Diagnostic Approach and Treatment of Choledocholithiasis

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ABSTRACT

Choledocolithiasis may cause acute cholangitis which is life-threatening condition. It has non specific clinical signs from mild to severe condition such as septicemia. Diagnostic and treatment modalities had developed a great deal recently. Therapeutic options include endoscopic retrograde cholangio pancreatography (ERCP), common bile duct exploration (CBDE), laparoscopic CBDE and stone retrieval. The important thing is to choose the appropriate method for each patient. We reported a case of choledocolithiasis in 40 years old, male patients who was clinically diagnosed as acute cholangitis. Diagnostic approach to find the etiology was done. Abdominal USG and CT were performed and showed multiple stones in gallbladder and intrahepatic biliary duct, suspected mass at caput of the pancreas and hepatomegaly. The ERCP showed dilatation of intra and extra hepatic biliary ducts with multiple stone in common bile duct (CBD), hepatic duct and gallbladder. The stent was placed for biliary drainage. The patient underwent cholecystectomy per laparoscopy, but further evaluation of the cholangiography still showed the presence of stones in intrahepatic biliary duct. Laparotomy exploration of CBD was done and it revealed multiple stones and dilatation of distal CBD. Surgical treatment selected for this case was choledocojejunostomy.

Keywords: *Choledocolithiasis, CBD, diagnostic approach*

INTRODUCTION

Choledocholithiasis is defined as the presence of stones in the common bile duct.[1] It can cause acute cholangitis which is a potential life-threatening condition. The clinical symptoms may be asymptomatic, but the likelihood of symptoms is significantly higher than for gallbladder stones. These are typically secondary to stones in the gallbladder and occur in about 15% of patient with gallbladder stones.[1,2] The majority of stones in the common bile duct have migrated from the gallbladder and are associated with calculous cholecystitis. Their composition reflects that of the primary gallbladder stones, being mainly cholesterol stones in the West and in the Far East, primary stones in the biliary tree are seen particularly in the context of parasitic or bacterial infection in the bile duct and are mainly of the mixed (brown stone) type.[2,3]

In the US, the incidence rate for gallstones is 10% to 20%. Approximately 600,000 cholecystectomies are performed in the US every year and choledocholithiasis

complicates 10% to 15% of this cases.[4] The clinical presentation varies depending on the degree and level of obstruction and on the presence or absence of biliary infection.

Effects of common bile duct stones[2]

Bile duct obstruction is usually partial and intermittent since the calculus exerts a ball-valve action at the lower end of the common bile duct. In the anicteric form, histologic feature of the liver virtually normal. While in the icteric form, it shows cholestasis. In chronic cases, the bile ducts show concentric scarring and eventually secondary sclerosing cholangitis and biliary cirrhosis.

Cholangitis, may spread to the intra-hepatic bile ducts and in severe and prolonged infections, cholangitic liver abscesses are seen. Escherichia coli are the most common etiologic cause. Others include klebsiella, streptococcus, bacteroides and clostridia.[5] Acute or chronic pancreatitis may result from stones wedged in or passing through the ampula of vateri.

Clinical syndromes [1,2,3,4]

The typical clinical manifestation, occurring in 70% of cases consists of biliary pain, jaundice, chills and rigors (i.e. Charcot's triad). Pain, usually has characteristic biliary pain involving the central upper abdomen, occurs in 90% of patients. The chills and fever due to bacteremia occur in 95% of patients. Clinical jaundice is present in 80% of cases. Clinical signs are nonspecific with mild hepatomegaly, tenderness and occasionally rebound tenderness. Depending on the illness progression, endotoxemia with shock or multiple liver absceses may result in hypotension, mental confusion and renal failure as preterminal events.

Elevation of alkaline phosphatase and γ-glutamyl transpeptidase (γ–GT) is characteristic, as mild jaundice with a hyperbilirubinemia between 2 and 5 mg/dL but rarely more than 10 mg/dL. Choledocholithiasis may result in dilatation of the extrahepatic and the intrahepatic bile ducts. However the absence of bile duct dilatation does not exclude the diagnosis of choledocholithiasis.

