DENGUE RELATED ACUTE ACALCULOUS CHOLECYSTITIS: A CASE REPORT

Dr Tan Yee Leng, Dr Farhad Fakhrudin Vasanwala & Dr Matthew Ng

ABSTRACT

We describe a case of a 41-year-old Chinese male who was seen in our department for fever, rash, and abdominal pain. He was found to have acute acalculous cholecytitis secondary to Dengue Haemorrahgic Fever. Acalculous cholecystitis can occur in dengue fever and dengue haemorrhagic fever. Treatment is conservative unless there is no resolution. In the context of Singapore with dengue fever and Dengue Haemorrhagic fever being highly endemic, there is a need to be familiar with the atypical presentation of this common disease.

SFP 2008; 34(4): 77-79

INTRODUCTION

We describe a case of a 41-year-old Chinese male who was seen in our department for fever, rash, and abdominal pain. He was found to have acute acalculous cholecytitis secondary to Dengue Haemorrahgic Fever.

CASE REPORT

Presenting complaint

Mr. H was a 41-Chinese year-old man working as a construction worker with no known past medical history of note. He was admitted on Sep 5th 2008 with fever associated with generalized myalgia and arthalgia for the past 3 days. He also complained of right upper quadrant pain and loss of appetite. He developed generalized flushing with red spots over the shins bilaterally on the day of admission. He had no recent travel in the past 6 months and there was no contact history.

Social history

He is a Singapore permanent resident. He worked as a project supervisor at a construction site at Kranji. He had been in Singapore for the past 8 years.

TAN YEE LENG, Medical Officer (Trainee), Family Medicine Continuing Care, Singapore General Hospital

FARHAD FAKHRUDIN VASANWALA, Registrar, Family Medicine Continuing Care, Singapore General Hospital

MATTHEW NG, Associate Consultant, Family Medicine Continuing Care, Singapore General Hospital

Clinical examination

On clinical examination, Mr. H was alert and non-toxic looking. He had a temperature of 38.7 degree Celsius, blood pressure of 119/76mm Hg, pulse rate 101 per min and respiratory rate 18 per min. There was no pallor, jaundice or peripheral edema. Generalized flushing with petechiael rash was noted over both lower limbs. Ear, nose and throat examination was unremarkable.

Examination of his abdomen revealed epigastric tenderness. There was no rebound tenderness or guarding. Bowel sounds were present. No masses, organomegaly or ascites were present. His cardiovascular, respiratory and neurological examination was unremarkable.

Laboratory investigations

Laboratory investigations on admission were as follows:

- Haematological results: Total white cell counts - 2.69 x109 /L [4.0-10.0], polymorphs 87.0% [40-75], lymphocytes 10.7% [15-41], monocytes 1.6% [2-10] with presence of atypical mononuclear cells, Haemoglobin 15.2 g/dL [14.0-18.0], platelets 66 x 109/L[140-440], haematocrit 42.6% [38-52].
- Liver function test results: Alkaline phosphatase(ALP) 94 U/L [32-103], aspartate transaminase(AST) 115 U/L [15-33], alanine transaminase(ALT) 147 U/L [7-36], total bilirubin 12 UMOL/L [3-24].

Other test results:

- Prothrombin time 11.7seconds [9.2-11.2], APTT, 39.3 seconds [27.0-36.1], amylase 77 U/L [44-161], C- Reactive Protein (CRP) 4.6 mg/L [0.2-8.8]
- On day 4 of the fever, he was screened for Dengue serology, peripheral blood film for malarial parasite and Leptospirosis.
- Electrocardiography showed sinus tachycardia. Chest X ray was normal with no air under diaphragm.

Initial diagnosis

Our initial diagnosis was that Mr. H has a viral fever as evidenced by thrombocytopenia and transaminitis. The presence of petechie and flushing suggested Dengue. The differential diagnoses were Leptospirosis and Chikungunya fever.

Progress

Mr. H was given intravenous fluids at the rate of 3.5 liter per day. His fever and dyspepsia was treated symptomatically. His full blood count was done daily.

Serology for Dengue, Leptospirosis and blood film for malarial parasites done at day 4 of fever were negative.

