LAPORAN KASUS

HELICOBACTER PYLORI INFECTION IN CHILDREN

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Abstrak

Faktor risiko infeksi *Helicobacter pylori* adalah tinggal di negara berkembang, kondisi sosial ekonomi yang rendah, jumlah anggota keluarga yang banyak, etnik dan genetik. Tatalaksana dan diagnosis *Helicobacter pylori* belum memuaskan karena adanya resistensi antibiotik pada pasien *Helicobacter pylori*. Kami melaporkan seorang pasien perempuan usia 8 tahun 6 bulan yang terinfeksi *Helicobacter pylori*. Diagnosis ditegakkan berdasarkan anamnesis, pemeriksaan fisik, dan hasil laboratorium. Pasien diduga terinfeksi *Helicobacter pylori* karena mengalami nyeri perut berulang. Dari laboratorium didapatkan serologi IgG *Helicobacter pylori* positif. Pada hasil endoskopi biopsi ditemukan kuman *Helicobacter pylori.* Pasien mendapat therapi eradikasi lini pertama untuk infeksi *Helicobacter pylori* yaitu amoksisilin, klaritromisin dan omeprazol selama dua minggu. Setelah dua minggu pengobatan keluhan pasien tidak ada.
Kata kunci: Helicobacter pylori, anak, nyeri perut berulang

Abstract

Risk factors for acquiring *Helicobacter pylori* infection include residency of developing country, poor socioeconomic conditions, crowded family, and possibly an ethnic or genetic as predispositions. The diagnosis and management *Helicobacter pylori* has not been satisfied yet, however, there is problem of increasing resistancy antibiotic due to *Helicobacter pylori*. Objective: We report a 8 year and 6 month old girl who suffered from Helicobacter pylori. The diagnosis was based on history, clinical finding, and laboratory work-up. Suspicion on the presence of *Helicobacter pylori* was started when the girl had recurrent abdominal pain. Serology IgG *Helicobacter pylori* was positive and we had done endoscopic examination and biopsy. Therapy this patient was first line eradication Helicobacter pylori which give amoxicillin, clarithromycin and omeprazole for two weeks. There are no sympthoms after two weeks therapy
Key word: Helicobacter pylori, children, recurrent abdominal pain

Introduction

The first isolation of *Helicobacter pylori* in 1982 by Marshall and Warren ushered new era in gastric microbiology.(1) The diagnosis and treatment of upper gastroduodenal disease have changed dramatically. Peptic ulcer approaches as an infectious disease nowadays, which needs elimination of etiology.(2)

Although spiral organisms had been observed in the gastric mucus layer many times in the preceeding century, the isolation of *Helicobacter pylori*, in conjunction with increased interest of pathogenesis gastroduodenal diseases, as well as the relatively frequent availability of clinical specimens via endoscopic biopsy, has led an important breakthrough of medical care.(1)

Risk factors for acquiring *Helicobacter pylori* infection include residency of developing country, poor socioeconomic conditions, crowded family, and possibly an ethnic or genetic as predispositions. Many patients still attribute symptoms of dyspepsia to an ulcer, and believe that ulcers are caused by diet, stress, and lifestyle factors; however, it is now clear that eradication of *Helicobacter pylori* is central management of the illness.(3) The diagnosis and management *Helicobacter pylori* has not been satisfied yet, however, there is problem of increasing resistancy antibiotic due to *Helicobacter pylori*.(3)

The aim of this presentation is to remind us about diagnosis and management of *Helicobacter pylori* in children.

Case Report

A 8 year 6 month old boy with main complaint of recurrent abdominal pain 3 days before admission, was admitted to Dr.M. Djamil Hospital on January 24rd 2009. She had been hospitalized in Sungai Dareh Hospital for 3 days and. She had been consulted surgery department and referred to Dr. M. Djamil Hospital Padang with diagnosis colic abdomen. In emergency room Dr. M. Djamil Hospital, there was no desease at surgery department and she was consulted to pediatric department.

