Case Report:
MANAGEMENT OF PEPTIC ULCER INFECTION DUE TO Helicobacter pylori INFECTION AND ABSCESS LIVER BOWEL PERFORATION

Tri Asih Imro’ati1, Ummi Maimunah2
1Resident at the Department of Internal Medicine, Faculty of Medicine, Airlangga University, Surabaya
2Division of Gastroentero-Hepatology, Department of Department of Internal Medicine, Faculty of Medicine, Airlangga University, Surabaya

ABSTRAK

Keywords: Ulkus Peptikum, Infeksi Helicobacter pylori, Perforasi Usus, Abses Liver

ABSTRACTS
It has been reported a male patient 69 years old, with body heat complaints, upper abdominal pain, and bowel black, with a history of drinking aspilets for 9 years. From the results obtained hypertension, anemia, cardiomegaly, epigastric tenderness and right upper quadrant, hepatomegaly, leukocytosis, hipoalbumin, abdominal ultrasound picture of multiple liver abscesses right lobe, old myocardial infarction inferio ECG, CT-scan of the abdomen obtained multiple liver abscesses in the right lobe, wall to wall abscess partially attached gallbladder and duodenum, perihpatic fluid collection right pleural effusion and left. The initial diagnosis was multiple liver abscesses, melena, erosive gastritis, anemia due to bleeding, hypertension stage I (JNC VII), hipoalbumin. Patients undergoing endoscopic surgery laparoscopy to drain abscesses installation. Operating converted to laparotomy due to perforation was found in the duodenum, transverse colon, and gallbladder. Histopathological examination of chronic superficial gastritis obtained, duodenal ulcer pepticum acute stage; H. pylori positive. Pas culture results obtained Escherechia coli susp. ESBL. Patients received therapy for eradication of H. pylori (amoxicillin, clarithromycin, PPI for 14 days, metronidazole and meropenem in accordance culture. Concluded the most likely cause of liver abscess is an infection of E. coli yang translokasi melalui perforasi ulkus peptik. The patient subsequently underwent outpatient controls obtained in poly UBT and HPSA is still positive. (FMI 2013;49:252-258)

Keywords: Peptic Ulcer, Helicobacter pylori Infection, Bowel Perforation, Liver Abscess

Correspondence: Tri Asih Imro’ati, Resident at the Department of Internal Medicine, Faculty of Medicine, Airlangga University, Surabaya

INTRODUCTION
Infection with Helicobacter pylori (H. pylori) is a cofactor associated with the development of the three upper gastrointestinal diseases, namely: gastric or duodenal ulcer (1-10% of infected patients), gastric cancer (0.1 to 3%), and gastric mucosa-associated lymphoid-tissue (MALT) lymphoma (<0.01%). The risk of the occurrence of the disease in infected patients varies in the general population. The majority of patients with H. pylori infection do not experience clinically significant complications (McColl 2010), (Chey & Wong 2007). Perforation occurs in 5-10% of patients with duodenal ulcer. More than 95% of duodenal ulcers occur in the first part of the duodenum. In about 50% of patients with duodenal ulcer,
perforation occurs without a history of previous dyspepsia. Mortality of perforated duodenal ulcer decreased from 40% to < 10% for the diagnosis and early treatment. The operation should be carried out after the patient teresusitasi. Of the pathogenesis of duodenal ulcer, it is clear that H. pylori plays > 90% in these patients. Closure of perforation followed by H. pylori eradication therapy is the treatment of simple and safe option (Khan et al 2005).

Liver abscess is a space-occupying lesions in the liver that is infectious which is the most common cause of pyogenic and amubik. Pyogenic liver abscess is usually rare but potential cause of death with 20 incidents of 100,000 patients hospitalized in a population of western countries. Severity depending on the source of infection and the condition of the patient base. Amubik liver abscess is often endemic in tropical countries Entamoeba histolytica and more on people (especially young males) with impaired cell mediated immunity. The principle of treatment is drainage of pus, appropriate antibiotics, and address the source of infection (Dutta & Bandyopadhyay 2012). Here is the report a patient with peptic ulcer (due to H. pylori infection) which is perforated duodenum, transverse colon, and gallbladder, and liver abscess complications

