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Case Report

Febrile jaundice patient with hypophysis tumor and clinical symptoms of Weil's disease: what do we think?

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ABSTRACT

Febrile jaundice is a condition that can be caused by infection and non-infection, one of the causes of infection is leptospirosis. Leptospirosis is caused by bacteria of the genus Leptospira. Leptospirosis is a fairly complex condition and can involve various organs. Severe leptospirosis is called Weil's disease. Weil's disease is a febrile jaundice condition involving various organs, especially the liver and kidneys. The recommended serological tests for Leptospirosis are microscopic agglutination test (MAT) and IgM enzyme linked immunosorbent assay (ELISA). Negative serological results can occur in some cases with typical clinical conditions, requiring re-examination. Pituitary tumors are benign tumors with slow growth. It is very rare to cause liver or other organ metastases.

Keywords: Leptospirosis, Weil's disease, IgM anti-Leptospira, Pituitary tumor

INTRODUCTION

A common clinical manifestation seen in routine practice is febrile jaundice. This can be caused by infectious and non-infectious disorders. Family members and physicians may observe this condition while patients may be fully ignorant that they have a condition. Patients with fever and jaundice should be subjected to a thorough history, physical examination, and detailed investigations before any treatment is given. This is done in order to rule out any other symptoms that could indicate a different diagnosis, such pain or other systemic signs. It's critical to rule out a number of potential causes when a patient exhibits jaundice and a prolonged fever.[1]

Infectious causes can be varying from bacterial (typhoid, typhus, borreliosis, leptospirosis), viral (hepatitis, dengue, COVID, Lassa, Ebola, mumps, measles, rubella), and parasitic (malaria, toxoplasmosis, schistosomiasis). The majority of non-infectious etiologies are connective tissue disorders, medications, and malignancies. Febrile jaundice may be caused by cholestasis or hemolysis that releases pyrogenic material and hemoglobin into the bloodstream, as well as biliary secretion blockage. The three stages of bilirubin metabolism are prehepatic, intrahepatic, and post-hepatic. If any of these stages are disturbed, jaundice may result.[1] Jaundice is a common issue that can result from a number of illnesses. Jaundice with conjugated (with or without cholestasis) or unconjugated bilirubin must be distinguished from one another.[4]

Leptospirosis is a zoonotic infection found in all over the world, that is caused by spirochetes from the genus Leptospira. It is more common in tropical areas, particularly during the rainy season, where there are few supplies and rats are present.[3]

In this case report we present a 43 years old female patient with febrile jaundice with cerebral tumor and clinical symptoms of Weil's disease.

CASE REPORT

A 43 years old female patient came to the Emergency Department of Wangaya Regional Hospital on 14 July 2024 with a primary complaint of severe headache. The

headache had been felt since 1 day before and was felt continuously. The headache was felt mainly in the forehead to the back of the head. Complaints of headache were accompanied by dizziness which was mainly felt when opening the eyes and changing body positions. The patient felt nauseous and vomited 6 times. The patient also had a fever since 1 day before coming to the hospital. The fever was not accompanied by nosebleeds, bleeding gums, black stools and reddish spots on the body. There was no history of patient visiting malaria endemic region. In addition, the patient also complained of pain in the pit of the stomach which felt sharp but did not penetrate to the back. The chest felt full and hot. The patient complained of pain in the lower abdomen which was felt especially when urinating. Every time the patient urinated, the patient felt dysuria. The patient felt urination and defecation within normal limits. The patient complained of pain in her calves, especially in her left calf. The patient's family said that there were several rats around the house. There was no puddle of water in the patient's environment and the patient's family said there was no smell of rat urine. The patient's job is a chicken butcher. None of the patient's family and surroundings have the same complaints. The patient denied a history of hypertension, diabetes mellitus, and a history of heart disease, but the patient had a history of a brain tumor that was known since 6 months ago. The patient had never been treated for his brain tumor. The patient only takes paracetamol to reduce the fever and headache.

