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CASE REPORT

Dengue Haemorrhagic Fever presenting as Acute Abdomen

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حمى الضنك النزفية تظهر بأعراض البطن الحادة

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الملخص: هذا وصف لحالة أنثى سيريلانكية عمرها 38 عاما أحيلت إلى الجراح المناوب وهي تعاني من أعراض البطن الحادة. عانت المريضة على مدى ثلاثة أيام من حمى وصداع وآلام في البطن وإسهال، إلا أن الفحص السريري كان لا يتسق مع تشخيص حالة البطن الحادة. كان عدد الصفائح الدموية (22×10^9/لتر). تم تشخيص الحالة على أنها حمى الضنك النزفية وكان فحص المصل لحمى الضنك إيجابيا. لقد ارتبطت أوبئة حمى الضنك مع مجموعة متنوعة من الأعراض المعدية والمعوية، بما في ذلك أعراض البطن الحادة. إن أعراض البطن الحادة في المرضى الذين يعانون من حمى الضنك النزفية تجعل طرق التشخيص والعلاج صعبة.

مفتاح الكلمات: حمى الضنك؛ الحمى النزفية؛ البطن الحادة؛ تقرير حالة ؛ عُمان.

ABSTRACT: We describe a case of a 38 year-old Sri Lankan female who was referred to the surgeon on call with a picture of acute abdomen. She presented with a three-day history of fever, headache, abdominal pain and diarrhoea; however, the physical examination was not consistent with acute abdomen. Her platelet count was 22 $x10^9$/L. A diagnosis of dengue haemorrhagic fever (DHF) was made and dengue serology was positive. Dengue epidemics have been associated with a variety of gastrointestinal symptoms and signs, including acute abdomen. Acute abdomen in patients with DHF makes the diagnosis and management challenging.

Keywords: Dengue; Haemorrhagic fever; Acute abdomen; Case report; Oman

Dengue virus is a mosquito-borne flavivirus and the most prevalent arbovirus in tropical and subtropical regions of the world.[1] The 2005 World Health Assembly resolution WHA58.[3] on the revision of the International Health Regulations (IHR) has included dengue as an example of a disease that may constitute a public health emergency of international concern with implications for health security due to disruption and rapid epidemic spread beyond national borders.[2] Acute abdomen is an uncommon presentation of dengue haemorrhagic fever (DHF). It is important to take DHF into consideration when making a differential diagnosis for patients with acute abdomen, a history of travel to dengue endemic areas and thrombocytopenia. Reaching the right diagnosis may help in preventing unnecessary surgical interventions for patients with DHF.

Case report

A 38 year-old Sri Lankan lady presented with a three-day history of fever associated with abdominal pain, vomiting and diarrhoea. She also gave a history of headache. These symptoms had started on the day she arrived in Oman from Sri Lanka. She denied any history of skin rashes, urinary symptoms or contact with sick people. She had been working in Oman for the previous four years and had visited her home country, Sri Lanka, for four weeks coming back to Oman three days prior to admission to hospital. Clinical examination showed a sick looking and mildly dehydrated woman. There was no evidence of jaundice, lymphadenopathy or skin rash. She was febrile and hypotensive, but she was not tachypneic or tachycardic. An abdominal examination revealed a generalised abdominal tenderness mainly in the right hypochondrium, but there was no guarding or rigidity. Other systemic examinations were unremarkable. Initial investigations showed haemoglobin of 14.2 g/dl; haematocrit count of

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39.9 %; platelet count 22 x10^9/L; white blood cell count 11.5 x10^9/L with neutrophils of 2.4 x10^9/L and lymphocytes of 4.8 x109/L; mildly raised alanine transaminase (ALT) of 86 IU/I, and alkaline phosphatase of 184 IU/L. Urea and electrolytes, coagulation profile and amylase were normal. An abdominal ultrasonography showed free fluid in the abdomen with a slightly thickened gall bladder wall, but no evidence of gall bladder calculi. The patient was referred to the surgical team with an initial diagnosis of acute cholecystitis. She was kept nil by mouth and was started on intravenous fluids and ceftriaxone injections. On the second day of admission, the patient continued to have severe abdominal pain and a clinically detected guarding abdomen. An urgent computed tomography (CT) scan of the chest and abdomen with oral and intravenous contrast was done. It showed bilateral pleural effusion with right lower lobe collapse and segmental atelectasis, and abdominal and pelvic ascites, but the gall bladder was normal. In view of the CT findings, acute cholecystitis was ruled out and an alternative diagnosis was considered. Malaria screening was negative. The blood film revealed very mild leukocytosis with reactive lymphocytes and a mild neutrophil left shift; the platelets were markedly reduced and showed occasional large forms. The features of the blood film were compatible with a viral infection; in view of the recent travel history to a dengue endemic country, dengue haemorrhagic fever (DHF) was suspected.

The patient was managed conservatively and made a remarkable improvement clinically with a gradual increase in the platelet count. She was sent home on the fifth day after her admission, symptom free and with a platelet count of 224x10^9/L. Dengue serology was done on admission and was immunoglobulin M (IgM) positive; it was repeated two months later and the result was equivocal. Polymerase chain reaction (PCR) for dengue virus was requested on admission, but it was not done due to technical reasons in the laboratory. It was, however, done with the repeated serology two months after the initial presentation and was negative.