CASE REPORT

A male patient, Tn. M, 69 years old, a retired high school teacher from Ngawi, came to a private hospital on March 24, 2013 with complaints of body heat from the 10 days before hospital admission (SMRs). Hot body patients improved after taking paracetamol, then up again. The patient also complained of upper abdominal pain along with his body heat. Decreased appetite since ill. The patient felt weakness, and easy to forget. Sometimes swollen feet that disappears after waking diarrhea every 3 days and the colour of the fesses is black. BAK as usual around 500-700 cc per day. The patient was in hospital MRS Ngawi, received a blood transfusion and albumin, and underwent abdominal ultrasound examination. From the ultrasound found a lump in the liver, then the patient is advised to undergo in the CT-scan of the abdomen. By the family, the patient was taken to Surabaya to undergo the operation. Of the abdomen obtained sociable, uniform distribution, and unusual found young cells. 1015 SG obtained from urinalysis, blood -, pH 6.0, leucocytes +, +1 protein, bacteria -, sediment: 1-2/lp erythrocytes, leukocytes 1-2/lp, epithelial +. Abdominal ultrasound examination date from March 23, 2013 obtained an enlarged liver size, mass appeared in the right lobe of the liver size 12x9 cm, normal parenchyma, normal ekogenisitas, DD hepatoma suspected abscess picture. Of ECGs obtained 90x/menit sinus rhythm, LAD, old inferior myocardial infarction.

Obtained from physical examination GCS 456, BMI 22 kg/m2 (good nutrition), blood pressure 153/94 mm Hg, pulse 93x/menit, respiratory 18x/menit, axillary temperature of 36.3 °C. Obtained from the head neck conjunctival pallor. From the obtained symmetrical thoracic, cardiac iktus 2 cm lateral to the mid line of the left clavicle, single S1 S2, no murmur or gallop obtained, resonant breath sounds, crackles and wheezing are not obtained. Of the abdomen obtained sociable, positive bowl sounds normal, epigastric tenderness and right upper quadrant, a palpable liver 2 fingers below the costal arch, flat and sharp edges, spleen not palpable. Obtained from acral extremities warm dry pale.

Obtained from laboratory tests Hb 7.7 g/dL, Hct 24.0%, MCV 82.5 fL, MCH 26.5 pg, MCHC 32.1 g/dL, leukocytes 11.840/uL, 12.1% lymphocytes, granulocytes 82.3% neutrophils, platelets 329,000/uL, BUN 9.17 mg/dL, serum creatinine 0.7 mg/dL, uric acid 4.9 mg/dL, random blood sugar 105 mg/dL, AST 23 U/L, alanine aminotransferase 19 U/L, direct bilirubin 0.34 mg/dL, total bilirubin 0.53 mg/dL, albumin 2.28 g/dL, globulin 4.3 g/dL, Na 135.5 mmol/l, K 3.44 mmol/l, Cl 89.7 mmol/l, AFP 4.22 ng/mL, HBsAg negative, negative antiHCV. Obtained from the peripheral blood smear normokrom normocytic erythrocytes, leukocytes of normal, decreased platelets, uniform distribution, and unusual found young cells. 1015 SG obtained from urinalysis, blood -, pH 6.0, leucocytes +, +1 protein, bacteria -, sediment: 1-2/lp erythrocytes, leukocytes 1-2/lp, epithelial +. Abdominal ultrasound examination date from March 23, 2013 obtained an enlarged liver size, mass appeared in the right lobe of the liver size 12x9 cm, normal parenchyma, normal ekogenisitas, DD hepatoma suspected abscess picture. Of ECGs obtained 90x/menit sinus rhythm, LAD, old inferior myocardial infarction.

