CASE REPORT

Transient Blindness in A Child with Dengue Shock Syndrome

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ABSTRACT Dengue hemorrhagic fever (DHF) is characterized by acute fever associated with haemorrhagic diathesis and tendency to develop fatal shock (dengue shock syndrome). Signs and symptoms of DHF are generally secondary to plasma leakage and hemorrhage. Dengue shock syndrome (DSS) is a severe clinical manifestation of DHF usually as a consequence of severe plasma leakage. During the last 5 years, there have been reports of DHF patients with unusual manifestations, including some patients with central nervous system (CNS) involvement. We report a 5 year old DHF patient who experienced transient blindness, which was the first case found in Soetomo Hospital, Surabaya. **[Paediatr Indones 1997; 37: 132-136]**

Introduction

Dengue shock syndrome (DSS) is a severe clinical manifestation of dengue haemorrhagic fever (DHF). Symptoms and signs of organ involvement in DHF are usually secondary to plasma leakage, which in severe cases may result in shock which may be prolonged which causing anoxia of organs and hemorrhage.[1,2] During the last 5 years, some DHF patients with unusual manifestations, including central nervous system (CNS) involvement have been reported. Some of them showed associated hepatic dysfunction, renal failure, or ocular involvement. We report a patient with DSS who experienced transient blindness admitted to Dr. Soetomo hospital. Transient blindness or *amaurosis fugax* is a reversible deficit in visual function that lasts less than 30 minutes. The principal mechanism for transient blindness is ischemia.[3]

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Case Report

A 5-year old boy was referred by Suaka Insan Hospital, Banjarmasin, to the Department of Child Health of Dr. Soetomo Hospital on November 7, 1996 with the diagnosis of DSS. The illness started with high fever, headache, and vomiting. On the third day of the illness his fever persisted, so that the patient was admitted to Suaka Insan Hospital. He then developed epigastric pain, hematemesis, followed by decreased blood prressure, rapid pulse, and cold clammy skin and restlessness, which were consistent with DSS. He was given intravenous fluid, antibiotics, and corticosteroids. The fluids given were plasma 450 ml, whole blood 150 ml and dextrose 5% in lactated Ringer's solution 700 ml. Because of worsening of the condition, the patient was consulted to Dr. Soetomo Hospital. Diuretic was given to prevent overload.

Physical examination on admission revealed a sick child with the body weight of 25 kg. The blood pressure was 120/80 mmHg, the pulse rate was 76 x/minute. There was dyspnea; the respiratory rate was 40 x/minute and the temperature was 37,6°C. There was no anemia, jaundice, or cyanosis. The heart was normal. There was decrement of breath sound on the right chest. There was ascites. The liver was palpable 2 cm below the right costal margin. The spleen was not palpable. There were petechiae on the extremities and the extremities were warm. Serial laboratory examinations performed in Banjarmasin showed increased hemoglobin concentration (from 12.4 to 18.5 g/dl) and packed cell volume (from 38 to 55), and decreasing platelet count (from 137,000 to 32,000). In Soetomo Hospital, hemoglobin and PCV showed some improvement from the 5th to the 10th day of illness, but the platelet count remained low (less than 60,000/μl).

Serological test showed positive for IgM on both in November 8 and November 13, while IgG showed negative on November 8 and positive on November 13. The serum electrolytes were normal. Renal function test was normal. Fibrin degradation product (FDP) examination was planned to confirm a suspected DIC; unfortunately due to technical reason this examiation could not be performed. Chest x-ray revealed a normal heart and a right pleural effusion.

The diagnosis of post-DSS was established. The patient was given intravenous fluid therapy of dextrose 5% in lactated Ringer's solution, antibiotics, and platelet transfusion. On the 3rd day of hospitalization he suddenly screamed and was panic. His arms touched the wall, because he could not see anything. This blindness lasted for about half an hour. However, after one hour, he was calm and regained his sight. Physical examination revealed the blood pressure of 110/80 mmHg; and the pulse rate 104 x/minute. The respiratory rate was 28 x/minute and the temperature was 37.7°C. The heart and lungs were normal. The liver was palpable 2 cm and the spleen was not palpable. The extremities were normal.

Eye examination showed that the pupils were isocoric with the diameter of 4 mm. Light reflexes were normal and the conjunctiva did not show hyperemia. Based on

these finding, a transient blindness was suspected. We consulted him to the ophthalmologist who found that the anterior and posterior segments of the eyes were normal. The patient was advised for follow-up at the out patient clinic of ophthalmology.

