Chronic subdural hematoma presenting as diplegia-A rare presentation

Sir,

Correlative clinical neurology, imaging findings and surgical results run hand in glove. There are, however, at times we do face discordance/unexplainable clinical parameters or radiological features. Patients with chronic subdural hematomas usually present with refractory headache, amnestic deficits, lack of concentration and hemiparesis.^[1] However, atypical presentations have been described. We herein present one such rare presentation of chronic subdural hematoma presenting as cerebral diplegia with preserved mentation and urinary incontinence.

A 60-year-old male presented to emergency medicine department with gradually progressive weakness of all limbs and urinary incontinence for a total duration of 20 days. He had a trivial fall about 20 days back due to fall in the bathroom. Over next 5 days, the attendants noted that he was dragging his left foot in a circular fashion. He had gradual involvement of his right lower limb with urinary incontinence in about 10 days and was bed ridden. He had no difficulty in using his upper limbs. Three days later he developed weakness of left upper limb which was noted when he was not able to hold a glass of water. He was evaluated with a CT scan brain plain and referred to us. Upon arrival to emergency department, the patient was conscious and coherent. Pupils were bilaterally 2.5 mm and were briskly reacting to light. Bilateral fundii showed papilledema. He had spastic paraplegia of both lower limbs and left upper limb (power- spastic 0/5). Right upper limb was 3/5, distal weaker than proximal. His deep tendon reflexes were brisk and plantars bilaterally extensor. There were no signs of meningeal irritation. Sensory examination was unremarkable. His repeat CT scan brain plain and contrast showed bilateral chronic subdural hematoma, right > left, with midline shift to the left by 10 mm and imminent uncal herniation. There was no collection noted in the interhemispheric region. The higher cuts through the parietal region too were clear. After evaluation of his hematological parameters (bleeding time, clotting time, prothrombin time and aPTT) which were normal, he was managed by bilateral frontal and parietal burr holes, evacuation of altered subdural blood and closed drainage system,

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Author	Presentation	Progression	Imaging	Follow up
Shields <i>et al.</i> , ^[2] 1980	Paraparesis	Gait disturbances-paraparesis with alert mentation	CECT brain-small ventricles with effacement of sub-arachnoid spaces	2 months
			Cerebral angiogram-extra cerebral lentiform mass	
Lesoin <i>et al.</i> , ^[3] Progressive Case 1983 quadriparesis weak		Case 1-initially altered mentation followed by weakness of both lower limbs over a period of 3	CECT brain-bilateral chronic subdural hematoma	Both patient improved to
		months which rapidly progressed to quadriparesis	Cerebral angiogram-bilateral	normalcy in a month
		Case 2-right leg weakness rapidly progressed to quadriparesis in 5 days duration	extra cerebral lentiform collection	
Kyriacou <i>et al.</i> ,[4]	Paraparesis	Both lower limbs simultaneously	CECT brain-bilateral chronic	3 months
2012			sub dural hematoma	Improved to normal
(Present case)	ent case) Quadriparesis Weakness started in left upper limb - right lower limb - left upper limb - right upper limb		CECT brain-bilateral chronic subdural hematoma, Right>Left	Improved in the immediate post-op period

CECT: Contrast enhanced computerized tomography

under scalp block. In the immediate postoperative period, there was improvement in his motor power to 4+/5 in all the limbs. The drainage system was removed after 72 hours. However, to rule out a cervical pathology, a lateral radiograph of the cervical spine was performed which was normal.

In the absence of sensory findings, the pattern of progression of the weakness was the main contention which was akin to the progressive lesion in and around the paracentral lobule (parasagittal) as opposed to the pathology located far away, laterally. We reviewed the literature for presentation of chronic subdural hematoma with presentation as quadriplegia and found very few articles^[2-4] [Table 1].

The proposed mechanisms described are-

- Compression of anterior cerebral arteries (proximal-altered sensorium; distal-clear mentation)
- Compromise to venous drainage leading to cortical function
- Direct compression of the cortex.

A combination of both arterial and venous flow abnormalities along with perfusion abnormalities might well be the cause of diplegia, referred to as diaschisis. The disease process of chronic subdural was described as a "the great neurological imitator" by Patter and Fruin.^[5] The intention to present this case is to highlight the fact that the physiological changes that occur in the microcirculation are still not completely understood and represent a gray zone for further exploration.

It is prudent to be aware of such abnormal presentation of a relatively common neurological disease for early treatment.

Acknowledgement

We thank Prof. BP Sahu for inspiring us to prepare the manuscript.

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Quick Response Code:	Website: www.ruralneuropractice.com		
	DOI: 10.4103/0976-3147.140024		