The patient came with somnolent consciousness with GCS E4V5M6 and appeared to be in pain. The patient's vital signs showed blood pressure of 100/60 mmHg, pulse rate of 100 beats/minute, respiratory rate of 20 breaths/minute, temperature of 37.4 ^{o}C, oxygen saturation of 99%. On physical examination, scleral icterus was found in both eyes accompanied by conjunctival suffusion in the patient's left eye. The patient's conjunctiva did not appear anaemic. On thorax examination, the heart and lungs were within normal limits. On abdominal examination, there was tenderness in the epigastrium, supra pubic and splenomegaly. Both of the patient's feet felt warm with a capillary refill time of <2 seconds, no oedema was found in the legs, and there was tenderness in both gastrocnemius muscle especially the left side.

Laboratory examinations conducted in the form of complete blood count obtained white blood cells (WBC) results of 11.02×103/Ul, haemoglobin (Hb) 15.3 g/dl, haematocrit (HCT) 41.9%, platelet count (PLT) 170×103/Ul. Liver function tests showed an increase in serum glutamate pyruvate transaminase (SGPT) with results of 329 U/l and serum glutamic-oxaloacetic transaminase (SGOT) with results of 459 U/l. Kidney function tests also showed an increase in blood urea nitrogen (BUN) 75 mg/dl and serum creatinine (SC) 3.1 mg/dl. Random blood glucose examination was normal at 111 mg/dl. Electrolyte examination showed sodium levels of 132 mmol/l, potassium 3.5 mmol/l, and chloride 95 mmol/l.

The patient was consulted to a neurologist. The neurologist diagnosed the patient with severe cephalgia and provided therapy in the form of IVFD NaCl 0.9% 20 dpm, ketorolac 2×1 ampoule IV, eperisone 3×1 tablet, omeprazole 1×1 ampoule IV, and diazepam 1×2 mg tablet and a consultation plan with an internist. The internist diagnosed the patient with suspect Weil's disease and observation transaminitis. The therapy given was ceftriaxone 2×2 grams IV, curcuma 3×1 tablets, folic acid 2×2 tablets, and a follow-up examination plan was carried out in the form of bilirubin profile examination, alkaline phosphatase, gamma-glutamyltransferase, urinalysis, and complete blood count repeat the next day.

On the next day laboratory examination were performed and the results in complete blood count shows WBC 6.75×103/Ul, Hb 14.3 g/dl, HCT 41.8%, and PLT 143×103/Ul. Urinalysis results on macroscopic examination obtained leukocyte esterase levels are 75 Leu/Ul, negative nitrite, 1+ protein, negative ketone, negative blood, negative bilirubin, and negative urobilinogen. In urine sediment, leukocytes were found 3-4/WFV, squamous epithelium 1-2/WVF, and positive bacteria. Total bilirubin levels were found 1.91 mg/dl, direct bilirubin 1.0 mg/dl, and indirect bilirubin 0.91 mg/dl. Alkaline phosphatase was found 58 U/l, and gamma GT 111 U/l.

The patient was hospitalized for 7 days and underwent several examinations to confirm the diagnosis such as abdominal ultrasonography (USG) examination which showed fatty liver (grade I-II) and splenomegaly. Multislice computed tomography (MSCT) scan results showed a solid mass with a necrotic centre in the sella turcica extending to the suprasellar with suspected macroadenoma. HBsAg and anti-HCV examinations were negative. The patient's lipid profile showed a low-density lipoprotein (LDL) value of 89, high density lipoprotein (HDL) 10, total cholesterol 203, and triglycerides 493. Uric acid examination showed a value of 5.8. The patient underwent IgM anti-Leptospira serology examination on the fifth day and showed negative results.

