ACUTE APPENDICITIS IN A CHILD WITH DENGUE HEMORRHAGIC FEVER

(A Case Report)

Harmon Mawardi
*Department Of Child Health, Medical Faculty Trisakti University

Abstract:

A 5-year old girl was sent by a Pediatrician on December 21, 1998 to a private hospital in east Jakarta at 2.00 p.m. with a main complaint of abdominal pain. She had abdominal pain for 2 days with no history of fever and bleeding. Based on physical examination, the diagnosis established at that time was acute appendicitis. Patient was consulted to a surgeon to get a second opinion. According to that surgeon, it was an acute appendicitis and suggested to perform appendectomy. Meanwhile, the laboratory findings did not show any signs of Dengue Hemorrhagic Fever (DHF) before the operation. Ten hours postoperation, the patient looked apathetic, lethargy, and had reduced blood pressure. Laboratory findings showed hemoconcentration and thrombocytopenia. The diagnosis was established as Dengue Shock Syndrome (DSS). The result of pathological anatomy examination confirmed acute appendicitis. The problem was addressed in this case study was overlapping of disease signs and symptoms between silent DHF and acute appendicitis. (J Kedokteran Trisakti 2000;19(2):54-9).

Key words: Appendicitis pediatric, dengue fever

Introduction

The Clinical spectrums of DHF vary from a silent dengue infection undifferentiated febrile, dengue fever (DF), DHF to severe DSS (12). Abdominal pain is an early clinical symptom of DHF similar to acute appendicitis in children (23). Patient or direct family members frequently do not convey a complete history of illness regarding DHF to the physician except a major complaint of abdominal pain. On the other hand, acute appendicitis may also reveal that similar symptom. Although laboratory examinations are performed, they do not always ensure evidence of an overlapping between DHF and acute appendicitis. Case of acute appendicitis accompanied with DHF is a high risk in surgery, as illustrated in this case.

The aim of this paper was to remind physicians to be cautious in establishing the diagnosis of appendicitis in children, because it is dangerous to carry out appendectomy if it overlaps with DHF.

Case of Description

A 5-year old girl was sent by a Pediatrician to a private hospital in east Jakarta on Sunday, December 1998 with a main complaint of abdominal pain. Based on anamnesis, she had abdominal pain for 2 days with no history of fever and bleeding. On physical examination, abdominal pain was detected by palpation, no signs of hemorrhage and fever. Although laboratory examinations are performed, they do not always ensure evidence of an overlapping between DHF and acute appendicitis. Case of acute appendicitis accompanied with DHF is a high risk in surgery, as illustrated in this case.

Laboratory findings showed hemoglobin content 12.7 g/dl, leucocytes 13.100/ul, hematocrit 37%, and thrombocytes 225.000/ul. Clotting and
bleeding time were within normal limits. Surgeon suggested to perform appendectomy. Pediatrician visited patient at 4.00 p.m. diagnosed with acute appendicitis. Soon after receiving parental informed consent, the operation commenced 9.00 p.m. Duration of operation was 1 hour without any complications except provision of 200 ml intra peritoneal fluid. The surgeon suspected that the patient had DHF. Postoperation, he contacted the Pediatrician and informed him that patient had suspected DHF. The surgeon visited the patient at 7.30 a.m. the next day, her condition was stable without any signs of bleeding, abdominal pain and distention.

Laboratory examination showed hemoglobin content 13 g/dl, hematocrit 42%, and thrombocytes 115.000/ul. Patient was recommended to drink water gradually. At 8.30 a.m. she looked apathetic, lethargic, cold extremities and reduced blood pressure to 60/40 mmHg. Further disease management was directed to Dengue Shock Syndrome (DSS).

Because her condition became worse, the attending physician decided to send the patient to ICU. Patient was set up on a ventilator and central venous pressure (CVP) route that made it easier to control fluid equilibrium. Laboratory examination was done every 6-8 hours. The following result showed hemoglobin content 11.7 g/dl, leukocytes 22.900/ul, hematocrit 35%. Thrombocytes 108.000/ul, differential count 0/0/2/65/28/5, BSR 10 mm/h. Blood chemical examinations revealed pH 7.32, PCO₂ 24.4 mmHg, PO₂ 108 mmHg, HCO₃⁻ 11.8 meq/l, TCO₂ 12.5, base excess 12.9 meq/l, and O₂ saturation 99.8%. Based on laboratory findings, the present diagnosis was acute renal failure (ARF) associated with metabolic acidosis. Patient had profound shock and multiple organ hypoxia.

