiography. Radiographs should be taken in the anteroposterior and frog-leg lateral position and are used to determine the progressive stage of the disease. The four classification stages for LCP disease are (1) initial, (2) fragmentation, (3) reossification, and (4) healing.³ Radiographic findings can also be used to determine prognosis. Poor prognosis is generally associated with the degree of deformity of the femoral head and acetabulum, female sex, and age of onset after 8 years of age. Definitive diagnosis of LCP must be done by technetium bone scan or magnetic resonance imaging to show the avascular necrosis of the femoral head and subsequent acetabulum-femoral deformity.

Therapy for LCP disease depends on the child and severity of the illness, active treatment is not required for all children. The main treatment, when indicated, is containment of the femoral head within the acetabulum by casting, immobilisation, or more invasive surgical methods. In the majority of children, LCP disease is a local, self healing disorder. The acetabulum undergoes bony remodelling with subsequent improvement in femoral head deformities. The long term prognosis is directly related to the onset of symptoms and degree of residual femoral head deformity and results in an increased incidence of degenerative osteoarthritis in adulthood. It was recently shown that up to 80% of LCP disease patients who underwent containment, remained active, pain-free, with a good range of motion up to 40 years after onset of symptoms, regardless of radiographic appearance.⁴

Final diagnosis

Legg-Calvé-Perthes disease.

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Middle aged man with groin pain

Q1: What abnormality is shown and what is the diagnosis?

We can see a contrast filled oblong sac on the left inguinal region (fig 1; see p 179). This is herniation of the bladder in forming the wall of the direct inguinal hernia.^{1,2}

Q2: How do you manage this problem?

The management of this condition is in two parts:

(A) Repair of the inguinal hernia, which is the main cause of the pain, using a form of wire mesh to reinforce the posterior wall of the inguinal canal to prevent recurrence of the hernia.¹

(B) Repair of the bladder diverticulum by open diverticulectomy or laparoscopic diverticulectomy.^{3,4}

Q3: What complications may arise during the management of such patients?

If the surgeon is unaware of the possibility of herniated bladder forming hernial sac, the bladder may be inadvertently damaged during the hernia repair.

Discussion

This is a rare condition where there is herniation of the bladder in forming the wall of the direct inguinal hernia,¹ and only about 100 cases have been reported world wide.^{5,6}

The treatment of this condition depends on the diagnosis and divided into two parts: repair of the hernia and repair of the diverticulum (see answer to question 2 above).

Final diagnosis

Herniation of the bladder.

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An elderly man with chest pain, shortness of breath, and constipation

Q1: What do the chest radiographs show?

The upright posteroanterior and lateral films (see p 180) show a marked elevation of the right hemidiaphragm with distended loops of bowel interposed between the liver and right abdominal wall. Haustration identifies the large bowel, distinguishing colonic hepatodiaphragmatic interposition from subphrenic pneumoperitoneum or abscess. The lung fields and pleural spaces are clear. Note the normal heart size and median sternotomy wires. Interestingly, the hepatodiaphragmatic interposition of the right colon had not been seen on films taken five and seven years previously, but persisted after complete resolution of the symptoms and was still present on follow up two weeks later. The computed tomogram confirmed the hepatodiaphragmatic interposition of the colon and did not show any signs of pulmonary embolism.

Q2: What important physical sign may have been missed?

Absent liver dullness may be a useful diagnostic clue pointing to hepatodiaphragmatic interposition.

Q3: What is the differential diagnosis?

The differential diagnosis must include a number of cardiac and non-cardiac causes of chest pain and breathlessness. The initial approach would be to rule out life threatening causes such as myocardial ischaemia and pulmonary embolism as well as acute pneumonia. In this patient the diagnostic difficulty was compounded by the history of coronary artery disease and high risk of pulmonary embolism (postoperative state and malignancy). The pain occurred on walking (characteristic for angina), was intensified by inspiration (mimicking pleuritic pain typical for pulmonary infarction), and was associated with shortness of breath and hypoxia. However, the pain lasted from 30 minutes to two hours, was not associated with new ECG changes nor elevation of cardiac enzymes, it responded to bed rest (but not to nitrates), and a computed tomographic pulmonary angiogram revealed no embolism. Absence of fever, productive cough, sputum, and the chest radiograph findings tend to exclude pneumonia. The patient became asymptomatic when constipation was successfully treated with enemas, stool softeners, and laxatives. However, to verify that colonic hepatodiaphragmatic interposition is the only cause of angina-like pain, other investigations may be needed to exclude coronary ischaemia.

Q4: What were the predisposing factors to this condition?