Diagnostic Modalities in Choledocholithiasis

There are so many modalities to diagnose choledocholithiasis include abdominal ultrasonography (USG), endoscopic ultrasonography, CT scan, magnetic resonance cholangio pancreatography (MRCP), cholangiography, endoscopic retrogade cholangio pancreatography (ERCP) and PTC. [1,4,6-9]

Management

Management options for common bile duct stones have developed in the era of laparoscopic cholecystectomy (LC). Therapeutic options include: preoperative ERCP with or without endoscopic sphincterotomy (ES), open common bile duct exploration (CBDE), laparoscopic CBDE and stone retrieval, postoperative ERCP with or without ES; and expectant management. Selecting the most appropriate method for each individual patient can be problematic in light of the numerous options. Compounding this uncertainty is the difficulty of accurately predicting which patient has choledocholithiasis. [6-8,10-12]

Prognosis

The prognosis of choledocholithiasis depends on the presence and severity of complications. Of all patients who refuse surgery or are unfit to undergo surgery, 45% remain asymptomatic from choledocholithiasis, while 55% experience varying degrees of complications.[4]

CASE REPORT

Male patient, 40 years old, came to hospital with chief complain of epigastric pain since one month before admission. The pain was episodic, severe and may radiate sometimes in the right upper quadrant of the abdomen. He also suffered from fever, nausea and vomiting. He had yellow pigmentation in the sclerae and his skin. He reported that his urine was dark coloured and the stool was clay coloured.

He had been hospitalized in other hospital for about two weeks before he came to our hospital. At first he was diagnosed typhoid fever and hepatitis A. The result of abdominal USG was "choledocholithiasis". He was treated with antibiotics cefotaxim, gentamycin, metochlorpramide and Hp Pro (hepatoprotector). He referred to Jakarta for further treatment.

We repeated abdominal USG examination and the result were chronic cholecystitis, chronic liver disease with differential diagnosis of diffuse mass in the liver, hidronephrosis and also pleural effusion in the right lung. There was no history of blood transfusion and surgical procedure but he had suffered hepatitis and malaria before. He had no family history of hepatitis or gallstones. The patient worked as a government employee, low fat diet and routinely exercises.

From the physical examination, the general condition of the patient looked moderately ill, weakness, fully alert and jaundice. His blood pressure was 110/70 mmHg, respiratory rate 18 times per minute, pulse rate 80 times perminute and body temperature was normal. The

conjunctivae were not pale and the sclerae was icteric. Lymph nodes weren't palpable. During chest examination, we found rales on the right lung. Heart sound was normal and regular, there was neither murmur nor gallop. Abdomen was not distented, decreased skin turgor. The Murphy's sign was positive and made the liver examination difficult to be done. The spleen wasn't palpable, shifting dullness was negative and bowel sound was normal. On the extremities, there were neither edema nor signs of liver cirrhosis such as palmar erythema.

Laboratory results on January 19th, 2004 at emergency room were as follows: hemoglobin level 8 g/dL, hematocryte 22%, leukocyte 22,000/µL, platelet count of thrombocyte 446,000, BUN 276 g/dL, serum creatinine 2.5 g/dL, ALT 28 µ/L, AST 11 µ/L, albumin level 2.8 mg/dL, blood glucose 94 mg/dL. The blood electrolyte examination revealed sodium 124 mEq/L, potassium 4.3 mEq/L and chloride 98 mEq/L. The laboratory result were positive IGM anti HAV, direct bilirubin level of 4.9 mg/dL, indirect bilirubin level of 0.6 mg/dL, ALT 122 µ/L, AST 248 µ/L, alkaline phosphatase 551 µ/L, γ–GT 593 µ/L. Chest X-ray showed that cardiomegaly and there were infiltrates at both lungs. Electrocardiography was normal. Abdominal USG revealed choledocholithiasis.

The patient had been firstly diagnosed obstructive jaundice with suspect of acute cholangitis based on clinical symptom of jaundice, epigastric pain, fever, nausea and vomiting. The urine was dark coloured and the stool was clay coloured. During physical examination it was found that Murphy's sign was positive. From laboratory result, there was elevation of direct bilirubin more than indirect bilirubin and also elevation of alkaline phosphatase and γ–GT value. Abdominal USG which was performed at first hospital showed choledocholithiasis. The laboratory result of renal function also showed an elevation of BUN and serum creatinine. Thus, he was also diagnosed acute renal failure which might be due to dehydration.

During physical examination, there were rales on the right lung. Laboratory result was leucocytosis and the hest X-ray examination showed infiltrates in the lungs. Thus, we diagnosed him to have pneumonia with differential diagnosis of pulmonary tuberculosis. He was also diagnosed hiponatremia because sodium level was 124 mEq/L. We planned to repeat laboratory examinations of bilirubin level, alkaline phosphatase and γ–GT level. We planned to repeat abdominal USG at Hepatology department.

The patient was given intra venous fluid drip of normal saline. He was also given antibiotics cefoperazon tablet 1 g twice daily, ranitidine ampulla twice daily supplementation tablets of folic acid , bicarbonate natricus and vitamin B_{12} three times daily and sucralfat.