To restore the renal function, dopamine was also given because of her inadequate blood pressure. Further laboratory findings showed thrombocytes 52.000/ul. Blood gas analysis was still in the state of metabolic acidosis and temperature increased to 40°C, and consciousness became worse accompanied with convulsions. At 7.00 p.m., abdominal bleeding of 300 ml occurred and patient was given blood transfusion. The next laboratory result showed thrombocytes of 45.000/ul. Vital signs did not improve significantly.

At 9.20 p.m. patient suffered from bradycardia and maximum resuscitation was performed but failed. Patient finally was deceased at 9.50 p.m. Pathological anatomy examination in the appendix tissue related to acute appendicitis.

**Discussion**

Hemorrhagic fever due to viruses are usually divided into 4 categories namely tickborne, mosquito-borne, zoonotic and African hemorrhagic fever. Dengue virus belonged to mosquito-borne. From the fourth categories of those diseases, there are four main viral hemorrhagic fever that have caused significant outbreaks of contagious disease.⁴

Indonesia ranks second after Thailand with increased of morbidity rate from 0.05% in 1968 to 8.14% in 1973. However, case fatality rate (CFR) of DHF decreased from 41.3% in 1968 to 3% in 1984.² The variations of season in Indonesia do not interfere with the incidence of DHF. However, from September to February DHF cases are prominent and achieve its peak level usually in January.¹

There are 4 types of dengue viruses namely Den-1, Den-2, Den-3 and Den-4. All are classified arboviruses and infections are through arthropods such as flies, mosquito and cockroaches. Arboviruses are divided into several groups. A. egypti is the most common vectors that infects humans. Group A consists of Chikungunya, Sindbis, Simliki, Venezuela equine encephalitis, Western equine encephalitis and some others. Group B consists of all types of dengue (1,2,3 and 4), Yellow fever, Wwsnila, Jananese encephalitis and others. The other groups are California and Bwamba.²

In Indonesia all type have been isolated successfully from the blood. Especially in Jakarta the majority of severe and fatal cases are dominated by D-
3 virus. Surveillance performed in Indonesia concluded that Den-2 and Den-3 viruses were alternately dominant. 

Many theories have developed to explain the pathogenesis of DHF, but pathological mechanism are still not fully know yet. According to the history of DHF, development in the last hundred years, only 2 theories have been dominant and trusted by most authors. These are theories based on virulence and immunopathology. First theory explains that if someone infected by a dengue virus, he will be ill when is virulence is strong enough to resist host immunity In fact, all viruses maybe potential to be a cause of death. The second theory, according to their investigationin humans and mice, concluded that after beinginfected with a certain viral serotype, immune response will be stable for certain time. However, it can not prevent the next viral infection. This theory is known then called as secondary infection theory of sequential heterolog virus. If somebody has a primaryinfection by another serotype, it is possible to activate complement producing mediators like (1) anaphylatoxin causing enhancement of capillary permeability, (2) aggregating thrombocyte causing thrombocytopenia and (3) damage of endothelium causing activation of coagulating factors.

Viral infection of dengue may cause a broad spectrum of clinical manifestations, including sequentia by silent dengue infection, mild undifferentiated febrile illness, dengue fever (DF), dengue hemorrhagic fever (DHF), and dengue shock syndrome (DSS). Variations of clinical features are shown similar to iceberg phenomena where top of the mountain are patients admitted to the hospital, meanwhile the mild cases as the base of mountain. Approximately for every case admitted in the hospital with DSS has actually 150-200 mild dengue cases has occurred in that population. Mild clinical manifestations will heal spontaneously without being treated (self limiting).

In fact, it is difficult to differentiate DF and DHF in field. However, WHO has established a guideline for diagnosis of DHF in 1986 based on clinical features and laboratory findings. WHO criteria consists of ; (1) sudden and continuous high fever for 2-7 days with unknown origin, (2) hemorrhagic manifestations varying from a mild (positive tourniquet, epistaxis, gums bleeding, hermatemesis and melena), (3) enlargement of liver, (4) shock signified by changing of pulse rate (high) frequency, low intensity and amplitude), clammy or wet extremities, cyanotic and restles. Laboratory findings showing; (1) thrombocytopenia (<100,000/ul) and (2) hemoconcentration (hematocrít>20% e.g. 35% to 42%).