The initial therapy that was given by the internist were ceftriaxone 2×2 grams IV, curcuma 3×1 tablet, and folic acid 2×2 tablets, the therapy continued and given additional therapy esomeprazole 1×40 mg IV. The initial therapy that was given by the neurologist was IVFD NaCl 0.9% 20 dpm, ketorolac 2×1 ampoule IV, eperisone 3×1 tablet, omeprazole 1×1 ampoule IV, diazepam 1×2 mg tablet continued and given additional therapy in the form of hydrocortisone 2×1 ampoule IV, betahistine 3×6 mg orally, paracetamol 3×1000 mg orally, and ondansetron 2×4 mg IV. After being treated for 8 days, the patient's complaints improved and another laboratory examination was performed such as liver function test and renal function test. The results were SGPT 135 U/l, SGOT 223 U/l, BUN 45 md/dl, and SC 1.4 U/l. The patient was planned to go home by the two specialist doctors and for a

check-up at the internal medicine and neurology polyclinic after 3 days.

Table 1: Faine criteria for leptospirosis.[2]

Criteria	Score
Part A: Clinical manifestation	
Headache	2
Fever	2
Fever >39 ºC	2
Conjunctival suffusion	4
Meningism	4
Myalgia	4
Conjunctival suffusion+meningism+myalgia	10
Jaundice	1
Albuminuria/nitrogen retention	2
Haemoptysis/dyspnoea	2
Part B: Epidemiological factors	
Rainfall	5
Contact with contaminated environment	4
Animal contact	1
Part C: Bacteriological and laboratory findings	
Isolations of leptospira in culture–diagnosis certain	
PCR[a]	25
Positive serology	
ELISA[b] Ig M positive	15
SAT[c] positive	15
Other rapid tests[d]	15
MAT[e] – single positive in high titer[f]	15
MAT[e] – rising titer / seroconversion (paired sera)	25
Presumptive diagnosis of leptospirosis is made of	
Part A or part A and part B score of: 26 or more	
Part A, B, C (total): 25 or more	
A score between 20-25 suggest leptospirosis is a possible diagnosis	

[a]Polymerase chain reaction, [b]enzyme linked immunosorbant assay, [c]slide agglutination test, [d]other rapid test – latec agglutination test/leptodipstick/LeptoTek lateral flow/LeptoTek Dri dot test, [e]microscopic agglutination test, [f]any one of the tests only should be scored

DISCUSSION

Combining the patient comorbidities, her job, and laboratory findings the differential diagnoses included leptospirosis, hepatitis, and liver metastatic of the cerebral tumor. But the clinical findings are more dominant for leptospirosis.

Leptospirosis is a zoonotic infection found in all over the world, that is caused by spirochetes from the genus Leptospira. It is more common in tropical areas, particularly during the rainy season, where there are few supplies and rats are present. Leptospira is maintained in nature due to chronic kidney infection in domestic and wild carriers (rodents, among other small mammals, as well as cattle and domestic dogs and cats) that colonize the brush border of the proximal renal tubule. Direct or indirect contact with an infected animal's urine or tissues can result in infection. Leptospira spirochetes spread throughout the bloodstream after entering the body through skin abrasions or cuts, mucosal membranes, or aerosol inhalation.[3]

After 2 to 30 days of incubation, symptoms may appear. The disease progresses through two stages, known as the septic and immunological phases, though these are frequently combined in the most severe cases. The most typical symptoms include headaches, myalgia, and fever. 50% of the cases may result in nausea, vomiting, and diarrhea. A conjunctival suffusion is one of the disease's highly distinctive symptoms.[4] Leptospiremia and the associated vasculitis explain the broad spectrum of its clinical presentation.[3]

Leptospirosis may manifest as a severe illness, a self-limited systemic disease (90 percent of infections), or a subclinical condition. In the first 5 to 7 days, there may be a septic phase marked by the presence of Leptospira organisms in the blood or cerebrospinal fluid. These organisms can cause high fever, headache, abdominal pain, nausea, vomiting, diarrhea, coughing, and conjunctival suffusion, which is characterized by red eyes without secretions. Patients may manifest with jaundice, renal failure, breathing difficulties, hemoptysis, aseptic meningitis, heart arrhythmias, and pancreatitis during the 4–30 days posterior immunological phase, which also produces circulating IgM antibodies and leptospiruria. Weil's disease is a severe form of leptospirosis. It is characterized by impaired liver function, as well as lung and renal dysfunction, and may manifest following the acute phase. When serious disease is left untreated, the mortality rate rises to 40%.[3]

