INTRODUCTION
Sudden visual loss affecting bilateral vision, if not due to trauma, is usually the result of multiple lesions in the occipital lobes especially seen in eclamptic hypertensive encephalopathy.\(^1\) Pituitary apoplexy can also present with bilateral visual loss accompanied by severe headache and diplopia.\(^2\) Pituitary apoplexy is not infrequently seen postpartum in patients with severe postpartum haemorrhage. Postpartum bilateral blindness can be due to vascular insult involving both the bilateral middle and posterior cerebral arteries (Gerstmann’s syndrome).\(^3\)

This case report describes the occurrence of cerebral angiopathy in a lady leading to postpartum cortical blindness.

CASE REPORT
A 30-year-old postpartum patient with an uneventful prenatal course started complaining of blurred vision, severe headache and bilateral pedal oedema two days preceding labour. Her blood pressure was normal throughout pregnancy and urine examination showed no proteinuria. Pregnancy was terminated due to fetal distress by lower segment caesarean section under general anesthesia. She developed blindness after recovery from anesthesia. Blood pressure, pulse and respiration was normal during surgery.

One day after caesarean section, she developed a confusional state besides blindness and became very agitated and aggressive, developed irrelevant speech, neither feeding nor caring for her baby. She also developed low grade fever, insomnia and loss of apetite with bizarre behaviour and strange thoughts.

On the 7\(^{th}\) postpartum day, she was admitted to Nishtar Medical College Hospital, Multan. There was not history of medical or psychiatric problems previously, hypertension, convulsions or thrombosis in this pregnancy, or postpartum haemorrhage during or after caesarean section.

On examination, patient was delirious, not oriented in time and space with incoherent speech and not responding to verbal commands. Cardiovascular system was essentially normal. Blood pressure was 110/80 mm Hg. Clinically, she was slightly anaemic. The respiratory and gastrointestinal system was found to be normal. On ocular examination, pupillary light reflex was normal. Pupils were not dilated and fundoscopy was normal.

On neurological examination, she had impairment of higher mental functions, left sided hemiparesis, bladder incontinence, confusion and inability to distinguish right from left. There was no neck stiffness or meningeal irritation.

A plain CT scan of the brain was done immediately after admission, which revealed infarcts in the right occipito-parietal and left posterior parietal regions as shown in Figure 1. Complete blood count and ESR was normal. Urine examination was essentially normal. Urea serum electrolytes, liver function tests and coagulation profile were within normal limits.

**CASE REPORT**

A 30-years-old third gravida with previous normal pregnancies and an unremarkable prenatal course had an emergency lower segment caesarean section at a periphery hospital for failure of labour to progress. She developed bilateral cortical blindness immediately after recovery from anesthesia due to cerebral angiopathy shown by CT and MR scan as cortical infarct cerebral angiopathy, which is a rare complication of a normal pregnancy.

**Key words:** Cortical blindness. Postpartum. Cerebral angiopathy.

**ABSTRACT**

A 30-years-old third gravida with previous normal pregnancies and an unremarkable prenatal course had an emergency lower segment caesarean section at a periphery hospital for failure of labour to progress. She developed bilateral cortical blindness immediately after recovery from anesthesia due to cerebral angiopathy shown by CT and MR scan as cortical infarct cerebral angiopathy, which is a rare complication of a normal pregnancy.

**Key words:** Cortical blindness. Postpartum. Cerebral angiopathy.

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EEG showed encephalopathy. Abdomino-pelvic ultrasonography was normal. Antiphospholipid antibody was negative. A Visual Evoked Potential (VEP) was performed, which showed normal amplitude with prolonged P-100 latency. MRI of the brain was done after one week. It demonstrated sub-acute cerebral haemorrhagic infarction involving both parietal lobes.

She was put on Ceftazidime and Clindamycin with respective doses of one gram and 600 mg, 8 hourly. During the initial days, she received Acycloir 10mg/kg body weight TID for 3 days. She was also receiving dexamethosone 6 mg, 8 hourly and was tapered off from steroid therapy. TED stockings along with Heparin 5000 IU, 8 hourly were also given from day one until discharged. The patient was followed-up at monthly intervals. After 5 months, the hemiparesis improved, power increased from 1/5 to 3/5 and bladder control returned but total bilateral blindness persisted.

**DISCUSSION**

The diagnostic triad of cortical blindness consists of decreased visual acuity normal pupillary response and normal ocular fundus. This triad occurs in patients who had severe haemorrhage during delivery but in this patient there was no history of intra-partum or post-partum haemorrhage. Cortical blindness implies retrogeniculate lesion with anterior visual pathway remaining intact. Since the visual cortex is supplied by both the middle cerebral artery and the posterior cerebral artery, so a bilateral total cortical blindness signifies a vascular insult involving both the middle and posterior cerebral artery. A total blindness involving both the vessels is rare occlusion of the left posterior cerebral artery or the inferior division of the middle cerebral artery can result in infarction of the left angular gyrus, situated at the confluence of the temporal, parietal and occipital lobes, providing a host of findings. This is called Gerstmann’s syndrome. The findings include difficulty in telling right from left, naming the digits and difficulty in calculating. In any single patient, all features may appear together, or one or more may occur in isolation. This patient had difficulty of telling right from left but she could name digits.

Postpartum cortical blindness is a rare complication. It is considered to be due to the hypercoagulable state associated with pregnancy, eclampsia and severe blood loss due to either intra or postpartum haemorrhage. Although visual disturbances are common in severe pre-eclampsia, blindness, either alone or accompanying convulsions, is rare. The cause of blindness in severe pre-eclampsia is attributed to a varying degrees of retinal detachment and or occipital lobe ischaemia or infarction. Over a 14-years period, Cunningham et al. studied 15 cases of blindness due to severe pre-eclampsia/ eclampsia and all recovered vision in 4 hours to 8 days.

Visual changes such as blurred vision or scotoma are common and more typical of late onset pre-eclampsia. One study reported visual symptoms in 44% of patients with late onset pre-eclampsia. This patient had an uneventful pregnancy with only transient visual disturbances in last few days of pregnancy. Her blood pressure and urine examination was normal during pregnancy and even after 7 days of admission, but unfortunately the patient is still suffering from total bilateral blindness at 5 months after delivery. Total cortical blindness due to eclampsia was recently reported in a young woman persisting for a year after the episode with persistent hypodensities in cerebral white matter. The patient was not suffering from any hypercoagulable state as APLA was found to be negative. Aetiopathogenesis behind this dramatic clinical picture could not be found known where thrombotic or embolic predisposing factors were conspicuously absent.

**REFERENCES**