She had recurrent abdominal pain 3 days before admission, exspecially in epigastrium and often woke up at night because of epigastrium pain. She was often recurrent abdominal pain since 3 months ago. Diarrhea 3 days before admission for 2 days 3-6 times/day with amount of ½ glass per-time,soft consistency, without blood, or mucus. She had vomit since 2 days before admission, 1 time a day with amount of 2 glass . The vomit was contained of food and drink, no sprayer. There were no fever, cough or breathlessness. She can drink, except oralit. The appetite was decreased, she ate noodle a once a day. The last miction was 1 hour ago, amount of normal, no change of colour, she was never history out of stone.

She got exclusive breast milk, the patient get breast milk until 2 years old. Streaming rice at 7 months, smooth rice at 9 months and solid meal since 12 months old. At the moment, the patient get family food 2-3 times/day amount ½-1 plate of rice with piece of meat and fish.

Phisical examination was normal. Her body weight was 23 kg (BW/A 85,2%, P10-25 CDC 2000), body height was 130 cm (BH/A 100%, P50 CDC 2000, BW/BH 85,2%), heigh age 8 years 6 months, with nutritional status was undernourished.

The Hemoglobin content was 14,2 gr %, white blood count 6600/mm^3, differential count

0/0/2/52/40/6. Urine and fecal examination were normal.

We diagnosis with Observation recurrent abdominal pain due to erosive gastritis. Differential diagnose Helicobacter pilory and duodenum ulcer. Nutritional status undernourished. The treatment was Ranitidine 2 x 25 mg. We planned to do IgG and IgM *Helicobacter pylori*, endoscopy and biopsy.

On the 3th hospitalization, the condition was good. The physical examination still the same. In laboratory finding IgG Helicobacter pilory: 1.11 (positive), normal < 1.00. The therapy this patient was changed. The therapy for this patient was amoxicillin 350 mgs three times daily, clarithromycin 200 mgs twice daily, omeprazole 20 mgs one time daily for two weeks. We planned for endoscopy after got agreement of the family.

On the 5th hospitalization, The result of endoscopy examination was esofagitis grade A (GERD), corpus ulceratum and moderate antrum gastritis. She was examined biopsy in corpus while endoscopy examination.