Although leptospirosis-related laboratory results are typically unspecific, 2-3 patients may present with a mild leukocytosis (shift to the left) and thrombocytopenia. There may be a rise in inflammatory markers including C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR). Serum creatinine often increases in patients with more severe renal symptoms, and hypokalemia and hyponatremia may also happen. Conjugated hyperbilirubinemia is frequently detected in mild clinical symptoms and can reach levels of 40–80 mg/dl. Additionally, there is a little rise in serum transaminases.[6] The liver profile of patients is characterized by a marked direct hyperbilirubinemia (up to 80 mg/dl), accompanied by a small increase in transaminases (<200 U/l) and other cholestasis-indicating enzymes (ALP, GGT) that are 2-3 times the normal value. Leptospira spirochetes enter the Disse gap, damage the hepatocytes, changing their intercellular binding, and cause bile to leak from the bile canaliculi into the sinusoids, which explains for the extremely high levels of direct bilirubin, with no major changes in ALP or GGT. Furthermore, transaminase levels are not raised since there is no hepatocellular necrosis.[3] A urinalysis revealed occasional proteinuria, microscopic

hematuria and pyuria. Serum amylase and creatinine kinase levels may also be increased. The cerebrospinal fluid (CSF) fluid is examined for indications of aseptic meningitis, including normal glucose levels, moderate protein increase, and lymphocytic pleocytosis.[6] The severity of renal involvement ranges from established acute kidney failure to mild, non-oliguric acute kidney injury. While most survivors regain their kidney function, some individuals have chronic renal disease that lasts a lifetime.[3]

Our patient develops severe headache and fever at the same day followed with nausea and vomiting. The headache seems to be correlated with her history of cerebral tumor which she discovered after having chronic headache. Our patient also developed conjunctival suffusion on her left eye one day after she was treated in the hospital. The patient also had a general myalgia but seems to be more dominant on both of her calves especially left calves. On the laboratory finding we got the patient had a mild elevation of leukocytes, elevated liver enzyme, elevated kidney functions, elevated total bilirubin and direct bilirubin, which suggest the involvement of multiple organs. Our patient had an increase of total and direct bilirubin. But quite a significant increase of liver enzyme (>200 U/l) and also increase levels of gamma GT. The patient also had an increase of renal function test without oliguric symptoms. On the urinalysis our patient also had proteinuria, pyuria, and microscopic hematuria.

There are direct and indirect detection methods available. Dark-field microscopy and the polymerase chain reaction technique are two direct detection techniques that can be used in both blood and urine. The microscopic agglutination test (MAT), the conventional indirect method, has a 77.4% sensitivity and a 97.6% specificity. It involves the reaction of live antigens with antibodies in a blood sample.[3] An assay for IgM antibodies, which can be detected after about the fifth day of sickness, is another serologic technique for diagnosing leptospirosis. Although they are screening tests, positive IgM assays are not an indicator for confirming leptospirosis. These antibody assays have the potential to enhance the diagnostic capacity of many laboratories, particularly in developing countries, where most cases occur.[5] Particularly in places with limited resources, laboratory confirmation of serological tests can be challenging. Antibodies peak in 3–4 weeks and can be found from day 6 to day 10 of sickness. Acute and convalescent comparison has high sensitivity and specificity. A serological examination can be used to identify general or serogroups.[6] Serology is used to diagnose leptospirosis in the majority of cases. The microscopic agglutination test (MAT), the reference standard assay, involves reacting live antigens representing various leptospire serogroups with blood samples and then observing the resulting agglutination under darkfield microscopy. A 4-fold increase in MAT titer to one or more serovars between acute-phase and convalescent serum specimens obtained concurrently is considered a serologically proven case of leptospirosis. However, a single titer of at least 1:200 obtained after the onset of symptoms also suggests recent or current infection with leptospirosis. The MAT is a serogroup-specific assay and should not be used to infer the identity of the infecting serovar, but knowledge of the presumptive serogroup may be of epidemiologic value in determining potential exposures to animal reservoirs.[5] Our patient's IgM anti-Leptospira examination was found negative, it may be due to the time of the testing which is on the 5th day where the antibodies is too early to be developed or it may be due to the administration of the antibiotics from the first day. We tested the patient on the 5th because our patient clinically has been getting better.