On the 5th day of hospitalization intravenous fluid drip was discontinued and eye examination by ophthalmoscopy revealed normal vision, with normal anterior and posterior segments of both eyes. On the 7th day of hospitalization (12th day of illness), the patient was discharged in a good condition.

Discussion

This patient was referred with the diagnosis of DSS. Clinical as well as laboratory fingngs met the criteria of the World Health Organization (WHO).[1,2,4,5,11] It is known that prolonged shock is often complicated by metabolic acidosis and severe bleeding, which indicates a poor prognosis. However, if the patient is appropriately treated before irreversible shock has developed, rapid recovery is the rule.[1,4,7]

The high hematocrit found in patient with shock suggested that shock is due to hemoconcentration and decreased plasma volume as a consequence of leakage of plasma into extravascular spaces through the damaged capillaries. The supporting evidence for this is that a considerable amount of fluid accumulates in the serous cavities e.g. peritoneal, pleural and pericardial cavities.[4] In this case, there were ascites and right pleural effusion. The evaluation of fluid during hospitalization in Suaka Insan Hospital showed that there was apparently a fluid overload of 1000 ml.

In this case, immunoglobulin studies performed on the fifth day of illness showed the IgM component while not untill the eleventh day of illness did the IgG show a positive result. Consequently it was a primary infection, in which IgM usually appears on the fifth day and increases in 1-3 weeks, lasting for 60-90 days. IgG appears on the fourteenth day in primary infection and on the second day in secondary infection and can be detected during one's life. The serological diagnostic of primary dengue infection can only be examined after the 5th day but the secondary infection should be examined as soon as possible because of the quick increase of IgG.[9]

Previous studies in Indonesia proved that DEN-3 was associated with more severe clinical manifestations and fatal cases.[4,5] In our case the virus isolation was not done, so we could not assure whether DEN-3 was the cause of the disease that consequently played a major role in producing severe dengue infection.

The above mentioned definition makes transient blindness a subtype of transient ischemic attack (TIA), a reversible focal neurologic deficit of 24 hours duration or less. In fact the principal mechanism for transient blindness is ischemia. The other accepted mechanism, believed to be less common, is epileptic discharge. Seizures within the visual cortex are caused by neoplasm, arteriovenous malformation, meningoencephalitis, ischemia, or trauma and typically have an excitatory component as

well. The patient experience hallucinations, usually of unformed images, at some time during the abnormal discharge. The hallucination usually consists of flickering lights that do not move across the visual field as they do in migraine.

Ischemia produces transient blindness by vascular occlusion or reduced blood flow through non occluded vessels, including hypotension and hyperviscosity states.[3,12,13] Transient blindness may be caused by many conditions, both local and systemic. Local causes of transient blindness mostly caused by ocular causes, i.e., blepharospasm, corneal abnormality, recurrent hyphema, vitreus debris, tumor, and others. While systemic causes of transient blindness includes emboli, valvular disease, endocarditis, mural thrombi, vasculitis, hyperviscosity / hypercoagulability.

In the workup of patient with transient blindness it is suggested that the examiner proceeds as follows:[3]

1. Determine if the scintillation of migraine are present.
2. If scintillation are not present, differentiate embolic from non-embolic causes of transient blindness and distinguish monocular from binocular transient blindness. If non-embolic cause is found, refer to appropriate specialist or treat.
3. If an embolic cause is suggested or cannot be excluded, refer for cardiac evaluation. This step is necessary to rule out a cardiac source for emboli.
4. If cardiac evaluation is negative for an embolic source, patients suitable for carotid endarterectomy who have signs of arteriosclerosis and monocular transient blindness should have transarterial cerebral angiography to rule out an operable extracranial carotid lesion.

A finding of reduced ophthalmic artery pressure as determined by ophthalmodynamometry, bruits over the internal carotid artery and angiography helps to confirm the diagnosis.[12] In this case, on the 3rd day of hospitalization, the patient complained of transient blindness for half an hour. Unfortunately it took time for the ophthalmologist to be at the patient's side so that when he examined the patient the symptoms had disappeared, and ophthalmoscopic examination were within normal limits as well. It would not be possible to know the exact mechanism of the transient blindness since proper examination by way of ophthalmodynamometry and angiography were not performed. Besides, could there have been specific factors/mediators short acting activities which had played a role in this transient blindness?

The therapy of transient blindness is a varying combination of aspirin, dipyridamole, CO_2-O_2 mixture, paracentesis and ocular massage, and intravenous acetazolamide. After 24 hours, the clinical picture is usually irreversible, though exceptions to this rule have been reported.[12,13]

In this case the patient was not given therapy toward the blindness, because the ophthalmologic examination did not show any abnormalities and yet the patient recovered completely.

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