Quest Journals
Journal of Medical and Dental Science Research
Volume 10~ Issue 2 (2023) pp: 30-32
ISSN(Online) : 2394-076X ISSN (Print):2394-0751
www.questjournals.org

Research Paper

A Case of Missed Diagnosis of Chronic Bilateral Subdural Hematoma Resulting in Permanent Bilateral Loss of Vision

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Abstract
Introduction
Chronic subdural hematoma can lead to loss of vision. This is the first reported case of chroic subdural hematoma resulting to bilateral loss of vision.
Case Presentation
A 35 year old male who was involved in road traffic accident. There was multiple injuries with head injury but no neurological symptoms. He was admitted in a hospital but was later discharged without a brain CT scan. A year later he developed headache, quadriplegia and loss of consciousness. His management was multidisciplinary. Brain CT scan revealed chronic subdural hematoma. He had immediate burrhole and evacuation of hematoma. He later regained consciousness and movement of all his limbs, but never regained his sight.
Discussion
Subdural hematoma can affect vision through compression or vascular compromise at many points along the visual pathway .[1]There are few reported cases in literature of vision loss secondary to subdural hematoma. Most cases of vision loss with subdural hematoma affect the posterior visual pathway, with mechanisms including occipital infarct and compression of the posterior cerebral artery during transtentorial herniation. The anterior visual pathway can be compromised directly by gyrus herniation into the suprasellar cistern. Loss of vision secondary to subdural hematoma should be arrived at after ruling out concomitant orbital trauma and pre-morbid ocular pathology. This is supported by radiologic assessment.
Conclusion
Permanent loss of vision can be a late sequelea of chronic subdural hematoma. In the absence of neurological symptoms following head injury, a high index of suspicion is necessary for prevention. The patient still requires a thorough ocular examination, early radiologic investigation, proper counseling on this untoward complication, and urgent surgical intervention to prevent blindness.

Received 25 Jan., 2023; Revised 07 Feb., 2023; Accepted 09 Feb., 2023 © The author(s) 2023.
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I. Introduction

Chronic subdural hematoma can lead to visual loss. A few cases have been reported with temporal or unilateral loss of vision. This was a case of a young man who was apparently healthy with minor injuries after a road traffic accident but developed symptoms a year later relating to subdural hematoma. Among them was bilateral loss of vision which did not resolve with surgery. He was taken to the hospital after the accident but the diagnosis of acute subdural hematoma was missed until a year later when symptoms started manifesting , including loss of vision.

Case Presentation

The patient was A 35 year old male who presented with inability to move all limbs of one month duration and loss of consciousness of 5 days duration. He was apparently well until one month prior to presentation when he complained of headache which progressively worsened. There was no vomiting or blurring of vision, there was difficulty with speech and walking, and reduced appetite. There was inability to use all four limbs. 5 days prior to presentation he was noticed to be unconscious which necessitated his being rushed

to a peripheral hospital. The result of the brain CT scan done showed chronic subdural hematoma. He was referred to our centre for expert management.

There was history of road traffic accident a year earlier, the vehicle he was traveling in somersaulted multiple times. He was hitting both sides of his head on the inner parts of the vehicle until he stabilised himself by holding a part of the car. There was no loss of consciousness then, he walked out of the vehicle by himself. He sustained fracture of the right hand and was prior to presentation initially managed at a tertiary health institution outside the state.

He was apparently normal for about a year and going for treatment on his own until thirty-three days prior to presentation when he started complaining of headache which progressively worsened.

Examination showed a young man, unconscious, pale, anicteric, acyanosed, not dehydrated. Pulse rate 132/minute, blood pressure120/90mmHg, respiratory rate 18 cycles /minute.

GCS- E-1 V-5 M-1 . 7/15 (Quadriplegia)

Musculoskeletal system: left upper limb in POP cast from the elbow to the fingers.

Assessment – multiply injured patient with 1.Bilateral chronic subdural hematoma secondary to severe head injury following Road traffic accident 2. Left Upper limb fracture on management.

Plan, For emergency exploration. Obtain a high risk consent.

He had Bilateral burrhole craniostomy next day. Under local anaesthesia, crank oil hematoma was drained under controlled pressure. Wound irrigation was done.

First day post op, pupils: mildly dilated and reactive to light., CNS: GCS E – 1, V- 5, M-1 =7/15

ASSESSMENT : slightly improved clinical state.The Ophthalmology team was invited for eye care.