According to Munoz et al, there was only one laboratory-confirmed case in 39.9% of all suspected and/or suspected cases outbreaks. Serological enzyme-linked immune-sorbent assay (ELISA) and/or MAT were used to diagnose the 61 outbreaks that were reported. Sensitivity at the onset of symptoms is limited because the antibody response needed for MAT testing is frequently insufficient to detect until the second week of sickness (when the immunological phase begins). During the first week of the disease, a number of serologically based techniques have been developed to identify the host's early response. ELISA is the most often utilized. This test looks for IgM for both pathogenic and non-pathogenic Leptospira serogroups using common Leptospira antigens. Serological tests for both the MAT and IgM ELISA did occasionally produce negative results at the start of the investigation, with an onset of less than a week. However, the first negative serology test result will convert to a positive one with a re-serological examination after 10 days from the start of symptoms. Routine laboratory testing can help with diagnosis and treatment in some situations, particularly when a precise leptospirosis diagnosis is not readily available at a medical institution or in close proximity to a medical facility with a rapid turnaround time.[6]

The diagnosis of leptospirosis can also be established according to WHO recommendations, where a diagnostic score, namely the Faine score. Leptospirosis is suspected if the score is ±20, with a strong suspicion if the score is ±24.[2] Our patient had a score of 18 from the Faine criteria for leptopspirosis.

Oral doxycycline is used to treat mild diseases, whereas intravenous penicillin or ceftriaxone is used to treat severe diseases. Treatment support is crucial. Intense intravenous fluid resuscitation is recommended for patients with acute renal injury, and early hemodialysis initiation lowers mortality if the patient develops oliguric kidney failure.[3] Antibiotics seem to improve renal function and shorten the duration of fever and sickness. Experts advise using antibiotics for Leptospira symptoms even if there is no evidence of a reduction in mortality. Doxycycline and ceftriaxone are equally effective substitutes for penicillin, which is often administered for seven days (10 days for severe leptospirosis).[4] This patient had an improvement in

her clinical symptoms and also the renal function after being treated with ceftriaxone for 7 days.

Studies have found that viral hepatitis (A to E) is the most common infection causing febrile jaundice.[1] Depending on the type of hepatitis virus causing the infection, each person may have a varied clinical presentation of viral hepatitis. Patients may be completely asymptomatic or only somewhat symptomatic. Regardless of the kind of hepatitis virus infection, individuals with viral hepatitis usually experience four phases. The following list includes basic physical exam findings as well as findings unique to each kind of hepatitis. Phase 1 (incubation/viral replication phase) - patients are usually asymptomatic in this phase, and laboratory studies are positive for markers of hepatitis. Phase 2 (prodromal phase) - patients in this phase may present with anorexia, nausea, vomiting, malaise, pruritus, urticaria, arthralgias, and fatigue. These patients are often diagnosed as having gastroenteritis or other viral respiratory infections. Phase 3 (icteric phase) - patients in this phase present with dark-colored urine and pale-colored stool. Some patients develop jaundice and right upper quadrant pain coupled with liver enlargement. Liver enzymes will be elevated. Phase 4 (convalescent phase) - patients typically start noticing the resolution of symptoms, and laboratory studies show liver enzymes returning to normal levels.[7]

We have excluded hepatitis as the patient diagnosed based on her symptoms and laboratory findings. As we explained above the hepatitis is classified to 4 phases. The patient does have hepatitis symptoms such as anorexia, nausea, vomiting, malaise, and arthralgia which is a part of the prodromal phase, but the patient also had both of the sclera icteric and also elevated liver enzymes which is a part of the icteric phase. We later on checked the serological examination for Hepatitis B and C but it was found negative.