The current working diagnosis is suspected hepatoma liver abscess DD, melena due to alleged erosiva gastritis, anemia due to bleeding, hypertension stage I (JNC VII), hipoalbumin and hypokalemia due to less intake. Diet therapy is given H2 2100 kcal/day, infusion PZ: tutofusin OPS = 1:1, drip pantoprazole 40 mg in the PZ and tutofusin, metronidazole 3x500 mg drip, Ceftriaxon 2x1 g iv injection, metoclopramide injection 5/12 ampoules iv, transfusion PRC 1 bag/day, amlodipine 5 mg-0-0, 20% albumin transfusion of 100 cc up to albumin ≥3 g/dL, KSR 1x1 tablet. Diagnostic plan: rontgent photo thoracic, cardiology consul, abdominal ultrasound test.
Dated March 25, 2013, a complaint of patients still remain. TD 130/90 mmHg, pulse 90x/minute, axillary temperature of 37.7 °C, respiratory 20x/minute. Results of thoracic images contained cardiomegaly. The results obtained Abdominal ultrasound: enlarged liver size, intensity ekoparenkim normal, flat surface with sharp edges, portal vein and hepatic vein normal, visible picture of mass with multiple internal echo inside the right lobe with a size of 10.5 x 12.1 cm, no visible intratumoral vascular. biliary tract intrahepatal not widen, negative ascites: gallbladder difficult to evaluate; obtained a small cyst in the upper pole of the right kidney size 1.09 x 1.04 cm; conclusion: multiple abscess of right lobe of the liver, portal vein is still good, right renal cyst, organ another invisible disorders. Diagnosis: multiple liver abscesses. Consult digestive surgery is recommended for a CT scan of the abdomen. Therapy remains.

Dated March 26, 2013, the patient complained of fever, abdominal pain and bowel movements like black paste. TD 110/70 mm Hg, pulse 90x/minute, axillary temperature of 37.5 °C, respiratory 20x/minute. Working diagnosis: multiple liver abscesses, melena ec. erosive gastritis, anemia due to bleeding, hypertension stage I (JNC VII), hipoalbumin. Therapy: infusion tutufusin OPS: Kalbamin 1:1, antibiotics remain, lansoprazole pump 30 mg in 8 cc PZ/8 hours (3 times daily), vitamin K injection ampoules 3x1 iv, injection of 3x500 mg iv tranexamic acid, paracetamol when hot. Obtained from cardiology old myocardial infarct inferior and 2x5 mg ISDN therapy, trimetazidine hydrochloride 35 mg to 0-35 mg. 2.5 mg bisoprolol-0-0, echocardiography plan.

Dated March 27, 2013, the patient was still right abdominal pain and melena. Patients undergoing abdominal CT scans without and with contrast 3-phase, showed: multiple fluid collection in the right lobe with size X77 75.3 mm, 52.9 x50, 116.9 mm x139 with blood content in some lesions: first contrast shows slight rim contrast enhancement: lesion appears partially attached to the wall of the gallbladder and the duodenum: are perihepatic fluid collection and pleural effusion left right: hypervascularization and there is no staining of tumor picture. Conclusion: multiple liver abscesses in the right lobe, some with blood content, abscess wall partially attached to the wall of the gallbladder and the duodenum, perihepatic fluid collection right pleural effusion and left. Continued therapy plus somatostatin 1 ampoule in 12 cc PZ within 12 hours (2x/day), gastroscopy plan.

Dated March 28, 2013, the patient had no fever, melena and the abdominal pain is still felt. In the field of digestive surgery laparoscopy rencara pro plug drain the abscess. Plan check DL and preoperative albumin. On March 29, the results of echocardiography: PML prolapse, severe MR + medium, LV EF 63.6%, normokinetic. In the field of cardiology do not mind to do surgery, therapy: amlodipine 5 mg-0-0, ISDN 2x5 mg, trimetazidine hydrochloride 35 mg to 0-35 mg, 2.5 mg bisoprolol-00.

Dated March 30, 2013, the patient underwent laparoscopic surgery with general anesthesia. Results of laboratory tests: Hb 9.6 g/dL, Hct 32.3%, leucocytes 10.910/uL, lymphocytes 10.7%, 82.5% neutrophil granulocytes, platelets 323.000/uL, albumin 2.7 g/dL. From the reports mentioned surgery procedures: insertion smoothly, acquired liver abscess, drainage is done: obtained attachment duodenum, liver, transverse colon, gallbladder: conducted dilation, obtained duodenal perforation in part 1 and then turned into laparotomy surgery procedures, obtained duodenal perforation in part 1 with a diameter of 3 cm, the transverse colon perforation with a diameter of 2 cm, perforation of the gallbladder, duodenum and then do repair transverse colon, cholecystoplasty, subhepatal drain fitting, and sewing the wound. Post- surgery patients moved into the room, and examination of pus cultures taken histoPA gastric and duodenal biopsy tissue. BGA inspection results obtained pH 7.45, pCO2 43, pO2 328, BE 5.8, HCO3 30.8, satO2 100%, blood sugar 213 mg/dL, Na 138 mmol/L, K 2.2 mmol/L, lactate 2.8 mmol/L. Therapy: the patient fasting, infusion Clinimix E20: Aminofuscin OPS, KCL 50 mEq/day in RL, injection meropenem 3x1 g, metronidazole 3x500 mg iv, 2x40 mg pantoprazole iv, vitamin C ampoule iv 2x1, 2x4 ondacentron mg iv, paracetamol 3x1 g iv, 3x30 mg iv ketorolac, morphine 1 mg/hour pump.

