Parkinsonism, gait apraxia and dementia associated with intracranial calcifications

A case report

R. SANDYK

Summary

A 52-year-old Black man with therapy-resistant parkinsonism, gait apraxia and dementia but no endocrine abnormality was found to have extensive intracerebral calcifications which included the basal ganglia. Although most patients with radiological evidence of calcifications in the basal ganglia remain asymptomatic, a small group may present with an extrapyramidal syndrome that is usually progressive, resistant to therapy, and not associated with an endocrine disorder. Plain skull radiography and computed tomography should therefore be performed in patients presenting with a parkinsonian syndrome unresponsive to therapy.

Intracranial calcifications may be associated with a variety of disorders. Calcifications of the basal ganglia identified radiographically have been found together with numerous lesions, treatable parathyroid disease being the most common. A recognizable clinical syndrome, including extrapyramidal signs, is thought to be an integral aspect of basal ganglion calcification. The patient with intracranial calcifications described below had a neurological disorder consisting of therapy-resistant parkinsonism, gait apraxia and dementia in the absence of endocrine abnormality.

Case report

A 52-year-old Black man was admitted to hospital for control of his parkinsonism; he had had two previous admissions for control of his extrapyramidal syndrome with poor response to treatment. He had been taking carbidopa (1 tablet 3 times daily) and amantadine (200 mg daily) for the past 2 years.

His symptoms began 4 years before the present admission with difficulties in motor co-ordination and balance. Over the next 3 years his symptoms gradually progressed; he had frequent falls and difficulty in dressing himself, and his speech became slow, slurred and almost inaudible.

Examination revealed severe parkinsonism. His speech was slurred and incoherent and his mental faculties were impaired. His face was mask-like and he was drooling from the mouth. The glabellar tap, pout, palmomental reflex, head retraction reflex and jaw jerk were positive. Plantar 'grasp' was bilaterally positive and the toes were downturned. Muscle tone was increased in all limbs with resisted passive movements. Tendon jerks were brisk in all limbs with slight left-sided predominance. Moderate cog-wheel rigidity was elicited at the wrists, shoulders and neck muscles. He had to be assisted with standing or walking. There was marked hesitancy in initiating steps and his legs appeared to be glued to the floor. There was moderate flexion of the trunk and limbs with marked truncal instability. General examination was otherwise negative. Routine laboratory analysis showed that serum electrolyte levels, parathormone values and renal, liver and thyroid function were normal, as was the cerebrospinal fluid. Skull radiographs showed intracranial calcifications, particularly in the basal ganglia. Computed tomography (CT) confirmed dense calcifications in the basal ganglia, the regions adjacent to the lateral ventricles and the frontal and parietal lobes (Fig. 1).

Discussion

The association between Parkinson's disease and basal ganglion calcification is uncommon. In one study only 2 out of 67 patients (2.9%) with radiographic evidence of basal ganglion calcification had parkinsonian signs. However, parkinsonism is more common in patients with basal ganglion calcification secondary to endocrine abnormalities (such as hypoparathyroidism). Our patient presented with a neurological triad consisting of parkinsonism, gait apraxia and dementia in the absence of meta-

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Hemichorea-hemiballismus caused by lacunar infarction in the basal ganglia

A case report

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Summary

Unilateral hemichorea-hemiballismus developed suddenly in a 60-year-old man. Computed tomography revealed a small area of low density in the region of the left anterior limb of the internal capsule and the head of the caudate nucleus which was consistent with a lacunar infarction. Haloperidol gradually abolished the symptoms within 7 days of their onset. No further choreoballistic movements were experienced. This report reveals that ballistic movements may occasionally be caused by lesions lying some distance from the subthalamic nucleus of Luys.

Case report

A 60-year-old right-handed man was admitted to hospital complaining of sudden onset of right-sided weakness associated with uncontrollable jerking movements of his right arm. There was no previous history of trauma, loss of consciousness, seizures, headaches or other focal neurological signs.

General examination revealed a blood pressure of 210/110 mmHg and a regular pulse rate of 80/min. There was no cardiac abnormality and no carotid bruit, but signs of left ventricular hypertrophy were present which were confirmed on the ECG and chest radiographs. The rest of the examination was negative.

Neurological assessment showed the patient to be alert with normal speech. There were almost constant distal choreic movements of the arm and leg, with occasional brisk proximal ballistic movements. At night the choreoballistic movements diminished slightly, but interfered with sleep. The intensity of the movements increased with emotional or physical stress. No similar movements were detected in the left limbs or in the face. Strength was diminished on the right and tendon jerks were increased in the right arm. The plantar response was extensor on the right and flexor on the left. In addition, there was an upper motor neuron facial palsy on the right. Sensation and cerebellar function were normal. Fundoscopy revealed stage II hypertensive retinopathy.

Computed tomography (CT) performed on the 4th day after the onset of symptoms disclosed a small area of low density in the region of the left anterior limb of the internal capsule and the head of the caudate nucleus which was consistent with a lacunar infarct in the left basal ganglia region (Fig. 1). There was no change when the scan was repeated after administration of con-