The sella turcica, a bony indentation in the sphenoid bone, contains the pituitary gland. There are anterior and posterior segments of the pituitary gland. Whereas the posterior pituitary (neurohypophysis) is made of neural tissue, and the anterior pituitary (adenohypophysis) is made of glandular tissue. The anterior pituitary produces and secretes six hormones: growth hormone (GH), prolactin, thyroid-stimulating hormone (TSH), adrenocorticotrophic hormone (ACTH), follicle-stimulating hormone (FSH), and luteinizing hormone (LH). The posterior pituitary gland stores the hormones anti-diuretic hormone (ADH) and oxytocin.[9]

Pituitary gland tumors arising from the anterior pituitary gland are usually benign. Rarely, pituitary tumors may metastasize and are then termed pituitary carcinoma. Pituitary cancer includes pituitary carcinoma and systemic metastases to the pituitary gland.[9] The majority of pituitary tumors are benign and slow-growing. They are categorized by size or the cell from which they originate. Depending on their size, pituitary adenoma can be classified as big tumors, macroadenomas, or microadenomas. Tumors smaller than 10 mm are referred to as microadenomas, whilst those greater than 10 mm are referred to as macroadenomas. The size of giant pituitary tumors exceeds 40 mm. There are functional pituitary adenomas where the cell type that makes them up results in increased anterior pituitary hormone release. The symptoms may be differentiated from the mass effect and impaired function of the hormones. Hormonal deficiencies, headaches, and vision impairment can all result from the mass effect. Between 40% and 60% of patients are found to have visual impairment. Visual field abnormalities result from the compression of the optic chiasm caused by suprasellar expansion of the pituitary adenoma. The most common pattern is a bitemporal flaw, which is followed by homonymous defects. Diplopia may result from oculomotor nerve involvement, and invasive cancers may also impact the fourth, fifth, and sixth cranial nerves. Although it is a non-specific symptom, headache is frequently reported in pituitary adenoma patients. Patients with pituitary macroadenoma may exhibit one or more anterior pituitary hormonal deficits.[10]

Nearly 25% of all cases of cancer metastasize to the liver, making it one of the most frequent sites. Numerous primary tumors can cause metastases, but because they are the most prevalent, colorectal adenocarcinomas are the subject of the most research in the literature. Both the portal vein and the hepatic artery supply blood to the liver. Venous drainage from the pancreas, spleen, and nearly the whole gastrointestinal system is collected by the portal vein. Typically, this allows for the liver to process newly digested and absorbed nutrients in first-pass metabolism. In the context of cancer, it also indicates that a wide range of extra-abdominal and abdominal cancers can spread to the liver. Patients may experience a range of symptoms as a result of liver metastases, contingent on the location and severity of the disease. These consist of weariness, weight loss, jaundice, ascites, and abdominal pain.[8]

When evaluating suspected liver metastases, high-quality imaging is crucial. It can help pinpoint the primary condition and confirm the diagnosis. Triple-phase CT and MRI scans are the most widely used imaging modalities. The triple-phase CT scan consists of a non-contrast phase, arterial phase, and venous phase. Liver metastasis and primary liver tumors tend to have the strongest attenuation in the arterial phase and tend to be hypo attenuating in non-contrast studies. CT imaging evaluates metastatic tumor size, morphology, degree of liver disease, and the predicted future liver remnant. MRI is another modality that can be utilized if there is difficulty characterizing a liver lesion. Liver metastasis on T1 weighted imaging appears hypo-intense and hyperintense on T2 imaging 8. On this patient we did not perform Abdominal CT-scan and also MRI. From abdominal USG examination we found that the patient had fatty liver disease (grade I-II) and splenomegaly.

CONCLUSION

Weil's disease is a severe form of leptospirosis. It requires early diagnosis and appropriate management for Weil's disease patients. Repeat serology examination may be recommended in patients with typical clinical features with negative initial examination results. Pituitary tumors rarely cause metastasis.

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