Dated March 31, 2013, a patient in a weakened condition, hematoschezia ± 500 cc. TD 100/80 mmHg, pulse 142x/minute, respiration 24x/m, axillary temperature of 36.5 °C. From the laboratory results obtained Hb 12.6 g/dL, Hct 37.8%, leucocytes 19.230/uL, lymphocytes 4.2%, 93.3% neutrophil granulocytes, platelets 294.000/uL, lactate 2.3 mmol/L, temperature of 36.5 °C. From the laboratory results obtained Hb 12.6 g/dL, Hct 37.8%, leucocytes 19.230/uL, lymphocytes 4.2%, 93.3% neutrophil granulocytes, platelets 294.000/uL, albumin 2.86 g/dL. BGA inspection results obtained pH 7.5, pCO2 43, pO2 328, BE 5.8, HCO3 30.8, satO2 100%, blood sugar 213 mg/dL, Na 138 mmol/L, K 2.3 mmol/L, Ca 0.42 mmol/L, lactate 1.7 mmol/L. The diagnosis: post-laparotomy day I ec. Duodenal perforation + liver abscess drainage, sepis, hypokalemia, hypocalcemia, hyperglycemia reactive. Therapy: ICU patients moving, loading RL 500 cc, CVC pairs, injection of 3x500 mg iv tranexamic acid, Ca gluconate injection of 1 ampoule iv, others remain. Plan check DL, GDA series, and electrolytes.
The patient was transferred to the room.

Dated 14 April 2013, there was no melena and hematochezia, Hb 11.4 g/dL, Hct 34.6%, 9360/uL leukocytes, lymphocytes 13.5%, 75.9% neutrophil granulocytes, platelets 414,000/uL, BUN 14, 09 mg/dL, SK 0.45 mg/dL, BT 2.5 minutes (1-5), CT 10 (8-18), PPT 14.4 seconds (13.5), APTT 39.9 seconds (36.2). The patient was transferred to the room.

Dated 20 April 2013, the patient complained of body heat, drain the abscess has been removed. Hb 10.6 g/dL, Hct 31.9%, 5170/uL leukocytes, lymphocytes 19.9%, monocytes 11%, 68.7% neutrophil granulocytes, platelets 234,000/uL, urinalysis: BJ 1.025, pH 6, proteinuria +1, erythrocyte 25/μL (+2), leukocyte 100/μL (+2), 2-5/hpf erythrocytes, leukocytes 30-40/hpf, 4-6/hpf epithelial, leukocyte cylinder +, + bacteria, fungi +, + granular casts. The diagnosis: post-laparotomy day XI ec+s perforated peptic ulcer. urinary tract infections. Plan check urine culture and antibiotic sensitivity. Therapy: 2100 kcal diet soft TKTP. 1x500 mg levofloxacin orally, 3x500 mg paracetamol when hot.

Dated 28 April 2013, no complaints, was able to mobilize, comatos is, Hb 10.1 g/dL, leukocytes 8710/uL, 68.8% neutrophil granulocytes, platelets 398,000/uL, albumin 3.2 g/dL, the results urine culture: E. coli > 10e5 cfu, sensitive to the antibiotic amoxicillin, cotrimoxazol, ciprofloxacin, levofloxacin, cefotaxim, ampicillin sulfactam, and meropenem. Therapy: outpatient, poly control 3 days. Plan check H. pylori stool antigen and urea breath test.

Dated May 24, 2013, the patient controls. HPSA and UBT results obtained are still positive. Patients undergoing planned gastroduodenoskopi.