On the 2nd day of hospitalization, abdominal USG showed suspicion of chronic cholecystitis, chronic liver diseases with diffuse mass on liver, hidronephrosis and pleural effusion in the rigt lung. The result of direct bilirubin level 4.1 mg/dL and indirect bilirubin level 2.3 mg/dL, alkaline phosphatase 304 µ/L, γ-GT 99 µ/L, blood electrolytes revealed sodium 134 mEq/L and potassium 3.4 mEq/L.

On the 5th day, he was moved from intermediate ward to internal medicine ward and we repeated laboratory examination of BUN and serum creatinine level. The result were BUN 220 mg/dL and serum creatinine 12.3 mg/dL. The urinalysis test showed positive proteinurea, mild glucosuria, leukocyte 3-4, erythrocyte 0-1 and positive epithel.

Case discussion at kidney and hypertension department had concluded that acute renal failure in this patient was due to dehidration but we must also consider another factor such as drug toxicity, leptospira and malaria infection.

We performed renal ultrasonography on the 8th day and the result are both of the kidneys showed acute process, urinary bladder and prostate were normal. We gave him positive fluid balance therapy and there was good clinical respone. The creatinine clearance test result (CCT) 13 cc/minute with proteinuria 975 mg/24 hours and diuresis about 1500 cc/24 hours.

The sputum examination was negative. The PAP TB result was negative and repeated chest X-ray which was normal. We gave him cefoperazone 1 gram twice daily for 10 days and switch to ciprofloxacin 500 mg twice daily. He still had jaundice and felt nausea. Repeat of abdominal USG was conducted and the result were as follows: distension of biliary system, stones in gallbladder and common bile duct, suspected mass at caput pancreas, chronic cholecystitis. It was suggested to check CA 19-9 level and perform ERCP. It turned out that CA 19-9 level was normal and from abdominal CT scan showed multiple stones in gallbladder and intrahepatic biliary duct, suspected mass at caput pancreas with calcification and hepatomegaly with distension of bilateral intrahepatic duct. The ERCP was done and concluded distension of biliary duct intra and extrahepatic with multiple stones in CBD, gallbladder and hepatic duct. They also placed stent for biliary drainage. It was suggested to consult digestive surgery department. The serum bilirubin level decreased and so

did the BUN and serum creatinin level. Thus, we consulted to digestive surgery department for further treatment. They performed cholecystectomy and exploration of CBD per laparascopic which had found multiple black pigment stones and the place T-tube at CBD. Cholangiography was done after that and it showed multiple stones still exist in intrahepatic biliary tract. So, they conducted laparatomy exploration of CBD and choledochojejunostomy to release stones. The patient went home in good condition after about two months of hospitalization.

DISCUSSION

This is a case demonstration of diagnostic approach and treatment of choledocholithiasis. Although several diagnostic modalities had been performed, it was still difficult to making the diagnosis accurately and selected the appropriate treatment. The first diagnosis made for this patient is obstructive jaundice due to stone with differential diagnosis malignancy. The most common cause is stone in the common duct, other causes are carcinoma of the pancreas and ampulla, benign bile duct stricture and cholangiocarcinoma.[2] In our hospital the most common cause of obstructive jaundice is periampullary tumors.[12]

From the anamnesis the patient suffered from epigastric pain which is recurrent and associated with nausea and vomiting. He also suffered from fever, jaundice and right upper abdominal quadrant pain. We had diagnosed him acute cholangitis because those symptoms were fulfilled the classic Charcot triad.[1,2,3] During physical examination we found jaundice and Murphy sign was positive. Laboratory finding revealed increases of bilirubin level whereas the direct bilirubin is higher than indirect one. There were also raised of alkaline phosphatase and g-glutamyl-transpeptidase. The rises are due to increased synthesis or release of enzymes from liver and biliary plasma membranes.[1,3]

According to diagnostic algorithm; after anamnesis, physical examination and laboratory examination, we performed abdominal USG which allows the distinction between cholestasis with dilated bile ducts and cholestasis without duct dilatation. If USG shows dilated ducts, cholangiography is necessary to be performed.[1,2,8]

The first abdominal USG showed choledocholithiasis but the second examination suggests that there was malignancy at the liver and there were no stones. This could happen because abdominal USG are accurate in diagnosis of gallbladder stones, but CBD stones are missed frequently (sensitivity 15% to 40%).[4,7] The detection of CBD stones is impeded by the presence of gas in the duodenum, possible reflection and refraction of the sound beam by curvature of the duct and the location of the duct beyond the optimal focal point of the transducer.[3] The third abdominal USG showed that there were multiple stones in gallbladder and CBD, also suspected malignancy of caput pancreas. That is why we performed CT-scan which also showed multiple stones in gallbladder and intrahepatic biliary duct, suspected mass at caput pancreas and hepatomegaly with dilatation of intrahepatic duct. However, the CA 19-9 level result was normal.