The ocular examination was as follows:

Right eye		Left eye
Unconscious	OA	Unconscious
Copious purulent eye discharge	lid	copious purulent eye discharge
Intact	globe	intact
Proptosis		Proptosis
Non-pulstile		Non-pulsatile
No retropulsion		No retropulsion
injucted Chemosis	Conjunctiva	Injected chesosis
Punctate opacity at 4)'clock	Cornea	Area of opacity R/o ulcer inferiorly
Gld	Alc	Gld
Mild dilated unreactive	Pupil	Mild dilted unreactive
Tpt	lens	tpt
Fundus		

Ocular examination repeated 5 weeks later

Right	left	
Inerior punctuate opacity	Corneal	inferior diffuse opacity
Fully dilated unreactive	Pupil	Fully dilated unreactive
Tpt	lens	Tpt
Pale disc, distinct margins VCAR 0.1,		
Kertenbaum sign positive	Fundus	Pale disc, distinct margins, VCAR 0.1, Kertenbaum sign positive
Hard exudates	Macula	Hard exudates

An impression of bilateral optic atrophy secondary to intracranial space occupying lesion was made. Visual rehabilitation recommended.

By 5th day post op, He could vocalize but could not move any of the limbs. Had bilateral subconjunctival chemosis. commenced physiotherapy. Ocular examination showed he had bilateral conjunctivitis R/O orbital cellulitis, proptosis with exposure keratopathy,and papilloedema. Ocular toileting was done. Orbital CT was requested. He was placed on Cutt Ciloxan 2 hourly for 2 weeks, tetracycline ointment 8 hourly and taping of the eyelids at night.

Neurological review showed CNS: GCS: E- 4, V – 4, M-6. An assessment of Resolving Chronic Subdural Hematoma with paraplegia. By 10 days post op he was fully conscious.The nasogastric tube was removed for trial of oral feeding.

On the 16th day post op he was discharged from neurosurgery management.He was ambulated with wheelchair. He continued physiotherapy. Ophthalmology review showed Bilateral optic atrophy due to intracranial space occupying lesion. He commenced visual rehabilitation.

II. Discussion

This is one of the few reported cases of visual impairment following subdural hematoma. When there is head injury attention is focused on the hematoma and its evacuation to prevent uncal herniation neglecting the ocular complications. In the index case, late diagnosis was a major factor. The diagnosis of subdural hematoma was missed till about a year later when the complications arose.

Subdural hematoma can affect vision through compression or vascular compromise at many points along the visual pathway .[1] Most cases of vision loss with subdural hematoma affect the posterior visual pathway, with mechanisms including occipital infarct and compression of the posterior cerebral artery during transtentorial herniation. Posterior lesions may present with anterior signs. Necropsy studies have shown thattranstentorial herniation can result in damage at the level of the optic tract, chiasm or optic nerves.[2]

The anterior visual pathway can be compromised directly by gyrus herniation into the suprasellar cistern. Prechiasmal vision loss due to intracranial optic nerve infarction has also been reported in the setting of sudural hematoma. The nerve can be compressed against the basal skull structures. The precise mechanisms of these remain poorly understood. [3]

Hollander et al have suggested that rare cases of acute optic nerve following subdural hematoma are due to local pressure-induced optic nerve infarction.[4]

Chronic subdural hematoma has been reported to cause visual disturbances and loss of vision. The common presentation is homonymous hemianopia secondary to compression of the posterior cerebral artery during transtentorial herniation.[5] S. Hasegawa et al. reported a rare case of chronic subdural hematoma presenting with bilateral visual impairment caused by papilloedema in a 48 year old. Patient however recovered his vision after hematoma evacuation.[6]

Kretz et al reported the case of a 70 year old Caucasian German who developed a massive left hemispheric subdural hematoma under oral anticoagulation. He presented with acute, severe visual impairment on his left eye, which was noticed after surgical decompression. Neurologic and ophthalmologic examinations indicated sinistral optic neuropathy with visual acuity reduced to nearly amaurosis. No subsequent visual improvement. [7]

Loss of vision secondary to subdural hematoma should be arrived at after ruling out concomitant orbital trauma and pre-morbid ocular pathology. Early ocular examination would show normal fundoscopy and lack of papilloedema. These can lead to a diagnosis of optic neuropathy. This is supported by radiologic assessment. The extraocular causes of blindness in subdural hematoma can be arrived at by MRI/CT scan which can show optic, vascular or brain parenchymal trauma, or ischemia. This can be pressure induced as a result of cerebral edema or resultant ischemic injury resulting in edema. The diagnosis is most of the time made from autopsy studies. Kretz et al described the first in vivo diagnosis.[8]

Ours is the first reported case of bilateral loss of vision following subdural hematoma.

III. Conclusion

Permanent loss of vision can be a late sequelea of subdural hematoma. This can occur by several mechanisms. In the index case, there was head trauma but no symptoms developed till about a year by which time irreversible loss of vision had occurred. A high index of suspicion is necessary for prevention. Any case of head injury with or without loss of consciousness, obvious bruises, bleeding from craniofacial orifices, headache, vomiting, hemiplegia, loss of sphincteric function, visual disturbances etc still requires a thorough ocular examination, early radiologic investigation, proper counseling of the patient on this untoward complication, and urgent surgical intervention to prevent blindness. A patient with subdural hematoma who develops sudden or gradual loss of vision requires urgent surgery and evacuation.

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