**DISCUSSION**

*Helicobacter pylori*, a common pathogen in humans, is a microaerophilic gram-negative bacterium that chronically infects the gastric epithelial cell surface and settled on the mucin layer (Vogiatzi et al 2007). *H. pylori* is a chronic infectious disease that exist around the world that play a role in the onset of chronic gastritis, peptic ulcer disease, and gastric malignancy. From the latest international research note that the prevalence varies from 7%-87%, the lowest in North America and Western Europe (Wang & Peura 2011).

Indication of diagnosis and therapy of *H. pylori* is: active peptic ulcer disease (gastric or duodenal ulcer), a history of peptic ulcer disease diagnosis upright but untreated, low-grade MALT lymphoma, gastric cancer after endoscopic resection of early-stage, and dyspepsia were not found to cause (depending on the prevalence of *H. pylori*). Indication of diagnosis and therapy are still controversial include: non-ulcer dyspepsia, gastrointestinal reflux disease, people who use drugs non-steroidal anti-inflammatory (NSAID), iron deficiency anemia is not clear why, and populations at high risk of gastric cancer. Diagnosis of infection *H. pylori* is divided into requiring and not requiring endoscopy. Which require endoscopy include: histology (gold standard), rapid urease test, culture, and PCR: being that does not require endoscopy include: antibody test (quantitative and qualitative), urea breath test and fecal antigen test (Chua et al 2011, Wang & Peura 2010, McColl 2010, Chey & Wong 2007, Gatta et al 2005).
Primary therapy of infection of *H. pylori* include PPI, clarithromycin, and amoxicillin/metronidazole (clarithromycin-based triple therapy) for 14 days or PPI/H2RA, bismuth, metronidazole and tetracycline (bismuth quadruple therapy) for 10-14 days. Another recommended therapies can be seen more clearly in Table 1 below. The most important predictor for failure of anti-*H. pylori* therapy include poor compliance and antibiotic resistance (Chuah et al 2011, Wang & Peura 2011, McColl 2010, Chey & Wong 2007, Gatta et al 2005).

This patient had a history of drinking aspilet for 9 years, often melena, epigastric pain, suffered a perforated duodenum at the first part, the transverse colon, and gallbladder. Disease chronic superficial gastritis, duodenal ulcer pepticum acute stage, *H. pylori* positive note of the results of histopathological tissue biopsies during surgery. Patients were given standard triple therapy for the eradication of *H. pylori* (PPI, clarithromycin, amoxicillin) for 14 days.

Urea breath test, histology, culture or rapid urease test (rapid urea test) were positive at any time after the therapy showed treatment failure. It is recommended to do a post- treatment after 4 weeks after the end of therapy, based on the fact that the bacteria are left behind need time to improve and occupy again in the stomach in sufficient quantities to be detected. Up to 4 weeks negative test accuracy of around 98-100%. Urea breath test is the most preferred tests to monitor the post-eradication, except when no can use a stool test. The available data indicate that the fecal antigen test (*H. pylori* stool antigen) that uses a monoclonal antibody anti-*H.pylori* more reliable than the polyclonal stool antigen, thus recommended stool antigen test using monoclonal antibodies. Culture, histology, UBT, RUT and stool antigen tests depends on the density of bacteria and test the adequacy of false negatives can occur if the bacterial load down due to the use of antimicrobials (antibiotics or bismuth) or the use of PPIs (Attumi & Graham 2011, Malfertheiner et al 2007).

In these patients the UBT and the planned examination of the current HPSA control, after 4 weeks of the end of the standard triple therapy, and counseling was given for not taking a PPI at first, and the result of UBT and HPSA was still positive. Patients then planned gastroduodenoskopi for evaluation. *H. pylori* associated duodenal ulcers by 95% and 70% of gastric ulcers (Napolitano 2009). Peptic ulcer is a mucosal lesion of the stomach or duodenum where acid and pepsin play an important role in its pathogenesis. This occurs because of an imbalance of aggressive activity of acid and pepsin with a defense mechanism that prevents the destruction of the mucosa (Khan et al 2005). Risk factors that play a role in this disease include smoking, alcohol, stress and NSAIDs, including aspirin. Refractory peptic ulcer disease can cause complications of bleeding and gastrointestinal complications. Perforation, stricture and obstruction of a gastrointestinal complication of duodenal ulcer. Perforation occurs in 5-10% of patients with peptic ulcer disease, in which more than 95% occur in the first part of the duodenum, 60% in the anterior wall of the duodenum, 20% in antral, 20% of the lesser curvature of the gastric ulcer and 5-10% occur in posterior wall and can cause pancreatitis or abscesses (Napolitano 2009, Hsu et al 2008, Khan et al 2005).

