Kernohan-Woltman notch phenomenon and intention tremors in case of chronic subdural hematoma

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Abstract

Movement disorders are atypical and rare presentation of chronic subdural hematomas. We report a case of 60 year man who presented with intention tremors and altered sensorium. The patient had Kernohan-Woltman notch phenomenon on clinical examination. CT scan brain showed a large left fronto-temporo-parietal chronic subdural hematoma with significant mass effect and midline shift. His symptoms relieved completely after surgical evacuation of the hematoma.

Key words: Chronic subdural hematoma, Kernohan-Woltman notch, tremors, intention tremor.

Introduction

Chronic subdural hematomas (CSDH) can present with a wide variety of symptoms, however movement disorders (the most common is secondary Parkinsonism) in CSDH are atypical and rare presentations. (1, 2) Kernohan-Woltman notch phenomenon appears when any supratentorial mass lesion cause

the midline shift and there is compression of the cerebral peduncle against the tentorial edge ipsilateral hemiparesis or hemiplegia and mydriasis. (3-5) The appearance of Kernohan-Woltman notch phenomenon has rarely been reported in patients with CSDH. (4-7) In present article we report an extremely rare presentation as intention tremors and Kernohan-Woltman notch phenomenon that resolved after surgical evacuation.

Case report

A 60-year-old man presented with progressive stiffness and weakness of all four limbs (left more than right), abnormal movements of right upper limb and altered sensorium of two days duration. There was not history of fall. There was no past history of diabetes or hypertension. He had pulse rate of 50 per minute. Other general and systemic examination was unremarkable. On neurological examination he was in altered sensorium. Tone was increased in all four limbs. There was weakness of grade 0/5 of left upper

and lower limbs. He was localizing with right upper limb. All the deep tendon reflexes were brisk and planters were extensor. There was mild papillary asymmetry (left>right) but both were reacting to light. He had irregular low frequency tremor those were becoming grossly uncontrollable with these attempted movements (Video-1). Routine hematological and biochemical investigations were within normal limits. CT brain plain showed a largely hyperdense collection with layering over left fronto-temporo-parietal region with significant mass effect and midline shift (Figure 1). He underwent left frontal and temporal burr hole under local anesthesia and evacuation of chronic subdural hematoma. He made significant improvement over next 24 hours in his sensorium. He regained consciousness, his weakness improved and abnormal movements disappeared over next 48 hours (Video-2).

Figure 1

CT brain plain showing a largely hyperdense collection with layering over left fronto-temporo-parietal region with significant mass effect and midline shift

Discussion

Sudden onset of movement disorder in the elderly patients requires prompt radiological investigations to rule out treatable causes of these dysfunctions. (2) Movement disorders in CSDH patients probably caused by a pressure effect on basal ganglia structures (8, 9) or by altering the function of neurotransmitters. (9) compression of midbrain (interfere nigro-striatal dopaminergic transmission) (10) and compression of anterior choroidal artery (11) However in contrast to the resting tremors of the Parkinsonism (12, 13) the present patient had intention tremors. Intention tremors are mainly occurs in cerebellar disorders, characterized as a kinetic tremor with a prominent intention component and the ipsilateral extremity is involved when a cerebellar hemisphere is involved. (14, 15) Also as the cerebellum is connected to contralateral thalamus and cortex, both cerebellar lesions and contralateral thalamocortical lesions can result in cerebellar dysfunction. (15, 16) It has been suggested that the cerebellum, superior cerebellar peduncles, red nucleus, and thalamus can be involved in the generation of intentions tremors and the modulation of cerebello-thalamic projections has been shown to improve the tremors. (17, 18) On imaging studies atrophic structures or changes in signal intensity has been demonstrated in the cerebello-rubral thalamic tract and the etiological significance of the tract has been confirmed as the mechanism of the

intention tremors. (19) Direct pressure on or invasion of deep basal ganglionic structures and brain stem compression due to upward or downward herniations which displaces the midbrain against the tentorial edge (20, 21) could be responsible not only for the tremors but also for the hemiparesis. Hemiparesis can be found in up to 58% of cases of CSDH and mostly the deficit is contralateral to the lesion (direct pressure on the cerebral hemisphere). (22) In rare circumstances the focal neurological deficit can be ipsilateral to CSDH. (5, 22) Our patient had ipsilateral weakness, abnormal movements in the upper limb, marginal asymmetry of the pupils, a finding consistent with Kernohan-Woltman notch phenomenon. (3, 5) Apart from that the prompt evacuation leads to complete reversal of Kernohan-Woltman notch phenomenon and complete recovery of motor functions. (4-6)

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