BRAIN STEM INFARCT
(A Case Report)

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Introduction

Brain stem dysfunction can be transient. The recuperative powers of the brain sometimes can be astounding and individual patients, whom uninformed physicians might give up for dead, sometimes make unexpectedly good recovery. It is important to know when to continue to fight to preserve life than to be willing to diagnose death.

CASE REPORT

A 27-year-old male medical officer was admitted to Military Hospital Kirkee with third degree haemorrhoids. He was a known case of essential hypertension being treated with tab enalapril 5 mg bd. His blood pressure recorded at admission was found to be within normal limits. He was taken up for surgery in ASA grade II. Lord's dilatation and haemorrhoidectomy were done under general anaesthesia. The surgery and anaesthesia were uneventful.

On the 5th post operative day, while still in hospital, he was found to be unconscious at about 0645 hours. He was given forced diuresis as poisoning was suspected. His condition further deteriorated and he developed respiratory arrest. He was intubated, put on artificial ventilation and was brought to Command Hospital (SC), Pune. While being shifted to Command Hospital, the patient had a seizure. At the time of admission he was deeply comatose. Blood pressure was maintained with dopamine drip at the rate of 8 µg kg/min. Pupils were small and non-reacting. Deep tendon jerks were absent. Both plantars' were mute. There was no ocular movement to head turning or cold caloric test. There was no spontaneous respiration. These signs indicated absence of all brain stem reflexes [1]. To assess the cause of the coma a computerized tomography (CT) scan was done. During CT, ventilation was controlled with Bains coaxial circuit and Boyles machine. CT scan revealed a hypodense area in midbrain (Fig. 1) suggestive of infarct [2].

Though there was no behavioural or reflex response to noxious stimuli and brain stem functions had apparently ceased, we decided to give a trail of therapy and observe him. The therapy included hyperventilation to maintain an end tidal CO_2 around 30 mmHg, and diuretic therapy in the form of intravenous mannitol 0.5 g/kg 8 hourly with serum osmolatiy monitoring. Injection dilantin sodium was given to prevent seizure activity and steroids were given for free radical scavenging and membrane stabilizing effects. His blood pressure was maintained with inotropic support (dopamine drip) to maintain cerebral perfusion.

With the above regimen the patient started improving. Next day morning his blood pressure could be maintained without dopamine support. In 36 hours he started moving his limbs to verbal commands. He was gradually weaned off the respirator by changing the ventilatory modes from controlled to synchronized intermittent mandatory ventilation and later to assist support breathing. He was extubated after 48 hours. Patient was by then fully conscious and oriented, his cerebral and brain stem functions returned to normal and in a weeks time was discharged from the hospital. Repeat CT scan at the time of discharge showed marked reduction in the size of lesion.

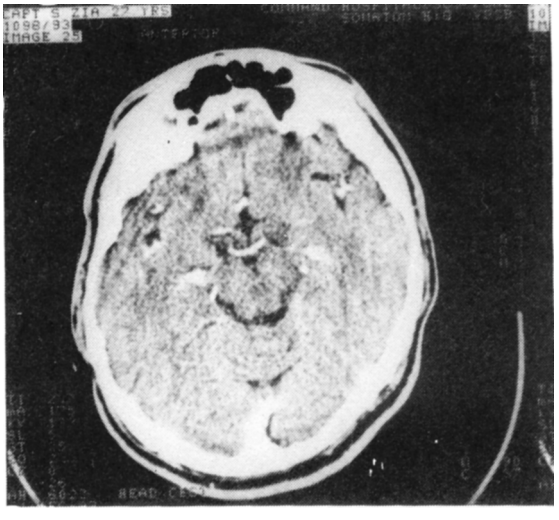

Fig. 1 :

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Discussion

The sudden onset, absence of preceding tangible cause, presentation as coma with pupillary abnormalities, disorder of ocular movement, and quadriplegia indicates occlusion or severe stenosis of basilar artery. In the majority of these cases death takes place in 2 days to 5 weeks [3,4]. Since the patient recovered completely, 4 vessel angiography was not considered necessary, as this was only of academic interest and would not have made any change therapeutically. Global and regional brain injury require different treatment modalities. In this case treatment included control of intracranial pressure by hyperventilation, diuretic therapy, oxygenation, and corticosteriods (though controversial) for cerebral protection (prevention of membrane phospholipid break down, reduction of oedema formation, and scavenging oxygen free radicals). Phenytoin was used to decrease seizure activity secondary to incidental hypoxic-ischaemic encephalopathy and to reduce cerebral metabolic oxygen requirement. Phenytoin is known to reduce cerebral oxygen requirement by 40 to 60 per cent, decrease cerebral lactate production and increase cerebral levels of glucose. Vasodilatation has also been reported. Sympathomimetically controlled blood pressure to help improve cerebral blood flow may have also helped in resuscitating him and protecting the brain [5].

The diagnosis of brain stem infarct was suspected in this case on the basis of sudden onset of symptoms in a known hypertensive supported by the hypodensity seen on CT scan. History of toxins and poisoning was not available and demyelination would have not produced such an explosive onset.

The aims of reporting this case were (a) to highlight that the recuperative powers of the brain can be sometimes surprising and measures to resuscitate the brain may reverse the deterioration and (b) that patients presenting in this fashion, with apparent cessation of cerebral and brain stem function, may be wrongly diagnosed as brain-dead unless an adequate trial of therapy is performed. This mistake may be avoided by resorting to an electroencephalogram and thus prevent the grave and unfortunate error of taking organs from such a patient for transplantation [6,7].

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