Taylor and Visick get some perforated peptic ulcer that can spontaneously covered by omentum, although there is free air in the peritoneal cavity. Rossoff concluded from his experience in Los Angeles, of the 377 cases, 43% closed itself (Nusree 2005). We need to know that the omentum has a function during episodes of peritonitis.

Table 1. Recommended treatment regimens for *H. pylori* (Chuah et al., 2011)

<table>
<thead>
<tr>
<th>Second-line therapy</th>
<th>Third-line therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bismuth-containing quadruple therapy</td>
<td>Quadrupe therapy for 10 days consisted of a PPI (standard dose 2x/day.), Bismuth (4x/hari standard dose), and 2 antibiotics according to antibiotic sensitivity testing</td>
</tr>
<tr>
<td>Levofoxacin-based triple therapy</td>
<td>PPI (standard dose 2x/day.), Bismuth (4x/hari standard dose), levofloxacin (1x500 mg) and amoxicillin (2x1 g) for 10 days</td>
</tr>
<tr>
<td></td>
<td>PPI (standard dose 2x/day.), Bismuth (4x/hari standard dose), levofloxacin (1x500 mg) and amoxicillin (2x500 mg) for 10 days</td>
</tr>
<tr>
<td></td>
<td>PPI (standard dose 2x/day.), Rifabutin (2x150 mg) and amoxicillin (2x1 g) for 14 days</td>
</tr>
<tr>
<td></td>
<td>PPI (standard dose 2x/day.), Tripotassium dicitratobismuthate (2x240 mg), furazolidone (2x200 mg), and tetracycline (2x1 g)</td>
</tr>
<tr>
<td></td>
<td>PPI (standard dose 2x/day.), Levofoxacin (1x500 mg) and amoxicillin (2x1 g) for 10 days</td>
</tr>
<tr>
<td></td>
<td>PPI (standard dose 2x/day.), Bismuth (4x/hari standard dose), and 2 antibiotics according to antibiotic sensitivity testing</td>
</tr>
<tr>
<td></td>
<td>PPI (standard dose 2x/day.), Bismuth (4x/hari standard dose), levofloxacin (1x500 mg) and amoxicillin (2x1 g) for 10 days</td>
</tr>
<tr>
<td></td>
<td>PPI (standard dose 2x/day.), Rifabutin (2x150 mg) and amoxicillin (2x1 g) for 14 days</td>
</tr>
<tr>
<td></td>
<td>PPI (standard dose 2x/day.), Tripotassium dicitratobismuthate (2x240 mg), furazolidone (2x200 mg), and tetracycline (2x1 g)</td>
</tr>
</tbody>
</table>

- **Table 1.** Recommended treatment regimens for *H. pylori* (Chuah et al., 2011)
First, rapid absorption and cleaning bacteria and foreign matter from the peritoneal cavity. Second, insert leukocytes into the peritoneal cavity. Third, attach and seal off the area, causing contamination of attachment (Platell et al 2000). In some patients with perforated ulcers, both duodenal damaged proximal and penetration into surrounding organs, a large perforation with a diameter > 20 mm or with duodenal stenosis, surgery requiring resection. Perforation of the peritoneal cavity associated with peritonitis, and requires emergency surgical intervention. Conventional laparotomy and laparoscopy technique for closure with omental patch holes is a therapeutic option for these patients (Napolitano 2009).

These patients have peptic ulcer disease is very risky as taking aspirin for 9 years. Then complications arise in the form of upper gastrointestinal bleeding and perforation of the bowel melaena. At first, these patients do laparoscopic surgery for liver abscess drain installation. Current procedures surgery, adhesions obtained duodenum, liver, transverse colon, and gallbladder. Once widened, obtained duodenal perforation in part 1 so that the operation is converted to laparotomy. Looks duodenal perforation with a diameter of 3 cm, the transverse colon perforation with a diameter of 2 cm, and perforation of the gallbladder. Do repairs duodenum and the transverse colon, cholecystoplasty, subhepatal drain fitting, and sewing the wound. From here can be seen that possibility in this patient perjadi perforated duodenal ulcer closure then occurs spontaneously by omental adhesions that occur-attachment. Germs from causing infection in the gut and then around the organs and liver abscesses.

