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Case report

Enlargement of the posterior horns of the lateral ventricles and recurrent falls: A clinical study

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ABSTRACT

This is a retrospective study of 12 patients with enlargement of the posterior horns of the lateral ventricles and recurrent falls. The cause was posterior cortical atrophy in eight patients and white matter disintegration in four. Changes in the gray and white matter in the parieto-occipital regions and ventricular expansion are associated with disintegration of the visuospatial and attentional mechanisms, compromising safe navigation and mobility, and increasing the risk of falls. Age, hypertension, and diabetes in the study could independently increase the risk of falls, and could be factors in the development of structural and functional changes in the areas involved.

1. Introduction

Posterior cortical atrophy (PCA) is a neurodegenerative condition[1] characterized by an array of manifestations. They include higher visual and spatial deficits with subsequent development of components of Balint's and Gerstmann's syndromes and transcortical sensory aphasia. Balint's syndrome is characterized by optic ataxia, gaze apraxia and asimultanagnosia and Gerstmann's by acalculia, finger agnosia, and left–right discrimination. Among patients with PCA several symptom clusters or subsyndromes emerge, which have recognized correlates; for example, Gerstmann's syndrome is due to left angular dysfunction and Balint's syndrome to bilateral parieto-occiptal dysfunction, amongst others. Memory and cognition are spared until late in the course of the illness.[2] The syndrome is associated with a variety of underlying pathologies. The pathological findings commonly reported are that of Alzheimer's disease. It is often defined as a variant or atypical form of Alzheimer's disease, although the clinical presentation is distinctly different to Alzheimer's disease. Although posterior cortical atrophy does not represent a single pathology, it may not be the pathology but the neurobiological changes that are most relevant to the clinical features.[3] Localized ventricular dilatation of the brain can be due to *ex-vacuo* dilatation resulting from either cerebral atrophy (gray or white matter or both) or to structural damage to the brain parenchyma as from stroke or trauma.

The purpose of this study is to join the dots between dilatation of the posterior horns of the lateral ventricles and falls and fallers. This is a retrospective study of 12 patients with unexplained recurrent falls seen over a period of 4 years. The inclusion criterion for this study was the finding of dilatation of the posterior horns of the lateral ventricles on visual inspection of the computed tomography (CT) scan of the brain and falls. Ventricular enlargement on CT was assessed by linear measurement of the ventricle to brain size and this was done for both anterior and posterior horns of the lateral ventricles and ventricular to brain size (VBR) calculated.

2. Case reports

2.1. Case 1

A 68-year-old man from an aged care facility presented with a history of recurrent falls and unsteadiness of gait over a period of 1 year but this could have been longer. He had epilepsy and was well controlled on dilantin sodium and valproate. He had been a heavy drinker but had reduced the intake over the past year or so.

When seen, he was alert with a near normal attention span. Formal and psychometric testing for memory and cognition

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revealed moderate impairment. The Mini Mental State Examination (MMSE) score was 13/30. Episodic memory for past and recent events were grossly impaired. On neurological examination the patient was asked to touch a point on a picture and to trace a line drawing. He had difficulty in coordinating eye and hand movements. He could not trace the outline of a map (optic ataxia). The patient was told to look at an object held at his side and then to look at the examiner's nose and the patient was unable to do so (apraxia of gaze). The patient was unable to appreciate the significance of a picture or scene as a whole, although individual parts are recognized. When asked to pick the objects on the tray he picked four out of 10 objects (asimultanagnosia). Left–right orientation was not possible, and neither was identification of fingers. Muscle strength and bulk were normal in all four limbs. Sensation for primary modalities were normal, as was position sense. Romberg sign was negative. There was a sway on standing and a tendency to grab on to surrounding objects. The gait was abnormal with listing to one side. CT scan showed marked dilatation of the posterior horns bilaterally and adjacent cortical atrophy. There was no significant cerebellar atrophy (Fig. 1).

2.2. Case 2

A 79-year-old right-handed woman was seen with unsteadiness of gait and recurrent falls. She was independent with self-care. She had a history of hypertension and hypercholesterolemia, which were adequately controlled with medications, but no history of cerebrovascular accidents. She was a nonsmoker and consumed small amounts of alcohol occasionally.

Physical examination revealed no evidence of cardiac decompensation. The blood pressure was 160/90 mmHg. Her speech was fluent and she was oriented to time and place. Her concentration was normal and she scored 28/30 on the MMSE. The cranial sensation and reflexes were normal. She manifested optic ataxia and partial asimultanagnosia. Left–right orientation was possible, as was identification of fingers. The gait was unsteady with a wide base and a sway to the right. Tandem stance and tandem walk were not possible. The CT scan of the brain showed no evidence of a focal lesion. The left posterior horn of the lateral ventricle was enlarged with cortical atrophy.

