Cortical blindness in preeclampsia (case report)

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Bilateral infarction in the distal posterior cerebral arteries produces cortical blindness (blindness with preserved pupillary reaction). The patient is often unaware of the blindness or may even deny it (Anton's syndrome, which is a form of anosognosia due to compromize of the visual association centre)[1,2]. Tiny islands of vision may persist, and the patient may report that vision fluctuates as images are captured in the preserved portions[1]. Preeclampsia is associated with abnormalities of cerebral circulatory autoregulation, which increase the risk of stroke at near normal blood pressures[3]. During focal brain ischemia, a gradation in brain perfusion exists such that a core of tissue is infarcted within minutes but a shell of surrounding tissue is only marginally ischemic. This *ischemic penumbra* may progress to infarction within minutes to hours[4].

CASE REPORT

A 40-years old female was admitted to the medical department at Al-Salam general hospital in Mosul, with bilateral blindness and headache for one week. One week earlier she was admitted to the obstetric and gynecology department at the same hospital with drowsiness, headache, epigastric pain, blurring of vision and a history of loss of consciousness for 30 minutes on the day of admission. She also suffered from accidental hemorrhage on the same day. She was gravida 13 and para 10 and had a history of preeclampsia in a previous pregnancy which was managed conservatively. Her BP was 140/80 and the fetal heart sounds were absent. She was diagnosed to have preeclampsia ,transfused with two units of blood and underwent an emergency Caesarean section; a dead male fetus was delivered. .Upon recovery from anaesthesia she complained of total blindness in both eyes, however nothing was written about that in her follow up notes and she was discharged home four days after the operation.

On examination, the patient was fully conscious, oriented and anaemic. There was no neck stiffness. Her B.P was 130/70, pulse 100/min.and respiratory rate 18/min. There was no light perception in both eyes and the pupils were dilated and reacting to light (direct and consensual). There were no other focal neurological signs and the plantars were flexor. Ophthalmoscopic examination revealed normal optic discs and normal retinae. Investigations showed a hemoglobin of 6.1 gm/dl., Hct. 19%, WBC *15,560/c.mm* (granulocytes 81.7%) and platelets 351000/cmm.; the RBCs. were microcytic and hypochromic. Blood urea was 24 mg/dl., random blood sugar 108/dl., serum sodium 145 mEq./dl, and serum potassium 3.8 mEq./dl.

Her MRI (Figure 1), obtained nine days after onset of symptoms, showed acute bilateral infarction of the occipital lobes, more evident on the left side. There were no signs of increased intracranial pressure.

The patient was given intravenous infusion of 200 ml. of 20% mannitol solution every 12 hours with dexamethasone 4 mg. i.v. 6- hourly for two days and upon receiving the MRI report both drugs were suspended and aspirin 100 mg./day was added together with atenolol 50 mg./day (B.P fluctuated between 140/80 a170/90). She was also transfused with three units of blood.

Six days after admission the patient started to have light perception and the intensity of headache decreased. On the next day visual improvement continued and she was able to recognize persons and count fingers at a distance of three meters. She was discharged home seven days after admission. Ten days later she regained good visual acuity and could look after herself. She was prescribed ferrous sulphate tab.tds for four weeks and maintained on aspirin 100 mg./day; atenolol was omitted one month after discharge. When seen two months later, her B.P was 120/85, she could recognize small objects and her colour perception was normal.

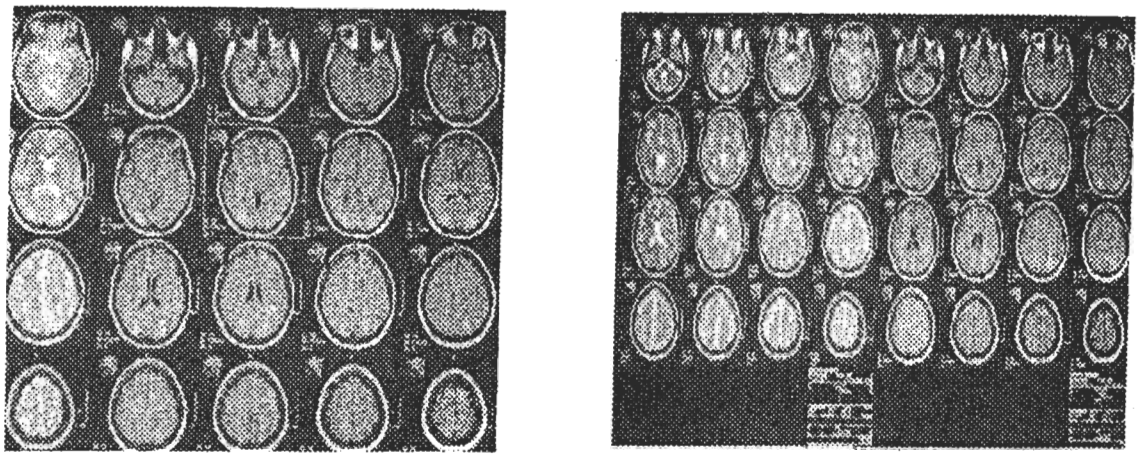

Figure (1)

DISCUSSION

In this patient, reperfusion of the ischemic penumbra might have accounted for the improvement in vision. Initially, the blurring of vision could have been due to cerebral edema as a result of the preeclampsia[5-8], due to the initial phase of infarction of the occipital lobes which progressed later on to a completed stroke, or due to decrease of cerebral perfusion pressure as a result of the accidental hemorrhage and possible cerebral edema[5,6,9,10]. In preeclampsia, cortical blindness could also be due to multifocal cerebral hemorrhages and early CT scans could be normal[11,12]. In this patient there was no evidence of cerebral hemorrhage in the MRI which was obtained nine days after the onset of visual symptoms. Cortical blindness could also be due to generalised endothelial vascular damage[13], or be associated with the HELLP syndrome(a disorder of pregnancy charcterised by hemolysis, elevated liver enzymes and low platelets that occurs in the setting of severe preeclampsia or eclampsia)[14]; in this patient there was no evidence of the HELLP syndrome. Patients with thrombotic thrombocytopenic purpura (TTP) may also suffer from cortical blindness[15], however there was no evidence of TTP in this patient.

Other causes of visual disturbances during pregnancy that could amount to total blindness include retinal vasospasm[16], central retinal artery occlusion[17,18], acute ischemic optic neuropathy[19], and retinal detachment[20], just to mention some. It is evident that in all such cases the blindness is not cortical in type.

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