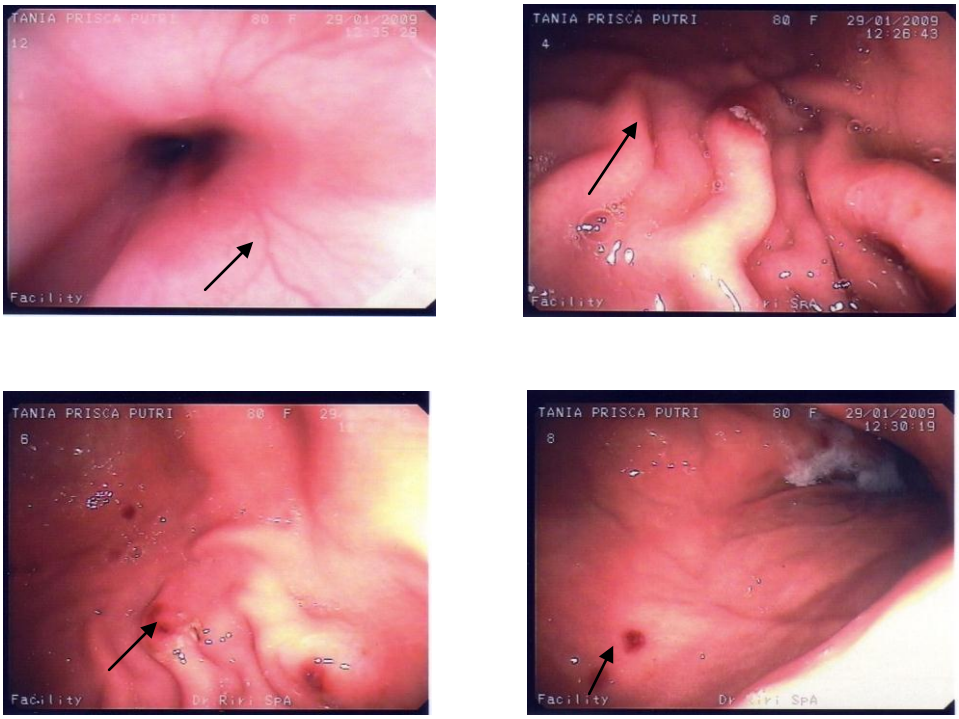

Figure 1. Endoscopic examination
Esofagus: hyperemic, erotion, no varises
Gaster: hyperemic mocous layer, erotion, ulceratum, hipersecretion liquid
Duodenum: hyperemic mocous, no ulceratum

On the 7th hospitalization, There is no simptom, the appetite was increased. The physical examination still the same. Follow up nutritional and antropometri status: Her body weight was 23,5 kg (BW/A 87%, P10-25 CDC 2000), body height was 130 cm (BH/A 100%, P50 CDC 2000, BW/BH 87%), with nutritional status was undernourished. The result of biopsi examination was chronic gastritis due to *Helicobacter pilory*.

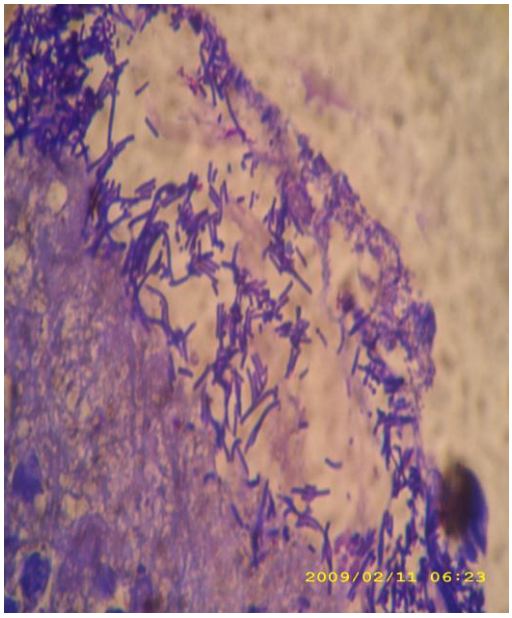

Figure 2. The result of biopsi examination:
Gastric mucus which half of ducts were hyperplastic, plenty of cell plasm, lymphocyted, eosinofilic.
Some place were found *H. pylori*

After two weeks therapy, the patient felt good and never got sympton like before.She came to control and the therapy was stopped.

Discussion

H. pylori infection in children always accompanied by gastritis more often than peptic ulcer. Gastritis in children usually give a sympton of stomatchache. Therefore recurrent abdominal pain in children assumed as clinical symptom that related to *H. pylori* infection.

Recurrent abdominal pain is common sign which brings patient to search medication and analogue with non ulcer dispepsia in adult. Gastritis usually shows abdominal pain complain in children. In some scientist, recurrent abdominal pain is related to *H. pylori* infection. About 30% recurrent abdominal pain in children found *H. pylori* in the antrum while 10% in the corpus. The ulcer in children seldom found but we should think about *H. pylori* infection as one possibility.(4)

Gastric disturbances consist of two. First, hypersecretion gastric acid (hyperchlorhidria) which can effect gastritis and sequele like duodenal ulcer and gastric ulcer. Second, hyposecretion gastric acid (hipochlorhidria) due to atrophy of gastric mucous. Vomites in this case usually caused by esofagus mucous iritation due to hipersecretion gastric acid.(4)

Acut or chronic diarrhea happened because of hiposecretion gastric acid. The effect of this case is gastric protection increase, so enteric infection can occur easily. The metaanalysis research showed diarrhea in of H. pylori patient caused by antibiotik, which intestine flora change qualitatively and quantitatively.(5)

Recurrent abdominal pain in children are analogued as non ulcer dyspepsia in adult. Moostrich concensus suggested that all patient with dyspepsia, tested as soon as possible with non invasif test such as serologic or urea breath test and if the

test was positive, immediately cured without endoscopic diagnostic.