On the 4-6th day of incubation period, patient enters febrile period for about 1-3 days followed by shock or plasma leakage on 3-7th day and ended by convalescence period on 7th day. Febrile period showed signs of facial flushing and headache, loss of appetite, vomiting, epigastric and right hypochondriac pain.

WHO in 1986 divided DHF into 4 grades; (I) fever with positive tourniquet test, (II) fever with spontaneous bleeding, (III) circulatory failure shown by a small and frequent pulse rate, restless, and (IV) profound shock (pulseless and reduced blood pressure). Diagnosis of DHF is based on WHO criteria in1986, consists of clinical features and laboratory examinations. This criteria is used to prevent overdiagnosis. Two of clinical criteria added with thrombocytopenia and hemoconcentration that tend to be higher are enough to establish the diagnosis. Pleural effusion and/or hypoalbuminemia enhance the diagnosis, especially appearance of anemia and/or bleeding. In shock cases, increased hematocrit and thrombocytopenia will support the diagnosis of DHF.

Basic principles of laboratory diagnosis are 2 types; I. Specific, virus isolation, detection of antigen or RNA in human serum or tissues. Serologic diagnosis consists of; (1) Haemaglutination Inhibition test (HI test), (2) Complement Fixstion test (CF test), (3) Neutralization test (NT test), (4) IgM Elisa (Mac Elisa), (5) IgG Elisa. Evaluation of serology when the titer of antibody is higher in convalescence compared with
acute phase (as much as 4 times or more).
II. Aspecific; thrombocytes, hematocrit, serum albumin, plasma sodium, transaminase, blood ureum, clotting time blue plasma lymphocytes, radiology, and others. (5)

Leukocytes count maybe normal or lowered in the phase, dominated by a typical lymphocyte known as blue plasma lymphocyte. Increased blue plasma lymphocyte >15% occur on the 3rd day (before decreasing temperature or becoming shock). (1,2)

Management of DHF depends on clinical manifestations and symptoms of emergency like shock, continuous vomiting, seizure, decreased consciousness, melena and hematesis. When symptoms are not apparent, patient is suggested be examine hemoglobin, hematocrit and thrombocytes count. If the next two days the fever still exist, and serial examinations of hemoglobin, hematocrit and thrombocytes tend to be higher, it is suggested to hospitalize the patient.

In the hospital, when there is on apparent any sign of shock, the maintenance of intravenous fluid drip (IVFD) may be given for 6-7 ml/kg/hour. Evaluation of vital signs and serial laboratory examinations should be performed every 6 hours for about 12-24 hours. Many types of solutions are usually provided, for examples ringer lactate (RL) <, NaCl 0.9%, dextrose 5% RL. If the patient’s condition improves gradually, the reduce speed of IVFD to 5 or 3 ml/kg/hour and terminate after 24-48 hours. (1,2)

However, when patient falls into severe state such as shock or preshock, the management must be changed immediately. In this case patient is classified as grade III or IV. Resuscitation solutions must be given by increasing volume og IVFD to 20 ml/kg/hour and vital signs should be monitored for about 30 minutes. If there is still no improvement, the same management may be repeated. In the next opportunity, if there is progression of disease, then the amount of fluid must be reduced gradually every 6 hours, respectively 10,7, and 5-3 ml/kg/hours, respectively 10,7, and 5-3 ml/kg/hour. We must be careful when giving IVFD more than 48 hours after the disappearance of shock, because hypervolemia may occur during the convalescence stage.

When shock still persist after loading the first 20 ml/kg/hour fluid, then the same mechanism may be repeated once again. If the condition is still unstable, patient is in profound shock and fluid must be changed with colloid solutions 10-20 (max 30)ml/kg/hour. One hour later, if the condition is still unstable, even after correction of metabolic acidosis, then other alternative should be considered depending on laboratory findings. When hematocrit is still high, colloid should be provided as much as 20 ml/kg/hour. But, if it tends to decrease, fresh blood transfusion should be given for about 10ml/kg.

The same mechanism may be repeated depending on the patient’s response. (1,2) The complications are usually shock, bleeding encephalopathy, and disseminated intravascular coagulation (DIC).