Colonic elongation due to longstanding constipation and probably adhesions (after prior stomach surgery) in an elderly patient (after hip fracture) with limited mobility taking a narcotic analgesic.

Q5: What is the management of this condition?

Most patients are treated conservatively with bed rest, increased fluid intake, fibre supplementation, laxatives, and enemas. In rare complicated cases (volvulus, internal hernias,

intestinal obstruction, subdiaphragmatic appendicitis) appropriate surgical intervention is required. In our patient, who was treated conservatively, the symptoms completely disappeared after he opened his bowels with no recurrence or complications on follow up (for two months) since treatment of constipation started.

Discussion

A temporary or permanent hepatodiaphragmatic interposition of the colon, small intestine (rare), or stomach (exceptionally rare) is an uncommon and usually asymptomatic process. However, it can cause serious complications and be a potential source of misdiagnosis for a variety of intrathoracic and intra-abdominal disorders.

The condition was named after Viennese radiologist Demetrius Chilaiditi who in 1911 reported three asymptomatic cases with temporary hepatodiaphragmatic interposition of the colon and described their anatomoradiographic aspects. The condition was first described by Cantini in 1865, and Beclere in 1899 presented the necropsy and roentgenological findings in a patient thought to have a subdiaphragmatic abscess.^{1,2} It has been proposed to use the term "Chilaiditi's sign" in asymptomatic patients and the term "Chilaiditi's syndrome" in symptomatic patients.^{3,4}

In Western countries the condition is uncommon and is found in 0.02% to 0.2% of routine chest radiographs with a male to female ratio of 4:1.^{1,5,6} In some reports the incidence is even lower with only 0.002% (one of 50 000 adults⁷) or 0.00003% (three of 11 378 000⁸). Importantly, an increase in prevalence of hepatodiaphragmatic interposition has been recorded in patients above 65 years of age: from 0.02% to 0.2% in men and from 0.006% to 0.02% in women.⁵ In one study the prevalence in the geriatric population was found to be 1%.⁹ In 135 persons with learning disabilities in New York, the incidence was 8.8%, or 63 times that in general population.¹ A high incidence of hepatodiaphragmatic interposition was observed in Iran: 0.22% in the normal population, 2% in women near term pregnancy, 2.7% in patients with chronic lung disease, and 22% in patients with postnecrotic cirrhosis.¹⁰

The normal anatomy and physiology of the intestine, liver, and diaphragm usually prevent hepatodiaphragmatic interposition. The pathophysiology is thought to be multifactorial and includes an enlarged subphrenic space, congenital and/or acquired elongation, malrotation or malfixation of the intestine with increased mobility, laxity of the hepatic suspensory ligaments, reduction of liver volume, and weakness (hyper-relaxation) of the diaphragm (due to defective innervation, either centrally or peripherally mediated).¹ Chilaiditi emphasised hepatic mobility as the primary pathophysiological cause of hepatodiaphragmatic interposition, while others postulated that a redundant colon with increased mobility is a prerequisite for development of this condition.¹ Chronic constipation, meteorism, aerophagia, adhesions, and mechanical obstructions are considered as important factors.^{3,4,8} It is worth noting that chronic constipation is the most common cause of colonic elongation and redundancy, leading to increased colonic mobility.³ Predisposing factors to intestinal hepatodiaphragmatic interposition are shown in box 1.

There are different anatomic types of intestinal hepatodiaphragmatic interposition. Most frequently it occurs under the diaphragm

Box 1: Factors predisposing to intestinal hepatodiaphragmatic interposition

1. Anatomical

- Congenital elongation, malrotation, or malfixation of the bowel.
- Redundant bowel with a long mesentery.
- Congenital or acquired laxity of hepatic suspensory ligaments.
- Reduction of liver volume (lobar agenesis, atrophic cirrhosis).
- Lower thoracic outlet enlargement with high abdominothoracic pressure gradient (pregnancy, obstructive airway disease, emphysema, scoliosis, ascites).
- Adhesions and mechanical obstruction.
- Obesity.