Discussion

Dengue virus infection is the most common cause of arboviral disease in the world. The estimated annual occurrence of dengue fever is 100 million cases of dengue fever with 250,000 cases of DHF, and a mortality rate of 25,000 per year.[1] In the last 50 years, the incidence has increased 30-fold with geographic expansion to new countries and, in the present decade, from urban to rural settings.[3] Dengue is endemic in most tropical and subtropical parts of the world. Some 1.8 billion (more than 70%) of the populations at risk for dengue worldwide live in member states of the World Health Organization's (WHO) South-East Asia and Western Pacific regions which bear nearly 75% of the current global disease burden due to dengue.[2] Dengue is transmitted by the mosquito vectors *Aedes aegypti* and *Aedes albopictus*. Dengue infection is caused by any of 4 different serotypes of the virus (DEN-1, DEN-2, DEN-3, and DEN-4). The incubation period is 4–7 days after an infective mosquito bite.[4] The recent emergence of DHF in the Indian subcontinent has been well documented in Sri Lanka. The four dengue virus (DENV) serotypes have been co-circulating in Sri Lanka for >30 years. Over this period, a new genotype of DENV-1 has replaced the old genotype. Moreover, new clades of DENV-3 genotype III viruses have replaced older clades. The emergence of new clades of DENV-3 in 1989 and 2000 coincided with abrupt increases in the number of reported dengue cases, implicating this serotype in severe epidemics. From 1980 to 1997, most reported dengue cases were in children. Recent epidemics have been characterised by many cases in both adults and children. Changes in local transmission dynamics and genetic changes in DENV-3 are probably responsible for the increasing emergence of severe dengue epidemics in Sri Lanka.[5] There is no published data on dengue fever from Oman apart from one case report.[6]

The clinical manifestations of dengue range from asymptomatic infection, self-limited dengue fever to DHF with shock syndrome. The risk of severe disease is much higher in sequential rather than primary dengue infection.[7] The most common symptoms of dengue virus infection are fever, headache, retro-orbital pain, photophobia, backache, severe myalgia and arthralgias. Other signs and symptoms include generalised maculopapular rash, lymphadenopathy, positive tourniquet test, petechiae and other haemorrhagic manifestations. The WHO has set criteria for

diagnosing dengue (with or without warning signs) and severe dengue depending on clinical signs and symptoms and laboratory investigations. The criteria for diagnosing probable dengue fever are living in or travel to a dengue endemic area, fever and two of the following criteria: nausea, vomiting, rash, aches, a positive tourniquet test, leukopenia and any warning sign. The warning signs are abdominal pain or tenderness, persistent vomiting, clinical fluid accumulation, mucosal bleeding, lethargy, restlessness, liver enlargement of >2cm and increase in haematocrit concurrent with a rapid decrease in platelet count. The diagnosis of severe dengue was defined by the WHO as severe plasma leakage leading to dengue shock syndrome (DSS); fluid accumulation with respiratory distress; severe bleeding as evaluated by the clinician, and severe organ involvement (acute liver failure, acute renal failure, encephalopathy, cardiomyopathy), or other unusual manifestations.[3]

About one-third of patients develop one or more complications that include bleeding, seizures, acute renal failure, and DSS. There have been reports of other uncommon clinical complications such as acute hepatitis and hepatic failure,[8] unusual neurologic manifestations,[9] and acute myocarditis.[10] There also have been reports of cases resembling surgical emergencies like acute pancreatitis, acute acalculous cholecystitis,[11] acute appendicitis[12] and non-specific peritonitis.

A retrospective study of patients with DHF and acute abdomen in a dengue epidemic area in southern Taiwan found that, of 328 patients with DHF/DSS, 14 had acute abdomen. DHF/DSS was initially suspected in only 2 of these 14 patients. Presumptive diagnoses of acute cholecystitis (6 acalculus and 4 calculus cholecystitis) were made in 10 patients, non-specific peritonitis in three patients, and acute appendicitis in one patient. Cholecystectomy, percutaneous transhepatic gall bladder drainage, and appendectomy were performed in three patients. In addition to acute abdomen, these patients had a variety of symptoms and signs such as fever, chills, myalgia, headache, rashes, and petechiae, which were similar to those found in patients with DHF without acute abdomen.[4] However, the predominant acute abdomen in these patients was the overwhelming concern and distracted clinicians from the typical clinical manifestations of dengue illness that should have been noticed.[13]

The treatment of DHF is mainly supportive care. In 2005, Wills *et al*. investigated fluid treatment for Vietnamese children with DSS.[14] With the use of simple clinical monitoring tools, these authors have shown that the administration of Ringer's lactate leads to the same outcome among children with moderate shock as does the administration of colloids.[14] Guidelines for intravenous fluid therapy of DHF have been developed by the WHO. For patients with shock, an initial bolus of 5% dextrose in normal saline or Ringer's lactate (10 to 20 mL per kg of body weight) infused rapidly is recommended, followed by continuous infusion (10 to 20 mL/kg per hour) until vital signs and urine output normalise. The infusion rate can then be gradually reduced until it matches plasma fluid losses.[3]

The symptoms and signs of our patient were mostly similar to those that have been previously described for dengue fever. Also the presence of thrombocytopenia, mild derangement of liver enzymes, evidence of plasma leakage (pleural effusion, ascites, hypoalbuminemia and raised haematocrit) and positive IgM serology for dengue all supported the diagnosis of DHF. However, the whole clinical picture was masked by acute severe abdominal pain and tenderness. The diagnosis was not made until the patient was referred to the infectious diseases team. This case underlines the need for medical staff to be aware of travel and tropical medicine including geographical epidemiology.

Conclusion

Acute abdomen is an uncommon presentation of DHF. It is important to take dengue fever into consideration when making a differential diagnosis on acute abdomen for patients returning from dengue endemic regions. It may help in preventing unnecessary surgical intervention in DHF patients. It is also important for medical personnel to be aware of the epidemiology of tropical diseases.

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