Acute appendicitis is usually suffered by children under 14-year old and adolescents. (4,9) The early symptom is usually abdominal pain. Especially for children, pain often revealed in epigastric, periumbilical, abdominal colic and McBurney point tenderness. (4)

Appendicitis is caused by infected fecalith or calculi and others. Mixed intestinal organisms generally grow but anaerob are particularly important as causes of intraperitoneal abscess after perforation or surgery. Associated disease may delay the diagnosis of appendicitis and increase the risk of perforation, but it is doubtful that systemic infections predispose to or cause appendicitis. (3)

Histologically, acute appendicitis is almost caused by some obstructions of the lumen. The appendical mesentery become narrow that the distal portion of appendix, undergoes torsion, producing acute ischemic necrosis. Appendiceal obstruction has also been attributed to hyperplasia of submucosal lymphoid tissue presumably as a result of infection. Nonobstructive appendicitis is rare. Both
Clinical manifestations and tissue changes are less severe in nonobstructive appendicitis, and resolution without perforation may occur. (3)

In acute appendicitis, pain is invariably present. Initially, when the pathology is confined to the mucosa and muscular layers of appendix, it is cramp and periumbilical pain. If visceral and parietal peritoneal layers involve in the inflammation, however, then pain is localized to the area immediately overlaying the appendix. Movement such as jumping, driving over bumps in car, coughing and sudden turning movements aggravates the pain. At this stage there is severe tenderness over the appendix, fever, tachycardia and leukocytosis. (3)

Diagnosis of acute appendicitis is rather difficult in children. It is reported that incidence of perforation is high enough in children (20%). All acute appendicitis admitted in RSAB Harapan Kita in 1996-1998, 36% were related to difficult cases. (9) Beside clinical manifestations, laboratory findings are also important. The teenager with early appendicitis is unlikely to have leukocytes count higher than 15,000/ul, but in infants it may show leukocytes response of 20,000/ul, or even more before perforation. Radiology may detect intestinal obstruction, a calcified appendicolith, or pneumonia. Acute appendicitis appears as an edematous enlarged and inflamed lesion by ultrasound examination. (3)

Emergency appendectomy is the treatment of choice for early acute appendicitis. Only under the most extreme circumstances should operation be delayed more than a few hours. Recovery is rapid, and the child is active in 3-4 days. It is essential that antibiotics be given before the operation when the appendix has ruptured to ensure adequate blood and tissue level of the drug used. (3)

The complications of appendicitis are usually divided to preoperation and postoperation. During preoperation can occur perforation and peritonitis. In children, the duration of appendicitis before tupture is usually so short. The mean time between the onset and the diagnosis for nonperforated appendicitis is 36 hours compared wuth 67 hours associated perforation. If the diagnosis is delayed for more that 36 hours, the perforation rate is 65%. Because visceral and parietal peritoneum is involved in the inflammation, prolonged paralytic ileus often follows generalized peritonitis. The most common postoperative complication is infection of the wound. (3)

The above case was first diagnosed as acute appendicitis based on physical examination and laboratory findings. None of febrile symptom was clinically obtained. Symptom of abdominal pain in DHF often occurs for about 37,4-51,7% in children and 17-50% in adult. (1) While the operation was carried out, there were no clinical and laboratory signs of DHF. But during the operation, the surgeon obtained significant peritoneal fluid and suspected it due to DHF. The similar case was not rare occurred in others hospital such as Sumber Waras Hospital in Jakarta and also in Medan. (11) The important thing is to examine serologic test to establish the diagnosis especially during the outbreak of DHF such as in Surabaya in 1998. (10)

Conclusion

Based on this evidence, a silent DHF might overlap with acute appendicitis. Limitation of specific signs and symptoms of DHF and acute appendicitis has been a problem. If overlapping diagnosis, then the operation should be postponed to prevent complication such as bleeding and shock. Otherwise, we should give the stronger antibiotic treatment until the situation became favorable to conduct appendectomy.

We had valuable experienced on this case. Actually, to establish a diagnosis of DHF we could have better confirm a specific serologic test and acute appendicitis by radiological examination. Based on this evidence, this is not a neglected and mismanages case, because pathological examination result indicated acute appendicitis. Diagnosis of DHF could have been established by clinical and laboratory examinations related to WHO.
criteria. It was rather difficult to make a decision at that time.

Reference:

1. Sumarmo PS. Peran serta masyarakat serta ilmu pengetahuan dan teknologi kedokteran dalam pencegahan penyakit menular (Full manuscript of promotion as Professor in Child Health Medicine at Medical Faculty University of Indonesia), Jakarta, 1990.