2. Functional

- Increased intestinal mobility.
- Longstanding constipation (due to immobilisation, diet, medications, etc).
- Gaseous distention of the intestine (meteorism).
- Diaphragm paralysis (centrally mediated or due to phrenic nerve injury).
- Aerophagia.

anterior and superior to the right lobe of the liver (as in the described case). Hepatodiaphragmatic interposition in the posterior subphrenic space is much rarer. A case of combined anterior and posterior types of colon displacement has been reported.¹¹

Only a minority of patients with intestinal hepatodiaphragmatic interposition have symptoms. These range from non-specific gastrointestinal symptoms such as nausea, anorexia, vomiting, flatulence, and constipation to signs of pseudo-obstruction and rarely to life threatening complications like volvulus or intestinal obstruction. The condition may rarely present with central chest pain, cardiac arrhythmias, or respiratory distress.⁷ Symptoms probably occur only when the intestine is distended or obstructed. Because the accumulated gas naturally ascends the patients usually become symptomatic in a sitting position or standing upright while bed rest diminishes the symptoms.¹ Our case is unusual in that the patient developed uncommon symptoms of chest pain and breathlessness (without gastrointestinal symptoms apart from constipation). After complete resolution of clinical symptoms, in many cases the radiological picture of intestinal hepatodiaphragmatic interposition remained unchanged (as in our patient), indicating the importance of colonic distention in the pathophysiology of the syndrome.

A wide range of coexisting disorders has been reported including hiatus hernia, skeletal abnormalities (spinal scoliosis), multiple congenital anomalies, obesity, pneumatosis cystoides intestinalis, melanosis coli, and lung cancer. Severe complications requiring surgery such as sigmoid volvulus, incarceration of colon, and suprahepatic appendicitis have been reported.^{5-7,12,13}

On physical examination a marked decrease or even absent liver dullness and/or a "mass" in the right upper quadrant or mid-abdomen (displaced liver) may be diagnostically useful.

Radiologically three signs are characteristic: (1) colon or small bowel interposed between the liver and the diaphragm (in symptomatic patients usually markedly distended), (2) elevated right hemidiaphragm, and (3) caudal and medial displacement of liver. However, when the bowel gas is lateral and posterior it may not get above the liver or displace it; this is termed incomplete hepatodiaphragmatic interposition.¹⁴

The differential diagnosis of radiographic findings include subdiaphragmatic abscess, pneumoperitoneum, cysts in pneumatosis intestinalis, hepatomegaly, posterior hepatic lesions, and retroperitoneal masses. In the first two of these conditions, which are associated with elevation of the right hemidiaphragm and subdiaphragmatic air collection, the haustral markings (usually best seen on lateral films) are absent. With pneumoperitoneum the free air is shifting (usually obvious in the lateral decubitus view) and may be bilateral. In subdiaphragmatic abscess, the air-fluid level is smaller and often associated with basal atelectasis and pleural effusions. Hepatodiaphragmatic interposition of the intestine may also be diagnosed with abdominal ultrasound.¹⁵ If doubt remains, contrast enema, thoracoabdominal computed tomography or nuclear scintigraphy are recommended.

Chilaiditi's syndrome may present with a wide range of symptoms and signs which could be misleading to the attending clinician. The entity may mimic a number of cardiac, respiratory, and other non-cardiac disorders. The clinical differential diagnosis may be particularly difficult in elderly persons because of the frequent coexistence of two or more conditions contributing to the clinical picture (as in the described case). Although in patients with chest pain, the differential diagnosis initially must include myocardial ischaemia, pulmonary embolism, aortic dissection or pericarditis, other types of non-cardiac chest pain should also be considered. Constipation and colonic distension as a cause of precordial pain,¹⁶⁻¹⁸ hypoxia, and respiratory distress^{19,20} has been described but infrequently diagnosed. Constipation which is common in elderly people affecting a third of elderly women and a quarter of elderly men,²¹ is a significant predisposing factor for intestinal hepatodiaphragmatic interposition.

This case emphasises the importance of considering Chilaiditi's syndrome in differen-

Box 2: Learning points

- Intestinal hepatodiaphragmatic interposition is a rare condition recognisable by chest radiography and is most often asymptomatic (Chilaiditi's sign).
- When symptomatic (Chilaiditi's syndrome) the interposition may present with a variety of clinical symptoms and signs, mainly gastrointestinal, but also with chest pain and dyspnoea.
- Certain groups are predisposed to this condition (elderly persons with constipation, the intellectually disabled, patients with chronic lung disease, emphysema, cirrhosis, and pregnant women).
- In elderly patients the differential diagnosis of chest pain and respiratory distress should include Chilaiditi's syndrome among other gastrointestinal disorders.
- The treatment is usually conservative (bed rest, increased fluid and fibre intake, laxatives, enemas), although rarely surgical intervention is needed (volvulus, obstruction).

tial diagnosis of chest pain and dyspnoea, especially in elderly people, as well as in patients with intellectual disability, chronic lung disease, or cirrhosis.

Final diagnosis

Colonic hepatodiaphragmatic interposition (Chilaiditi's syndrome).

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