In this case, patient diagnosed as recurrent abdominal pain caused by suspec *H. pylori* infection. Although urea breath test is the most sensitive of non invasif diagnostic test, in this case serologic examination is the choice of diagnostic due to less of facility and human resource for urea breath test.

The research of Gumel E in Turkey get in patient which recurrent abdominal pain, 60% seropositive for *H. pylori* IgG and 25% seropositive by IgA.[6] The definitive diagnose based on *H. pylori* examination of gastric biopsy from gastric mucous specimen of one or more gastric area. The histological examination showed severe gastritis including gastric atrophy and duodenal metaplasia. Research by Mahjoub F in Turkey mentioned that *H. pylori* in biopsi occurred 9,7% in male and 8% female. There is significant association between H. pylori with gastric activity (p=0,001).[7]

If serologic test was positive, eradication therapeutic should be given. This step was chosen by consideration of long time symptoms and disturbances normal life. Another cause such as urinary bladder stone, constipation, and psychist can be excluded, and conventional therapeutic with antacyd and H2 antagonist cannot help. Choosen therapeutic regimen is triple therapy with PPI that suggested by expert consensus in America and Europe. There is no repeat serologic test of the patient, because of no recurrent abdominal pain. If we get decreased of antibody titration more than 50%, means that eradication is successful.

Beside H. pylori culture was exactly diagnosis, culture can know profile sensitivitas bacteria dan antibiotik. Faber etc at the research in Israrel showed 42% resistant by omeprazole + amoxicillin with metronidazole and none of case resistant by omeprazole + amoxicillin with claritromycin.[8] Urea breath test was non invasive test have high sensitivitas and spesivitas.This test is important to know about H. pylori infection. Eradication rate, as defined by a negative 13C urea breath test 73,4% by omeprazole, amoxicillin with metronidazole and in 62,6% by omeprazole, amoxicillin with claritromycin.[8]

Many literature reported that eradication therapy just give real effect in *H. pylori* infection with peptic ulcer. Since this time, the patient never have abdominal pain and vomit symptom. For the future, a good hygiene and sanitation is still become attention to prevent eradication.[4]

The patient got eradication H. pylori first line theraphy eradicate totally *H.pylori*, *to* neutralize hypersecretion of gastric acid and change to normal condition. Resistancy antimicroba will make the theraphy fail.[9] Tripel therapy is combination of antisecretory agent and antimicroba agent for 7-14 days. From metaanalysis by Laheij etc in 1999 of 666 studies, therewere about 53.228 adult sample, eradication theraphy with PPI combine 2 of 3 antibiotics (claritromycin, amoxicillin, and metronidazole), has survival rate 79%-83%.[10] Metaanalysis research about eradication therapy H. pylori in children by khurana etc in 2007 made conclusion that using metronidazol and amoxicillin for 2-6 weeks, claritromycin 1-2 weeks, amoxicillin and proton pump inhibitor and macrolide for 2 weeks are the best

regiment in developing country.[11] Faber etc at the research in Israrel showed eradication was achieved in 73,4% by omeprazole + amoxicillin with metronidazole and in 62,6% by omeprazole + amoxicillin with claritromycin (p=0,078).[8]

Good nutrition is very important for growth and developmental of children, also attack diseases. Optimal intake dietary of is the patient necessary to protect severe problem of nutrition. Undernourished of this patient due to inadequate intake at home.

Prognosis depends on management of the disesase, early detection and adequate therapy are important to prevent complication, ulcus, gastrointestinal bleeding and survival better. If the diagnose was late, and theraphy was inadequate, ulcus, gastrointestinal bleeding, and cancer would occur, also relaps and resistancy of the drug are coming easily.[12]

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