DOI:10.5958/j.2319-5886.2.4.162

International Journal of Medical Research & Health Sciences

www.ijmrhs.com Volume 2 Issue 4 Oct - Dec Coden: IJMRHS Copyright @2013 ISSN: 2319-5886

Received: 16th Aug 2013 Revised: 10th Sep 2013 Accepted: 21st Sep 2013

Case report

CORTICAL BLINDNESS FOLLOWING TWO STAB WOUNDS TO THE SCALP IN AN ADULT: A CASE REPORT

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ABSTRACT

Visual disturbances have been reported as a consequence of acute severe blood loss. They are related to hypoperfusion related watershed infarcts in the posterior visual pathway apparatus.
In this case report, we report the clinical course of a young male adult who suffered transient blindness following an assault in which he sustained deep cuts to the temporal and parietal regions of the scalp. He was managed in our hospital with copious infusion of intravenous fluid (normal saline) and subsequent transfusion of 2 pints of whole blood. Blindness persisted from less than one hour following assaut to 96 hours post admission. Blindness resolved completely by the 9th day of admission but was associated with a homonymous visual field defect.

Keywords: Blindness, cortical, stab wound, blood loss

INTRODUCTION

Sudden onset of bilateral visual loss characterized by normal pupillary responses and normal appearances of the ocular fundus is considered as blindness of cortical origin until proven otherwise.
Cortical blindness has been found to be associated with a host of clinical states and procedures (Table 1).
They are due to lesions of the geniculocalcrine visual pathway in the posterior hemisphere[1]. The geniculocalcrine visual pathway is known to occupy a delicately supplied 'vascular border zone'. Thus, most cases of cortical blindness is associated with hypo-perfusion related watershed infarcts[2-4].
We report the case of a young man who suffered severe blood loss following an assault and subsequently developed a reversible visual loss.

Table 1: Causes of Cortical Blindness

Vascular	**Trauma**
Preeclampsia/ecclampsia	Head trauma
Stroke	Cervical trauma
Hypertensive encephalopathy	**Iatrogenic**
Cerebral venous thrombosis	Post cardiac catheterization
Severe hypotensive states	Cardiac surgery
Infections	**Metabolic**
Bacteria meningitis	Hypoglycemia
Mumps encephalitis	Uraemia / Haemodialysis
Cerebral malaria	Acute intermittent porphyria
Toxic	**Miscellaneous**
Iodinated contrast agents	Status epilepticus
Metrizamide	Status asthmaticus
Amphetamine	Intracranial haemorrhage
Chemotherapy	Brain tumour
Heroin	Liver cirrhosis and encephalopathy
FK 506	

CASE REPORT

A 25 year old undergraduate of a Nigerian university had previously enjoyed good vision in both eyes until that fateful day when he was assaulted by a known assailant. He sustained matchet cuts on both sides of his scalp. He was said to have bled profusely with an estimated blood loss of about 1.5 liters. He became disoriented shortly after the assault as he lost orientation in space. There was no associated blunt trauma to the head and no bleeding from any of the craniofacial orifices.

He was rushed to the Niger Delta University Teaching Hospital (NDUTH) where he was attended to and admitted. On examination, he was noticed to be drowsy but rousable, disoriented and mentally confused. He was very pale, anicteric and not dehydrated. His cranial nerves were grossly intact.

He had deep lacerations at the left temporal region (6cm long) and at the right parietal region (10cm long). His pulse rate was 140/min, regular, poor volume. Blood pressure was 90/46mmHg (supine) and Heart sounds were normal.

Chest and Abdominal examination was normal. Ocular examination revealed a visual acuity of no light perception (NPL) in both eyes and external eye and anterior segment examination were normal. Both pupils were round and reactive. Fundoscopy revealed a pink disc (C/D ratio of 0.3), round with well defined edges in both eyes. Both retinea were flat and normal. A provisional diagnosis of cortical blindness secondary to severe hypovolaemia was made. He was resuscitated with I.V Normal Saline and thereafter transfused with 2 pints of whole blood. The two stab wounds were repaired with appropriate sutures.

Investigation ordered included urgent PCV (23%), Electrolyte and urea (normal), random blood sugar (normal) and cranial CT Scan (not done as a result of absence of equipment in our centre).By the fourth day on admission following normalization of his cardiovascular status (PR 70bpm, BP 128/80mmHg), visual recovery was noticed with a visual acuity of hand motion (H.M) in both eyes. Visual recovery became full

on the 9th day of hospital admission (6/6{OD}, 6/6+ 4 {OS}). He was then discharged for follow up at the ophthalmic outpatient.

A visual field analysis requested at this stage confirmed a homonymous hemianopia first detected clinically on the 9th day of admission.

DISCUSSION

Cortical blindness is a hallmark of posterior cerebral arterial border zone infarcts[1]. It is caused by hypoxia of the visual pathways at the territories supplied by the distal posterior cerebral arteries. The possibility that vascular factors (a limited capability of the posterior vascular system to autoregulate blood flow) and cortical tissue vulnerability to hypoxia as being responsible for the hypoxic damage has been suggested[5].

The dog electromagnetic flow metre study during hypoxia and hypercapnia has demonstrated a decrease in the compensatory dilatation response of the basilar arterial system to occlusion of the carotid system[5]. Loss of the normal protective autoregulation of blood flow was suggested as one of the pathomechanism of pre-ecclampsia / ecclampsia induced cortical blindness[6]. A sparing effect of chronic hypoxia on the anterior cerebral artery has been suggested by an ultrasonic study on fetal brain circulation[7].Thus under chronic hypoxia the frontal lobes are spared longer than the lateral and occipital lobes. It has also been shown that the parieto occipital border zone is most susceptible to haemodynamic ischaemic damage as it is the most peripheral region of the distribution of the anterior, middle and posterior cerebral circulation[8]. Posterior cerebral arterial border zone infarction has been shown to be a consequence of profound systemic hypotension and has been found to be associated with cortical blindness[9-12].

The patient in our case report developed cortical blindness following an episode of severe blood loss. Ischaemia and infarction secondary to hypoperfusion in severe systemic hypotension is known to lead to cytotoxic oedema in border zone infarcts. Radiological evidence of neuronal oedema in hypotension related watershed infarcts manifests as hypodense lesion on CT images and hyperintense lesions on T2 weighted MRI images in very specific “distal field” territories of the middle and posterior cerebral arteries[13].

Although, it was not possible to obtain a CT or MRI images in our patient, clinical evidence suggests that the above radiologic changes are not unlikely. The clinical and radiologic changes in posterior border zone infarcts may be reversible. Clinical recovery is known to precede normalization of radiologic abnormalities[14,15]. In our patient visual recovery was noticed on the fourth day post admission earlier than what was reported in most studies. This may be due to aggressive effort in normalization and sustenance of the patient’s cardiovascular status.

In hypoperfusion related posterior border zone infarcts, prompt re-establishment of normal perfusion pressure may be associated with resolution of the cytotoxic oedema and visual recovery. In such cases prompt arrest of blood loss and restoration of normal cardiovascular functions may improve the prognosis of visual recovery.

CONCLUSION

Acute severe blood loss could result in cortical blindness. Prompt restoration of normal cardiovascular status is a key step that must be undertaken in such cases in order to ensure or optimize visual recovery.

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