2.3. Cases 3–12

These were nine men and one woman. The average age was 75 years (range 68–95 years). Comorbidities included two with hypertension (Cases 5 and 11) well controlled on medications and one of the two also had fits. Two of patients had a history of heavy drinking in the past (Cases 8 and 10). Two had diabetes (Cases 4 and 6) and were on oral hypoglycemic drugs. In two patients, the initial mistaken diagnosis was a progressive cerebellar disorder. Six of the 10 patients in the study group had one or more components of Balint's syndrome, namely optic ataxia, apraxia of gaze, and asimultanagnosia. Only two had all three components and four of the remaining eight had only optic ataxia. Five had gaze apraxia and five asimultanagnosia. Two had components of Gerstmann's tetrad, namely finger agnosia, right-left confusion, acalculia and agraphia. All had abnormal gait from a slight sway to gross ataxia. Muscle strength and bulk were normal in all. Cognition was normal in three patients (Cases 5, 6, and 11) and the remaining seven had mild (Cases 3, 4, 7, 10, and 12) or moderate impairment (Cases 8 and 9). All 10 had minimal to moderate dilatation of the sulci on the CT scan.

3. Discussion

The patients were selected because of recurrent falls on the basis of ventricular enlargement of the posterior horns of the lateral ventricles on visual inspection of the CT scans. Measurement techniques range from visual inspection to linear measurements to area and volumetric measurements.[4] Visual measurements are simple and easy to perform but the findings may be variable and inconsistent. Linear measurements on CT scans have been used to evaluate atrophic processes and ventricular enlargement and investigators have used ratios such as VBR.[4] Ventricular enlargement in our study was on visual inspection and the degree of ventricular enlargement evaluated by the VBR (Evans' index). An index with a ratio exceeding 0.3 was taken as indicative of ventricular enlargement (Table 1).

Furthermore, visual inspection can be used to designate atrophy by applying rating scales.[4] All our patients had minimal to moderate widening of the sulci on the CT scan and atrophy was characterized by the widened sulci. Koedam et al,[5] in order to assess the degree of atrophy in the clinical setting, developed a magnetic resonance imaging (MRI) visual rating scale for posterior atrophy. They described a four-point rating scale using MRI examinations, with 0 = no atrophy and 3 = severe end stage atrophy, based on the extent of widening of the sulci and volume loss of the gyri and an overall score on the presence of atrophy in sagittal, axial and coronal sections. We were unable to assess cortical atrophy reliably because MRI was not performed. Enlargement of the posterior horns of the lateral ventricles can be due to white matter degeneration or gray matter atrophy, in some instances the former more than the latter.

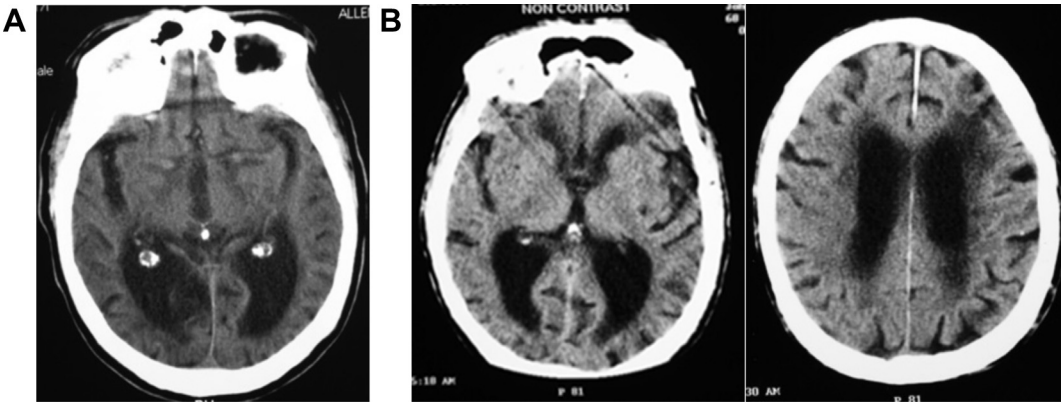

Fig. 1. (A) Computed tomography scan of Case 1 demonstrating symmetry of ventricular enlargement of the posterior horns and cortical atrophy and a small hypodense area medial to the right posterior horn. (B) Computed tomography scan of Case 6 demonstrating symmetry of ventricular enlargement of the posterior horns and a higher section showing hypodensities in relation to the lateral ventricles.