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Massive pericardial effusion in a patient during the recovery phase of dengue haemorrhagic fever

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Introduction

Dengue haemorrhagic fever (DHF) is characterized by plasma leakage into peritoneal and pleural spaces. However few clinically significant pericardial effusions were reported in DHF (1, 2). Herein we report pericardial effusion with cardiac tamponade during the DHF recovery phase.

Case Report

A 30-year old male presented with fever, arthralgia, myalgia and headache for 4 days.On admission he was febrile (100.4 F).His pulse was 72/min with blood pressure (BP) of 120/80 mmHg. His heart sounds were audible in normal intensity with no murmurs. Rest of the clinical examination was unremarkable. Investigations on the 4th day showed haemoglobin 14.6 g/dL, haematocrit 45, platelet count 65,000/uL and white cell count (WBC) 4,200/uL. On the 5th day ultrasound scan revealed free fluid in the Morrison's pouch for which appropriate fluid therapy was instituted. The lowest platelet count (12,000/uL) and the highest haematocrit (58%) were recorded on the 7thday. Repeat ultrasound scan showed ascites, pleural effusions but no pericardial effusion. He was discharged on the following day with no fever, normal appetite, haematocrit and rising platelets. Five days after discharge he developed fever, dry cough, pleuritic type of chest pain, progressive dyspnea and was admitted to hospital next day.

He was febrile (101 ^{0}F), on readmission with regular pulse at a rate of 108/min, BP was 110/70 mmHg and jugular venous pulse (JVP) was elevated. He had soft heart sounds with no murmurs. Left lung base was dull to percussion with absent breath sounds. Electrocardiogram revealed diffuse ST elevations with low voltage complexes. Chest radiograph revealed cardiomegaly with well demarcated cardiac borders without radiographic evidence of pulmonary oedema or pleural effusions. Transthoracic echocardiogram revealed a moderate pericardial effusion. Other investigations revealed a platelet count of 312,000/uL, haemoglobin - 13.2 g/dL, WBC of 10,000/uL with neutrophil of 83%. Dengue antibodies (IgM and IgG) were positive. C reactive protein was 96ng/dL and ESR was 90 mm in 1st hour. He was commenced on intravenous ceftriaxone 1g bd and clarithromycin 500 mg bd. On the following day he developed increasing dyspnea, tachycardia, elevated JVP and hypotension suggestive of cardiac tamponade. Chest radiograph (Figure 1) and repeat echocardiogram showed a massive pericardial effusion. He underwent pericardiocentesis where 300 mL of straw colour fluid was removed. Pericardial fluid was an exudate with no atypical cells, negative gram stain, bacterial culture and acid fast bacilli. Adenosine deaminase level was normal. He was commenced on ibuprofen 400 mg tid and prednisolone 30 mg daily for a presumed Dressler's syndrome (DS). Antibiotics were omitted after 3 days once blood, urine and pericardial fluid cultures became negative. Echocardiogram performed 7 days after pericardiocentesis showed mild pericardial effusion. He was discharged on 10th day after readmission (day 24 of illness)when chest radiograph (Figure 2) and echocardiogram revealed resolution of the pericardial effusion. His inflammatory markers improved and were normal at two weeks after discharge. Prednisolone and ibuprofen were tailed off over next 2 weeks as the repeat echocardiogram showed full resolution of the pericardial effusion. One month later he was apparently well and echocardiographic examination did not reveal recurrence of pericardial effusion.

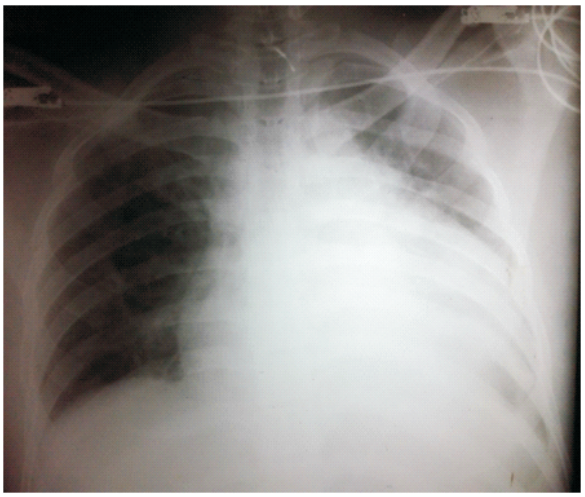

Figure 1: Chest radiograph on day 15 of the illness showing evidence of a massive pericardial effusion

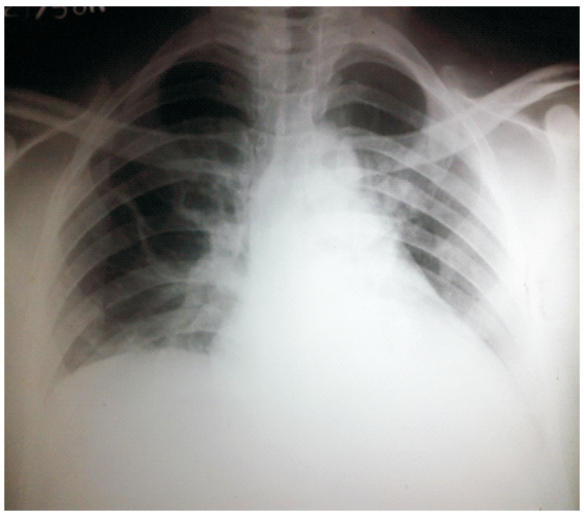

Figure 2: Chest radiograph after 10 days of treatment (day 25 of illness) showing resolution of pericardial effusion

Discussion

There were several cases of clinically significant pericardial effusions occurring during the critical phase of DHF (1, 2). The current case describes a pericardial effusion leading to cardiac tamponade in DHF during the recovery phase.

This patient who was discharged after recovering from DHF was readmitted with fever and progressive dyspnea. He had no evidence of hospital acquired bacterial infections (urosepsis, thrombophlebitis and endocarditis) which are known to occur in the recovery phase of dengue fever (3, 4).

He was commenced on antibiotics on the day 1 of second admission for suspected pyogenic pericarditis with effusion. Antibiotics were stopped 3 days later after ruling out a bacterial infection (negative blood culture, pericardial fluid gram stain and culture). He responded to steroids and NSAIDs with resolution of fever, chest pain and improvement of inflammatory markers. Pericardial fluid aspirate was an exudate with no malignant cells, negative gram stain, bacterial culture, normal ADA levels and negative culture for tuberculosis. Antinuclear antibody was negative making systemic lupus erythematosus unlikely which could have been precipitated with dengue (5). With the exclusion of other possibilities and response to anti-inflammatory therapy Dressler's syndrome was the most likely cause of pericardial effusion.

DS is an inflammatory pericarditis with or without pericardial effusion that occur secondary to injury (in the form of myocardial infarction, surgery, trauma or viral infections) to myocardium or pericardium (6, 7). The exact aetiology of DS remains unclear while autoimmune responses and viral infections have been considered as causes (7, 8).

Our case highlights the importance of considering DS as a differential diagnosis for pericardial effusion in a patient presenting with fever and dyspnea during the recovery phase of DHF.

Conclusion

Clinically significant pericardial effusions could occur with DHF not only during the critical phase but also during the recovery phase. High degree of suspicion is required for early detection and prompt treatment is necessary to reduce mortality.

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