Acute staphylococcal endocarditis following dengue haemorrhagic fever

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Introduction

Dengue fever is a leading health problem in Sri Lanka. Clinical presentation can vary from a simple flu like illness to hemorrhagic fever with shock. Available statistics show that 12144 suspected dengue cases and 71 deaths have been reported in Sri Lanka from July to September 2010[1]. Most people who develop dengue fever recover completely within 2 weeks. Apart from the complications such as haemorrhage and dengue shock syndrome, concurrent bacteraemia in patients with dengue fever is a very rare complication[2].

We report a patient initially diagnosed with dengue haemorrhagic fever, who subsequently developed a disseminated Methicillin Resistant *Staphylococcus aureus* (MRSA) infection and endocarditis.

Case report

A 20-year-old previously healthy male, presented with two days history of fever, vomiting and headache and reduced urine output. On examination he was febrile and dehydrated. His pulse rate was 98/minute and regular. Blood pressure was 80/50 mmHg with cold peripheries. Examination of cardiovascular system was normal; there were no murmurs and the ECG normal. Respiratory system and abdomen were normal.

Following initial treatment with intravenous and oral fluid, his blood pressure and urine output improved within 24 hours. He was subsequently treated as probable dengue haemorragic fever with intravenous and oral fluids, antipyretics and antiemetic.

Over the next five days the fever persisted while the white cells and platelets gradually decreased to a white cell count of 1700/µL with neutrophils 1054/µL and platelet count of 45,000/µL. At this time both dengue IgM and IgG antibodies were found to be positive by immunochromatografic studies.

After five days in hospital, patient's fever improved. However the following day the patient developed a high fever. Two days later, the white cell count increased to 11,000/µL. His blood and sputum was taken for culture and intravenous ceftriaxone was started.

After two days of antibiotic, fever still persisted and on examination there were bilateral diffuse crepitations in the lungs. Heart rate was 120/minute, blood pressure was 100/70 mmHg and heart sounds were normal but there was a new soft (grade 1) systolic murmur in the tricuspid area and the left knee was swollen and tender.

His chest ragiograph and CT of the chest showed two large opacities and multiple small rounded opacities distributed throughout both lungs. Blood and sputum culture showed a growth of methicillin-resistant *Staphylococcus aureus*. His ESR was 75mm/1st hour and white cell count was 12400/µL with 82% neutrophils. A 2D Echocardiogram revealed mild tricuspid regurgitation and 1 cm vegetation attached to the tricuspid valve. The knee joint aspirate showed high LDH level of 14844 U/L and culture was positive for MRSA.

Treatment for infective endocarditis was started according to the antibiotic sensitivity with intravenous vancomycin 1 g two times a day and intravenous clindamycin 600 mg three times a day for a period of 28 days. Progress was monitored with ESR and CRP levels periodically. After one month of treatment predischarge 2D echocardiogram showed reduction in severity of tricuspid regurgitation and complete resolution of the vegetation. Subsequently the patient was discharged on oral clindamycin for 2 weeks. He was followed up in the medical clinic for 3 months during which time he was closely monitored with ESR, CRP and 2D echo for any possible recurrences and was subsequently discharged from the clinic.

Discussion

This patient with dengue haemorrhagic fever was subsequently complicated by disseminated MRSA infection with staphylococcus endocarditis and multiple lung abscesses and septic arthritis.

Several cases of *staphylococcus aureus* infection has been reported following dengue fever[2,3], however this is the first case of this rare complication of dengue

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haemorrhagic fever to be reported in Sri Lanka. Several mechanisms have been proposed regarding increased susceptibility to secondary infections following dengue fever. One of the mechanisms proposed to explain these co-infections is endothelial dysfunction. Antibodies directed against dengue virus cross reacting with endothelial cells in association with nitric oxide causes endothelial cell apoptosis and disruption of the endothelial barrier[4]. When this type of damage occurs in the endocardium, bacteraemia that could occur from breaching of skin such as cannula sites can lodge in these damaged endocardium and cause endocarditis.

In our patient increased susceptibility to *staphylococcus aureas* may also be due to the immunosuppresion caused by a low neutrophil count of 1054 /µL. Apoptosis of polymorphonuclear leukocytes and their subsequent removal from circulation is probably responsible for neutropenia[5]. Decreased in vitro proliferative response of peripheral blood mononuclear cells (PBMC) to mitogens and to several antigens during acute dengue infection has been well documented[5]. Further it is also known that plasma levels of IL-10 which is a known immunosuppressant are increased in patients with DF and DHF[5].

Staphylococcus aureus is a usual skin flora that can enter circulation through venepuncture sites, cannula sites or through any skin injury. Frequent blood tests performed in dengue fever may predispose to this infection. This mechanism may have predisposed our patient to develop bacteraemia and infection of the tricuspid valve.

This case illustrates the need to be aware of the possibility of secondary bacterial infection as a cause for recurrence of fever following initial dengue haemorrhagic fever.

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