RARE CAUSE OF COMPLETE BLINDNESS : BILATERAL OCCIPITAL CORTICAL INFARCTION

(A Report on Three Cases)

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Introduction

The medical causes for sudden onset of complete blindness include central retinal artery block, occipital lobe lesion and malingering. The occipital lobe lesions are usually unilateral and result in homonymous hemianopia [1]. Very rarely the occipital lobe lesions may be bilateral and result in complete blindness due to cortical lesion.

We present here a series of three such cases seen in last thirteen years, that developed sudden onset of complete blindness due to bilateral occipital lobe infarcts. Bilateral occipital infarcts though theoretically possible but are very rarely seen in clinical practice and can only be diagnosed by any of radiodiagnostic imaging modalities like CT, MRI or angiography.

CASE REPORTS

Case 1

A 70-year-old lady developed complete loss of vision of sudden onset while she was walking. There was no loss of consciousness, no associated headache, vomiting or convulsions. She developed disorientation after the episode. She was a known hypertensive on irregular treatment. On examination, her vital signs were normal. vision was perception of light in both the eyes. Anterior segments of both the eyes showed semi dilated pupils. Posterior segments of both eyes were normal. Detailed examination of central nervous system and routine investigations did not show any significant findings. CT scan of the head revealed low attenuating lesions affecting both the occipital lobes suggesting bilateral occipital lobe infarction.

Case 2

A 65-years-old retired sepoy, a below left knee amputee, was brought to Command Hospital with complaints of generalised weakness and complete blindness. On examination he was found to be hypertensive with blood pressure recording of 170/110 mm of mercury. He had evidence of impairment of memory with confabulation. Neurological examination did not reveal any motor or

sensory deficit. He was conscious and his pupils were normal. Fundii showed grade II changes. Bilateral carotid pulsations were well felt. His EEG recording was indicative of a lesion in left temporo-parietal region with no epileptic activity. His CT scan head revealed extensive low attenuating non-enhancing lesions in both occipital lobes confirming bilateral occipital cortical infarction.

Case 3

A 57-year-old right handed school teacher was admitted at Command Hospital, Pune on 25 Oct 85 with complaints of sudden loss of vision in both the eyes followed by rapid unresponsiveness. He was a known hypertensive and diabetic of six years duration on irregular medication. On examination he was found to be febrile with neurogenic hyperventilation. His blood pressure was 180/110 mm of mercury. His pupils were pinpoint. He had signs of quadriplegia with positive Babinski sign. His CT scan head demonstrated evidence of bilateral occipital infarcts with generalised cortical atrophy.

Discussion

The most common cause of cortical blindness is occlusion of the posterior cerebral arteries (embolic or thrombus) [2,3]. Hypoxic-ischaemic encephalopathy. Progressive multifocal leucoencephalopathy, other leucodystrophies and bilateral gliomas are other causes [4]. Bilateral occipital lobe infarction though extremely rare can occur due to thrombosis or embolism affecting the vertebro basilar system of vessels usually triggered off by fall in the blood pressure. When the terminal bifurcation of basilar artery is involved the symptoms may be bilateral. Melamed and Abraham reported three similar cases [5]. In all the cases clinical and angiographic evidence of basilar artery occlusion was seen. These patients are generally in their sixties with a history of general atherosclerosis or history of vertebro-basilar transient ischaemic attacks. The onset of blindness is sudden; charac-

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Fig. 1 : CT scan of head of 1st patient
Low attenuating lesion areas in both occipital lobes without mass effect suggesting bilateral occipital infarcts

Fig. 3 : CT scan of head of 3rd patient
Bilateral low attenuating non-enhancing infarcts involving both occipital lobes.

Fig. 2 : CT scan of head of 2nd patient
Bilateral extensive infarcts denoted by low attenuating areas involving occipital lobes are seen.

teristically patients exhibit no other neurological signs. The eyes are still able to move through a full range, but optokinetic nystagmus cannot be elicited. Visual imagination and visual imaginary dreams are preserved [4]. With very rare exception no cortical po-

tential can be evoked in the occipital lobes with light flashes or pattern changes. The alpha rhythm is lost in EEG. A striking feature of patients with cortical blindness is subjective unawareness of ones disability (anosognosia). The other less common symptoms include visual hallucinations due to cortical irritation, associated apathy and mental disturbances. Rarely, oval pupils have been reported with bilateral cerebral infarctions. A large embolus in the basilar artery is usually fatal.

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