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VERTIGO AS A PRESENTATION OF SUBDURAL HAEMATOMA

Medical Science

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ABSTRACT

Vertigo is a common nonspecific complaint of patients who present to emergency department. Vertigo is caused by disturbance of the central processing of sensory signals from the vestibular apparatus that provide information regarding the position of the body in space. An important aspect in the management of patients with vertigo in the emergency department is the differentiation of associated acute stroke syndromes due to peripheral causes.1 Patients with chronic subdural haematoma can present with atypical manifestations like vertigo and nystagmus adding a lot more confusion to diagnosis.

KEYWORDS

Vertigo, Nystagmus, Benign paroxysmal positional vertigo, Chronic subdural hematoma

CASE HISTORY

A 31 years old apparently healthy female presented to ED complaining of one episode of vomiting last night and irritability since early morning. She had associated left ear pain for the past two days with no ear discharge. She had a history of giddiness four days back which was diagnosed as benign paroxysmal positional vertigo. Patient also complained of headache in the past seven days. On examination, patient was found to be drowsy, but arousable to call and obeying oral commands with no motor deficits. Her vitals were stable and physical examination showed no abnormality. ENT assessment was unremarkable.

She had focal seizures of the right upper limb during her course of stay in the ED and was treated for the same. Fundoscopy was normal. BPPV, cerebral abscess or temporal meningitis was kept in the differential diagnosis. MRI brain was donewhich revealed a large subdural collection in left frontal, parietal and temporal convexity.It was measuring 16mm in maximum thickness causing mid line shift of about 13mm to right with mass effect in the form of compression of left ventricle, subfalcine and uncal herniation with compression of the midbrain. Right occipital horn of the lateral ventricle was dilated with periventricular hyperintensities. (Figure1)

Patient sensorium was found to be deteriorating. Her GCS dropped to 8/15 from 13/15 at presentation.

Hence patient was electively intubated in ED and shifted for emergency craniotomy. She underwent burr hole evacuation of the SDH uneventfully and was discharged home after 3 days of stay in the hospital.

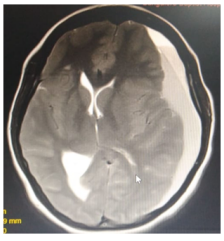

Figure 1. MRI brain showing a large subdural collection in left frontal, parietal and temporal convexity measuring 16mm in maximum thickness causing mid line shift of about 13mm to right with mass effect.

DISCUSSION

Dizziness is one of the most common presentations to the ED. Identifying the etiology remains a challenge to emergency physicians. Drachman categorized dizziness as A) presyncope which is a sense of impending loss of consciousness due to inadequate blood flow or metabolic impairment, B) Disequilibrium caused by motor dysfunction that impairs balance or gait C) Vertigo or a sensation of motion due to disorder of labyrinth D) light headedness which includes non-specific symptoms related to multiple sensory and psychiatric disturbances.[2]

Currently, misdiagnoses are frequent and the cost of diagnostic testing is very high. The traditional diagnostic approach, which relies on symptom quality or type (i.e., vertigo, presyncope, or disequilibrium) does not distinguish benign from the dangerous causes, and is inconsistent with the current best evidence. A new approach divides patients into three key categories using timing and triggers, guiding a differential diagnosis and targeted bedside examination protocol: 1) acute vestibular syndrome, where bedside physical examination differentiates vestibular neuritis from stroke; 2) spontaneous episodic vestibular syndrome, where associated symptoms help differentiate vestibular migraine from transient ischemic attack; and 3) triggered episodic vestibular syndrome, where the Dix-Hallpike and supine roll test help differentiate benign paroxysmal positional vertigo from posterior fossa structural lesions.[3]

Chronic SDH usually presents with a variety of causes which are not easily identifiable in the midst of a busy ED. Trauma is an important factor in the development of CSDH. However, a history of head injury might be absent in about 30%–50% of the cases. Indirect trauma seems to be more important. Other predisposing factors include anticoagulation, alcoholism, epilepsy, bleeding diathesis, low intracranial pressure secondary to dehydration or after the removal of cerebrospinal fluid and in those receiving renal dialysis presumably due to platelet dysfunction.

CSDH commonly presents with altered mental status, focal neurological deficit, headache and seizures. It may rarely present as isolated neurological deficits. Our patient had isolated right upper limb seizures.

CONCLUSION:

The diagnosis of CSDH should be considered in any patient with or without history of trauma with a change in mental status, focal

neurological deficit, headache with or without neural deficits. Neuroimaging should be kept in mind when such a patient presents to ED.[4]

Chronic SDH has been rightly termed “the great imitator” in view of diversity of symptoms noted.[5]

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