nock syndrome

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Introduction

Dengue infection rarely can cause severe multi organ failure. Liver failure or central nervous system dysfunction or both could rarely dominate the clinical picture in patients with acute dengue virus infection.

Case report

A 2 ½ year old boy presented with fever for one day and generalized convulsions. He had a past history of febrile convulsions. On admission, his white blood cell count (WBC) was $7x10^9$/L, platelet count $260x10^9$/L and haematocrit (HCT) 37%.

On day 4 of the illness, repeat WBC was $5.8x10^9$/L, platelet count $57x10^9$/L and HCT 40%. Dengue fever was suspected and he was closely monitored. On the same night his pulse volume became low and he had tachycardia. Blood was taken for investigations and crystalloid and colloid boluses were given. The investigation results were as follows: platelet count $24x10^9$/L, HCT 47%, aspartate amino transferase (AST) 1600 u/l, alanine amino transferase (ALT) 687 u/l. There was no history of paracetamol overdose.

On day 5, platelet count dropped to $10x10^9$/L and HCT rose to 53%. He was given repeated fluid boluses depending on HCT. Gradually he became drowsy, irritable, restless, and acquired a decerebrate posture. He was not dyspnoeic. The chest x-ray showed no effusion. By this time liver and central nervous system dysfunction was the prominent feature of his dengue infection. Child was electively intubated and ventilated.

On day 6 he developed bleeding from cannula sites, nasogastric tube and endotracheal tube. His activated partial thromboplastin time (APTT) was 48.1 seconds, prothrombin time (PT) 29.1 seconds and international normalized ratio (INR) 2.2. Blood picture was suggestive of disseminated intravascular coagulation (DIC) and he required blood products. On day 7 dengue IgM became positive.

On day 8 he became anuric for more than 6 hours and was started on peritoneal dialysis (PD). AST was 1800 U/L, ALT 3000 U/L, serum creatinine 230 µmol/L, blood urea 29.6 µmol/L and serum potassium 6.2 mmol/L.

On day 9, despite ventilation, child began to desaturate. Chest x-ray showed bilateral pulmonary opacities. Factor VII was given and ventilation was continued with high settings.

With fluid support, supportive measures and adequate antibiotic cover patient gradually improved. On day 17 he was extubated. However, the renal functions remained impaired. On Day 18 we observed impaired voluntary motor functions together with involuntary dyskinetic movements of face and limbs. Furthermore, he was not following objects and there was no eye contact. An urgent computer tomography film showed cerebellar oedema, cerebellar infarction and dilated ventricles (Figure 1).

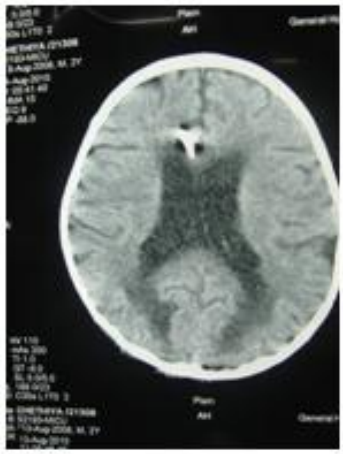

Figure 1 *CT scan of child*

The neurologist attributed the abnormal movements to hypoxic insult and started child on phenobarbitone and benzhexol. The eye surgeon opined that the poor eye contact was probably due to cortical blindness

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By day 22 the liver function improved. Dialysis was stopped by day 26 with normal renal function. Over the next 20 days abnormal movements gradually reduced. Surprisingly he started to focus but was unable to recognize the surroundings. His EVD was removed.

On day 44 he was discharged home. On discharge he could use his upper limbs but was unable to stand and walk. With repeated reviews, regular physiotherapy, speech therapy and other rehabilitation measures, now six months after that devastating illness this 3 year old boy is able to walk, see, hear and even speak without any cognitive impairment.

Discussion

Dengue has a wide spectrum of clinical presentation. While most patients recover following a self-limiting non-severe clinical course, a small proportion progress to severe disease. The group progressing from non-severe to severe disease is difficult to define[1].

The pathophysiology behind the complications is persistent shock, hypoxia, acidosis and DIC. Direct action of the virus and stimulation of T cells, macrophages and cytokines increases the capillary permeability causing hypovolaemia[2].

Usually liver failure is caused by prolonged hypotension rather than direct viral effect. Haematocrit rise by 40% of normal will cause ischaemia of liver and kidneys. This precipitates acute liver and renal failure[3,4].

Involvement of the central nervous system has always been thought to be secondary to vasculitis with resultant fluid extravasation, cerebral oedema, hypoperfusion, liver failure and/or renal failure. Thus the condition has been referred to as dengue encephalopathy. Encephalitis or direct involvement of the brain by the virus was thought to be unlikely[5].

Most case series on dengue encephalitis suggest that patients with the disease have a higher tendency to develop DHF/DSS. The neurological recovery was complete in most of these patients, although they had increased incidence of DHF/DSS. The mortality in cases of dengue encephalitis is increased, not due to the encephalitis per se, which usually has a benign course, but due to the increased incidence of DHF/DSS[6].

In this particular case, liver failure and central nervous system dysfunction dominated the clinical picture. The critical condition of this particular child did not permit us to confirm the diagnosis of dengue encephalitis by cerebrospinal fluid microscopy or electroencephalography. Nevertheless neurological symptoms appeared early in the course of illness coinciding with viraemic phase and complete neurological recovery of this child suggests dengue encephalitis.

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