Sudden onset blindness as a presenting feature of chronic subdural haematoma: case report

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Citation


Abstract

We present a rare case report of a patient with chronic subdural haematoma presenting with sudden onset blindness secondary to severe acute bilateral papilloedema. Following prompt surgical evacuation, the patient’s visual acuity returned back to almost normal. Previous reported cases in the literature of blindness following subdural haematomas were related to bilateral homonymous hemianopia due to compression of the posterior cerebral artery following tentorial herniation. These patients were either left blind or developed significant visual deficit despite surgical evacuation. Our case is the first in the literature where the blindness was reversible and due to severe acute papilloedema. There was also no evidence of posterior cerebral artery territory ischaemia. We discuss the importance of visual symptoms in subdural haematoma and emphasize the importance of prompt early intervention.

Clinical Presentation

A 21 years old man was transferred as an emergency from the regional district general hospital after he presented with sudden onset of complete blindness in both eyes in the early hours of the day of transfer. Prior to this, he has been complaining of severe progressive headache with episodic visual blurring over the previous seven weeks. There was nothing of note in previous background history.

On examination, he was alert, confused and obeying commands. He was eye opening spontaneously and there were no lateralizing signs. He was completely blind in both eyes and there was evidence of gross papilloedema bilaterally.

CT (computerized axial tomography) scan showed a significant left sided acute on chronic subdural haematoma with 1 cm midline shift and also a left middle fossa arachnoid cyst. These findings were confirmed on MR (magnetic resonance) imaging (Fig 1) with MRA (magnetic resonance angiography) ruling out an underlying vascular abnormality.

Figure 1

Fig 1: MRI scan showing chronic subdural haematoma causing midline shift. There is also a left temporal arachnoid cyst.

Considering the papilloedema and scan appearances, he underwent emergency burrhole drainage of subdural haematoma on the evening of transfer.

He had significant recovery of his vision within 24 hours of his surgery. Visual acuity has been recorded as 6/9 right eye, 6/12 left eye with good fields in Goldman perimetry but with enlarged blind spots bilaterally. He was subsequently discharged home and at the time of discharge, he was self caring, alert, oriented and had not suffered any deficits a result of the surgery. On review in the outpatient clinic in 6 weeks time, his visual acuity in the right eye was 6/6+2 and left eye 6/9+1. Repeat CT scan (Fig 2) showed that the...
subdural haematoma had resolved and the arachnoid cyst was unchanged.

**Figure 2**

Fig 2. Follow up CT scan showing complete resolution of the haematoma.

DISCUSSION

Blindness following subdural haematoma is very rare with few reported cases in the literature [4,9]. Most of these cases were related to bilateral homonymous hemianopia as a result of posterior cerebral artery infarction secondary to tentorial herniation [4,9]. Despite surgical evacuation, these patients were either left blind or with significant reduction in visual acuity. Two patients in Keene’s series with subdural haematoma who presented with blindness was also noted to have optic atrophy, indicating a pregeniculatge site of optic pathway damage [4]. Though the pathophysiology of this is not clear, Lindenberg et al [5,6] have demonstrated that in addition to occipital infarction, tentorial herniation may result in unilateral or bilateral optic pathway damage at the level of the optic nerve, chiasm, optic tract or lateral geniculation body. Herniation may, either by direct compression of the anterior visual pathway or indirectly through impairment of their local blood supply; cause unexplained “optic neuropathy” in patients with intracranial masses [4].

There has been reported cases of patients with papilloedema who has been left blind following neurosurgical decompression [8]. Russeger et al [8] reported a case of a 51 years old patient who developed bilateral blindness following decompression of chronic subdural haematoma. They suggested that a breakdown of the altered vasoregulation of optic nerves due to papilloedema at the time of intracranial pressure drop was the aetiological cause of optic atrophy and subsequent amaurosis. Similar observations were also noted by Keane [3] who suggested that decompression precipitates ischaemic optic neuropathy in discs in which perfusion is compromised by severe papilloedema.

Papilloedema occurs in patients with both acute and chronic subdural haematoma but is commonly observed in the acute phase. Early papilloedema is usually asymptomatic and even well developed papilloedema may have minimal or no symptoms. However, as papilloedema worsens patients may complain of transient obscuration of vision. These may vary from mild blurring to complete blindness [1]. However, in all cases recovery of vision is invariably rapid and complete [2, 7] Transient obscurations of vision are worrisome for the patient but do not predict visual outcome [1].

Our patient had transient episodes of visual blurring from which he recovered. This undoubtedly was due to the worsening of his papilloedema secondary to raised intracranial pressure until a point when he became completely blind. There was no evidence of posterior cerebral artery territory infarct in our case. This sudden onset blindness secondary to severe acute papilloedema is a very unusual presentation of chronic subdural haematoma and the first reported case in the literature. Another unusual feature in our case is that there was almost full recovery of vision following surgical intervention. This is unusual because once there is visual acuity decline or visual field deficit; the visual prognosis is extremely tenuous [1].

CONCLUSION

Our case is the first reported case in literature of reversible blindness secondary to papilloedema as a presenting feature of chronic subdural haematoma. We feel that transient episodes of visual blurring in a patient with subdural haematoma is an important symptom. This indicates that the papilloedema is getting worse due to persistently rising intracranial pressure. Urgent surgical evacuation should be considered in these patients.

References

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