Patient underwent ERCP which showed dilatation of biliary duct intra and extra hepatic with multiple stones in CBD, hepatic duct and gallbladder. Stent was placed for biliary drainage. There was no mass found on ERCP and because it has been the gold standard for making diagnosis of choledocholithiasis. Thus, differential diagnosis of malignancy could be ruled out in this patient.[1] The stent was placed for biliary drainage to decrease bilirubin level before definitive therapy was performed.

After the stent was inserted, we consult to digestive surgery department to conduct further treatment. They had conducted cholecystectomy by laparascopic and exploration of CBD. Furthermore, they performed cholangiography to evaluate whether stones still exist or not. From this examination, it was found that multiple stones still exist in intrahepatic biliary tract. They determined to perform laparatomy to re-explore the CBD and they found multiple stones which have black colour and 0.5 cm in diameters. Due to dilatation of distal CBD, they finally decided to performed choledochojejunostomy.

The patient also diagnosed acute renal failure when he first came to emergency room because the elevation of BUN and serum creatinine. However, after optimal balance of fluid therapy within several days of hospitalization, the BUN and serum creatinine level were decreased. The renal failure was thought due to prerenal cause like dehydration and exposure to nephrotoxic drugs such as aminoglycoside antibiotic. The cause of hyponatremia in this patient was non renal solute loss due to vomiting.

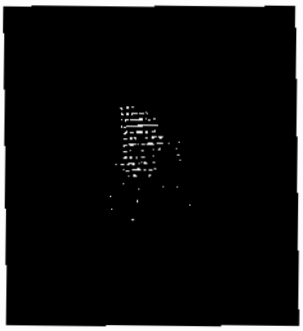

Figure 1. Normal chest X-Ray

Figure 2. Pneumonia differential diagnostic tuberculosis

Figure 3. Multiple stones in CBD, ductus hepaticus and fesica felea

REFERENCES

1. Schiff ER, Sorrell MF, Maddrey WC. Schiff's diseases of the liver. 9th ed. Lippincott Williams & Wilkins 2004.p.651-66
2. Sherlock S, Dooley J. Diseases of the Liver and Biliary System. 11th ed. Oxford: Blackwell Science Ltd 2002.p.597-623
3. Lee SP, Kuver R. Gallstones. In: Yamada T, Alpers D, Powell D, Owyang C, Silverstein F, editors. Gastroenterology, 2nd ed. Vol 2. Philadelphia: JB Lippincott Co, 1992.p.2187-2206
4. Dandan IS. Choledocholithiasis.Emedicine.com.2002
5. Flores C, Maguilnik I, Hadlich E, Goldani LZ. Microbiology of choledochal bile in patients with choledocholithiasis admitted to a tertiary hospital. J Gastroenterol Hepatol 2003;18:333-46
6. Rani AA.Kolangio-Pankreatografi Retrograd Endoskopik. In: Endoskopi dalam bidang Gastroentero-hepatologi.p.169-77
7. Lesmana LA. Obstruksi traktus biliaris. In: Sulaiman A, Daldiyono, Akbar N, Rani AA, editors. Gastroenterologi Hepatologi. CV Infomedika Jakarta 1990.p.104-8
8. Duensing RA, Williams RA, Collins JC, Wilson SE. Managing choledocholithiasis in the laparascopic era. Am J Surg Dec1995;170:619-22
9. Misra SP, Dwivedi M. Endoscopic management of choledocholithiasis: To cut, stretch or relaxes. J Gastroenterol Hepatol 1998;13:1180-82
10. Sugiyama M, Atomi Y. Treatment of acute cholangitis due to choledholithiasis in elderly and younger patients. Arch Surg. Vol 132. Oct 1997.p.1129-31
11. Bowrey DJ, Fligelstone LJ, Solomon A, Thomas G, Shandall AA. Common bile duct stenting for choledocholithiasis: A district general hospital experience. Postgrad Med J 1998;74:358-60
12. Lalisang T. Penanganan Bedah Ikterus Obstruksi. Dalam: Makalah temu ilmiah bedah digestif; 4-5 Mei 2001, Jakarta. Subbag Bedah Digestif FKUI-RSCM 2001

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