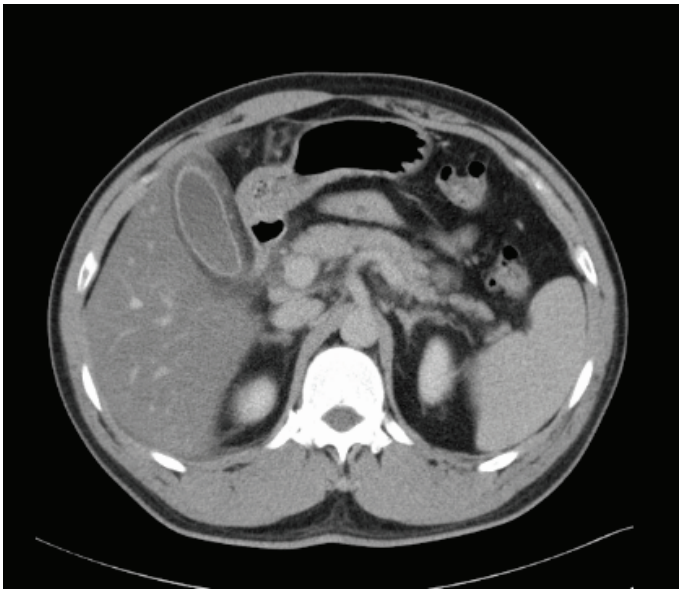

Figure 1. Acute acalculous cholecystitis: The gallbladder shows marked mural thickening and pericholecystic fluid. No intraluminal calculus is evident. Common bile duct is not dilated. The inflammatory fluid has tracked into the pelvic cavity via the hepatorenal pouch of Morrisons, right paracolic gutter and right iliac fossa.

On Day 6 of fever, the patient complained of increasing dyspepsia with loss of appetite. Fever was still present. Abdominal examination revealed right hypochondrial tenderness with a positive Murphy's sign.

Repeat investigations showed total white blood cells 1.09X109/L [4.0-10.0], neutrophils 65%[40-75], lymphocytes 29.4%[15-41], monocytes 5.5%[2-10], platelet of 20 x109/L[140-440], AST 327 U/L [15-33], ALT 261 U/L [7-36], ALP 142 U/L [32-103], total bilirubin 16 UMOL/L [3-24].

Computed tomography scan of the abdomen and pelvis confirmed acute acalculous cholecystitis. [Figure 1]

Further viral serologies [Chikungunya PCR and serologies, Hepatitis A, B, C markers, serologies for Ebstein Barr virus, Cytomegalovirus and Dengue] were sent on Day 7 illness. This second set of Dengue IgM antibodies was positive.

Patient was continued on supportive treatment with fresh frozen plasma and platelet transfusion. His fever settled on Day 10 of illness with resolution of his abdominal pain. Platelets came up to 146 on Day 11 of illness. Mr. H was discharged well on Day 11.

DISCUSSION ON ACUTE ACALCULOUS CHOLECYSTITIS

Dengue fever and Dengue Haemorrhagic fever is highly endemic in Singapore. In 2007, Singapore had 6765 cases of Dengue fever and 161 cases of Dengue haemorrhagic fever based on the Ministry of Health Singapore epidemiological date[1]. Hence as Family Physicians in the community we must be familiar with the typical and even atypical presentations of this common disease.

Clinical features

Acute acalculous cholecystitis (AAC) is an acute necro-inflammatory disease of the gall bladder without evidence of calculi or sludge. It accounts for 2-15% of all cases of acute cholecystitis. Patients present with right upper quadrant abdominal pain, fever, positive Murphy sign, and abnormal liver function tests. Ultrasonographic criteria for diagnosis of AAC include gallbladder wall thickening over 3mm, distention of gallbladder, localized tenderness, pericholecystic fluid and sludge[2].

Risk factors

AAC is traditionally known to happen in patients who are critically ill. They present with a more fulminant course with significant mortality as high as 90%. Risk factors include cardiac surgery, abdominal vascular surgery, major trauma, burns, prolonged fasting, total parenteral nutrition, multiple transfusion, or sepsis[3-7].

The disease can also occur in outpatients without evidence of acute illness or trauma. Specific infections [Table 1] can be responsible and should be managed appropriately.

TABLE 1: Infections associated with acute acalculous cholecystitis[8-11]

Virus	Hepatitis A, Cytomegalovirus, Ebstein Barr virus, dengue,
Bacteria	Salmonella species, Vibro cholerae, Escherichia coli,
	Klebsiella specis, Staphylococcus species, Serratia marcesens,
	Leptospira species
	Mycobacterium avium-intracellulare
Parasites	Isospora bellis, Microsporidia, Cryptosporidium, Malaria
Fungus	Candida species

Dengue related acalculous cholecystitis

Acalculous cholecystitis is a known associated condition in dengue and dengue haemorrhagic fever. In a series of 131 patients with dengue reported from Taiwan, 10 patients (7.63%) had acute acalculous cholecytitis.[12] Two of the patients underwent cholecystectomy and one underwent percutaneous transhepatic gallbladder drainage due to poor resolution. In another series of 27 dengue patients presenting with dengue and abdominal pain in a Northern Indian tertiary care hospital, 17 were found to have acalculus cholecystitis[13].

