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Internal Capsular Lesion Associated With Dizziness

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It has been known that the vestibular system is concerned with feelings of dizziness or vertigo. The vestibulothalamic pathway has also been described previously. However, there has been no confirmative report so far regarding the pathway through the internal capsule to the cortex. We have experienced 13 patients with symptoms of dizziness and/or vertigo whose lesions are located only around the internal capsule, mainly at the posterior limb and/or the genu. It is suggestive that fibers with dizziness may pass through a part of the internal capsule, probably through some part of the posterior limb and/or the genu.

Key Words: *Internal capsule, dizziness*

INTRODUCTION

Dizziness is a false and conflicting sensation of motion or position. Vertigo has connotations of rotation or a partly vestibular symptomatology in many patients, whether or not it is rotational. Normal maintenance of balance is dependent on proper functioning of the vestibular system, the visual system, position sense pathways, and the complex motor interactions between the cortex, basal ganglia, and cerebellum. Temporary malfunction of any portion of this system can result in a sense of imbalance characterized by the patient as dizziness.

Usually, dizziness occurred in association with lesion of basilar artery and/or posterior cerebral artery, but rarely with the internal carotid and/or middle cerebral artery. We have experienced 13 patients with internal capsular lesions, confirmed by brain CT scan. All 13 cases presented with the chief complaint of dizziness. The frequency of dizziness in our cases observed to be higher in the cases of lesion in the posterior limb.

MATERIALS AND METHODS

The records of the patients followed at the Neurology Clinic, Hanyang University Hospital from 1980 to 1990 were reviewed and 13 patients were found to have dizziness along with internal capsular lesion. The history and neurologic examination was obtained for each patient. Vestibular function test, computed tomography, angiography, electroencephalography, magnetic resonance image and evoked potential response were performed on each patient.

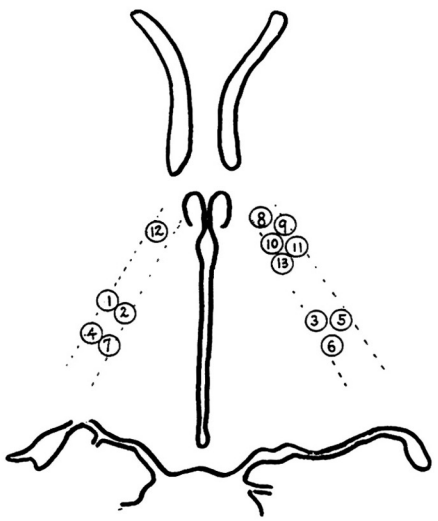

Fig. Location of Internal Capsular Lesions in 13 Patients Associated with Dizziness (The Arabian numerals represent the number of cases)

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RESULTS

The group with internal capsular lesion associated with dizziness consisted of 13 patients (7 men and 6 women, mean age: 54 years, range: 35 to 72 years) (Table). They had isolated episodes of dizziness as the initial symptom. They had complained of dizziness for a duration of 2 months to 3 years. Our patients have used the following descriptions about dizziness: spinning (environment moves), tilting of the environment, drunkenness. unsteady feeling in head, going sideaway, and lightheadedness. The pattern of the dizziness of our cases is likely to be central dizziness. Brain CT scan showed no other abnormality except for small infarcted internal capsular lesions.

There was a lesion of the posterior limb of the internal capsule in 7 patients, and the other 6 patients had a lesion close to or around the genu of the internal capsule. (Fig) In the vestibular function test (past pointing test, Romberg test, Tandem walking, Doll's eye test, caloric test, and rotational test), there had been no abnormality noted in all patients. The angiography findings did not adequately explain the dizziness. Electroencephalography showed no abnormal recordings. Evoked potentional responses (brainstem auditory evoked and somatosensory evoked responses) were within normal limits.

COMMENTS

Dizziness is a false and conflicting sensation of motion or position, it may seem to involve the patient or his environment or both. When the patient feels a motion of rotation, we might speak of rotational dizziness or vertigo. However, vertigo is a very specific condition in which environment appears to rotate or the patient himself feels rotation. It is purely subjective.