The etiology of pyogenic liver abscess may originate from biliary (lithiasis, cholecystitis, biliary enteric anastomosis, endoscopic biliary procedures, percutaneous biliary procedures, malignancy), portal (diverticulitis, anorectal suppuration, pelvic suppuration, postoperative sepsis, intestinal perforation, appendicitis, inflammatory bowel disease, colon and gastric cancer), arterial (endocarditis, sepsis, ENT infections, dental infections), trauma (open or closed abdominal trauma, kemoemboliasis, or radiofrequency percutaneous ethanol injection), or cryptogenic (cause unknown). Between 15-50% of patients in different studies, did not find the cause or source of the infection. Clinical symptoms early in the disease are not specific, covering malaise, anorexia, weight loss, headache, myalgia and arthralgia in most of the cases. The prodromal symptoms may appear for several weeks before the appearance of specific symptoms, such as fever, chills and abdominal pain, although pain is not always felt in the right upper quadrant. Most of the liver abscess located in the right lobe. Abscesses are touching the diaphragm can cause pleuritic pain, cough and shortness if the clinical presentation in conjunction with non-specific symptoms mentioned above can complicate the diagnosis. Septic shock can occur in some patients, especially in the state of obstruction of the biliary tree. Although rare, some patients experienced peritonitis following rupture of the abscess into the peritoneal cavity. Diagnosis apart from the clinical, laboratory examinations (leukocytosis, anemia, hipoalbumin, increased transaminases and alkaline phosphatase), ultrasound of the abdomen CT scan, MRI, and culture. In western countries the most common result is a culture of \textit{E. coli}, the most common being in Asia is \textit{Klebsiella}. Principles of therapeutic liver abscess is a pus drainage, appropriate antibiotics, and address the source of infection (Dutta & Bandyopadhyay 2012).

The patient's body heat obtained from the 10-day SMRs, upper right abdominal pain, appetite drops, weakness, hepatomegaly, leukocytosis, anemia, hipoalbumin, increase in transaminases and alkaline phosphatase, abdominal sonogram obtained over multiple liver abscesses right lobe, from CT scans obtained multiple liver abscesses in the right lobe, some with blood content, abscess wall partially attached to the wall of the gallbladder and the duodenum, perihepatic fluid collection and pleural effusion left and right, and the results of pus culture grew \textit{E. coli}. Therapy given to these patients is drainage of pus, metronidazole and meropenem corresponding culture results. From the results of pus culture, it is possible cause of liver abscess is infiltration of germs (\textit{E.coli}) of perforated peptic ulcer.

In the intestines of healthy humans there are more microorganisms than other organs. The assortment of these organisms there is useful or profitable, and there are destructive or dangerous. Microorganisms that exist in the adult intestine is primarily non-spore anaerobic bacteria. among other \textit{Bacteroides spp.}, \textit{Bifidobacteria spp.}, \textit{Eubacterium spp.}, gram-positive cocci, \textit{Clostridium spp.}, \textit{Fusobacterium spp.}, \textit{E. coli}, \textit{Enterococcus spp.}, \textit{Streptococcus spp.}, \textit{Enterobacteriaceae spp}. \textit{E. coli}, including the microorganisms that could potentially harm (Wallace et al 2011). Liver abscess pus culture results in these patients is \textit{E. coli}, so we conclude that it is likely these are due to liver abscess intestinal bacteria translocation through the slit perforated peptic ulcer.

**CONCLUSION**

Histopathological examination of chronic superficial gastritis obtained, duodenal ulcer pepticum acute stage; \textit{H. pylori} positive. Pus culture results obtained
Escherichia coli susp. ESBL. Patients received therapy for eradication of H. pylori (amoxicillin, clarithromycin, PPI for 14 days, metronidazole and meropenem in accordance culture. Concluded the most likely cause of liver abscess is an infection of E. coli translocation through a perforated peptic ulcer. The patient subsequently underwent outpatient controls obtained in poly UBT and HPSA is still positive.

ACKNOWLEDGMENT

The authors are indebted to Tiara Rica for her assistance in making the layout of the article.

REFERENCES