Pathophysiology of acute acalculous cholecystitis

Acute acalculous cholecystitis is due to gall bladder dyskinesia and ischemia, which result in local inflammation in the gall bladder wall. In severe cases, necrosis of the gall bladder tissue and perforation occurs[2]. In Dengue fever, the main pathophysiological changes could be due to increased vascular permeability causing plasma leakage and serous effusion with high protein content that causes thickening of gall bladder wall and gall bladder stasis[12].

Disease course of Dengue related acute acalculous cholecystitis

Acute calculous cholecystitis requires prompt management, especially in the critically ill. After blood cultures are obtained, an intravenous broad-spectrum antibiotic that covers common enteric pathogens including anaerobes should be started. Definitive therapy is cholecystectomy with drainage of any associated abscess. However, this is not the case for dengue related AAC. Dengue related AAC is usually self-limiting, and the gall bladder wall thickness usually returns to normal. Cholecystectomy is usually not advised in dengue patient unlike other subsets of patients. It is however indicated if there is rapid progression to gangrene and perforation[14]. Two dengue patients had been reported locally to present with dengue hemorrhagic fever and acute acalculous cholecystitis. Both patients recovered fully, needing only supportive treatment of platelet and fresh frozen plasma transfusions[15].

CONCLUSIONS

Acalculous cholecystitis can occur in dengue fever and dengue haemorrhagic fever. Treatment is conservative unless there is no resolution. In the context of Singapore with dengue fever and Dengue Haemorrhagic fever being highly endemic, there is a need to be familiar with the atypical presentation of this common disease.

REFERENCES

1. Weekly Infectious Disease Bulletin, Epidemiological week 38.Ministry of Health Singapore.http://www.moh.gov.sg/mohcorp/statisticsweeklybulletins.aspx.

2. Afdhal NH, Angulo P, Bonis PAL.UpToDate (www.uptodate.com). Acalculous cholecystitis.

3. Kallifas S, Ziegler DW, Flancbaum L, Choban PS. Acute acalculous cholecystitis: incidence, risk factors, diagnosis and outcome. Am Surg 1998; 64:471-5.

4. Cornwall EA, Rodriguez A, Mirvis SE, Shorr RM. Acute acalculous cholecystitis in critically injured patients. Ann Surg 1989; 210:52-5.

5. McClain T, Gilmore BT, Peetz M. Laparoscopic cholecystectomy in the treatment of acalculous cholecystitis in patients after thermal injury. J Burn Care Rehabil 1997; 18:141-6.

6. Hatada T, Kobayashi H, Tanigawa A, Fujiwara Y, Hanada Y, Yamamura T. Acute acalculous cholecystitis in patient on total parenteral nutrition: care report and review of Japanese literature. Hepatogastroenterology 1999; 46:2208-11.

7. McChesney JA, Northup PG, Bickston SJ. Acute acalculous cholecytitis associated with systemic sepsis and visceral arterial hypoperfusion: a case series and review of pathophysiology. DigDis Sci 2003; 48:1960-7.

8. Chong VH, Goh SK. Leptospirosis Presenting as Acute Acalculous cholecystitis and Pancreatitis. Ann Acad Med Singapore. 2007 Mar; 36(3): 215-6.

9. Thompson C, Salvato P, Stroud S. MAIC infection in acalculous cholecystitis. Int Conf AIDS. 1994 Aug 7-12; 10:178 (abstract no. PB0727).

10. Lagona E, Sharifi F, Voutsioti A, Mavri A, Markouri M. Epstein-Barr Virus Infectious Mononucleosis Associated with Acute Acalculous Cholecystitis. Infection 2007; 35:118-9.

11. Yombi JC, Meuris CM, Van Gompel AM, Ben Younes M, Vandercam BC. Acalculous cholecystitis in a patient with Plasmodium falciparum infection:a case report and literature review. J Travel Med. 2006 May-Jun; 13(3): 178-80.

12. Wu KL, Changchien CS, Kuo CM. Dengue fever with acute acalculous cholecystitis. Am J Trop Med Hyg 2003 Jun; 68(6):657-60.

13. Sharma N, Mahi S, Bhalla A, Singh V. Dengue fever related acalculous cholecystitis in a North Indian tertiary care hospital. J Gastroenterol Hepatol 2006 Apr; 21(4):644-7.

14. Gulati S, Maheshwari A. Atypical manifestation of dengue. Tropical medicine and International Health 2007 Sept; 12(9): 1087-95.

15. Jaufeerally FR, Surrun SK, Chang PE. Acute acalculous cholecystitis in dengue hemorrhagic fever. Indian J Med Sci 2007; 61:613-4.