Dizziness frequently occurs in patients suffering from ischemia in the distribution of vertebrobasilar circulation and/or posterior cerebral circulation, but rarely in the internal cartid and/or middle cerebral circulation. According to the report (Fisher, 1967) of 140 cases with either hemiplegic strokes or recurrent transient ischemic attacks with internal carotid artery and middle

Table 1. Patients Summary

	Sex	Age	Initial Sx	Associated Sx and signs	Lesion Site	Lesion Nature	Sx Duration (month)
Case 1	F	48	Dizziness	Hemiparesis	Post. limb Rt	Infarction	11
2	F	58	Dizziness	Headache	Post. limb Rt	Infarction	8
3	M	57	Dizziness	Numbness	Post. limb Lt	Infarction	3
4	F	52	Dizziness		Post. limb Rt	Infarction	3
5	F	69	Dizziness	Headache Hemiparesis	Post. limb Lt	Infarction	24
6	M	60	Dizziness		Post. limb Lt	Infarction	10
7	F	44	Dizziness		Post. limb Rt	Infarction	2
8	F	52	Dizziness		Post. limb+ Genu Lt	Infarction	3
9	M	50	Dizziness	Headache Hemiparesis	Post. limb+ Genu Lt	Infarction	4
10	M	35	Dizziness	Paresthesia	Post. limb+ Genu Lt	Infarction	2
11	M	60	Dizziness		Post. limb+ Genu Lt	Infarction	4
12	M	55	Dizziness		Post. limb+ Genu Rt	Infarction	5
13	F	72	Dizziness	Lightheadedness	Post. limb+ Genu Lt	Infarction	36

cerebral artery lesion, 12 (8%) mentioned dizziness. It may occur in isolation, with other symptoms of vertebrobasilar insufficiency, or with persisting symptoms and signs of infarction of the brainstem and/or cerebellum (Williams and Wilson, 1962).

When other symptoms and signs are present, the diagnosis is usually obvious, whereas when dizziness occurs in isolation, it is difficult to differentiate from other benign disorders.

Our cases do not have symptoms and signs related with other neurologic disorders such as brainstem, thalamus and cerebellar lesions. The dizziness is not transient and is to be milder, continuous, and prolonged. The patients suffered from spinnig sensation and/or drunkenness, tilting, unsteady feeling in head, going sideaway, and lightheadedness. They complained of dizziness lasting 2 months to 3 years. Also our cases must be differentiated from peripheral disorders such as benign inner ear disorders. Benign positional vertigo and Meniere's disease have their own typical symptoms and signs in contrast to our cases. And our cases do not show a characteristic nystagmus.

In addition, there was no evidence of labyrinth infarction such as deafness or abnormal caloric response. Some patients with vertebrobasilar insufficiency had at least one episode of vertigo; in some cases isolated vertigo was initial symptom (Grad and Baloh, 1989). Although our cases are superimposed with a vertebrobasilar insulfficiency, no case developed typical symptoms of vertebrobasilar insufficiency. And vertebrobasilar infarction could not be excluded absolutely by brain CT scan and angiography. But brain CT scan was normal except the lesion of the internal capsule and clinical symptoms and signs were not indicated posterior fossa lesions. The angiographic finding was abnormal in two patients (mild stenosis of middle cerebral artery and its terminal branches, and focal atherosclerotic changes in internal carotid artery) but the findings did not adequately explain the clinical symptoms and signs.

The magnetic resonance image was performed in some patients and the results were not showed lesions of the posterior fossa. Evoked potentials (brainstem auditory evoked response and somatosensory evoked response) were performed and none of them were suggestive of any evidence of brainstem lesion. Also, our cases do not have a history of migraine and epilepsy.

A vestibular cortical area was first clearly identified in the suprasylvian gyrus of the cat (Walzl and Mountcastle, 1949). And a similar result was obtained from humans. The vestibular area in the cortex has been shown to be adjacent to transverse gyrus of Heschl (Penfield, 1957). Recent studies in humans have revealed vestibulothalamic projections. According to one report (Hawrylyshyn et al., 1978) there are two distinct vestibulothalamic projections: a) an anterior relay to the nucleus ventrointermedius, b) a posterior relay to the medial geniculate body. And vestibular pathways below the thalamic level have also been identified (Copack et al., 1972).

But, thalamocortical connections have not been discovered or confirmed yet. Electrical stimulation of the cerebral cortex, either of the posterolateral aspects of the temporal lobe or the inferior parietal lobe near the sylvian fissure may evoked intense vertigo. The pathways of dizziness from the thalamus to the cortex might be through the some places of subcortical areas. According to another report (Fisher, 1967) of 60 cases of internal capsular lesion, none of them complained of dizziness.

However, we have experienced 13 patients with lesions of the internal capsule associated with dizziness. They had pathologic lesions around the internal capsule and there were no other causative etiologies concerned with dizziness. Therefore, we presume the fibers related to dizziness may pass through the internal capsule to the cortex, probably through some part of the posterior limb and/or genu. Some patients still complaint of dizziness after subsiding the assoociated sign such as hemiparesis. It is probably due to "irritative phenomenon" around the internal capsule. In other words, dizziness with prolonged deficits is probably due to "destructive phenomenon".

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