

Reversible dementia and gait disturbance as a result of polypharmacy

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DESCRIPTION

A 53-year-old man presented with gradually progressive difficulty in walking, excessive lethargy and forgetfulness of 3 months duration. There was a noticeable decrease in verbal spontaneity, initiative and occasional episodes of urinary incontinence. Treatment records revealed that patient was consulting three different specialists for his multiple comorbidities such as painful diabetic neuropathy, hypertension and insomnia. His medication pills were retrieved and it was ascertained that he was consuming amitriptyline and carbamazepine for last 4 years. Recently pregabalin along with escitalopram had been added to the regimen, that is, around 3 months previously, which corresponded with the onset of this neurological worsening. On examination, mini mental state examination (MMSE) score was 20/30 and his gait was broad based, shuffling with difficulty in turning. The timed get up and go score was 34 s. Sensory examination revealed bilateral distal symmetrical sensory motor neuropathy. Fundus showed bilateral mild non-proliferative diabetic retinopathy, no papilloedema.

Brain MRI (figure 1) showed prominent ventricular enlargement out of proportion to sulcal

atrophy, rounded frontal horn, temporal horn enlargement, periventricular signal changes and an Evan's index of more than 0.3 suggestive of normal pressure hydrocephalus (NPH). Patient underwent diagnostic lumbar puncture to rule out infection. The cerebrospinal fluid opening pressure was normal. Pregabalin and escitalopram were withdrawn from the treatment schedule. A follow-up of 6 months has shown consistent improvement in gait (timed get up and go score improved by 15 s) and cognitive functions.

NPH is a potentially reversible cause of dementia in elderly and this case highlights polypharmacy as its most likely cause due to its temporal relation. Reversible dementia and gait disturbance with valproic acid, hydrocephalus during natalizumab treatment and development of communicating hydrocephalus after infliximab infusion are similar recent reports.¹⁻³

Approximately 50% cases of NPH are idiopathic. A wide variety of conditions mimic the symptomatology of NPH such as depression, Alzheimer disease, Parkinson disease, multi-infarct dementia, frontotemporal dementia, Lewy body disease and corticobasal degeneration. These conditions can be definitely excluded by the characteristic imaging findings of NPH. Sulcal atrophy especially prominent medial temporal cortical atrophy (Alzheimer disease) and significant white matter ischaemic disease (vascular dementia) favours a diagnosis of hydrocephalus ex vacuo.

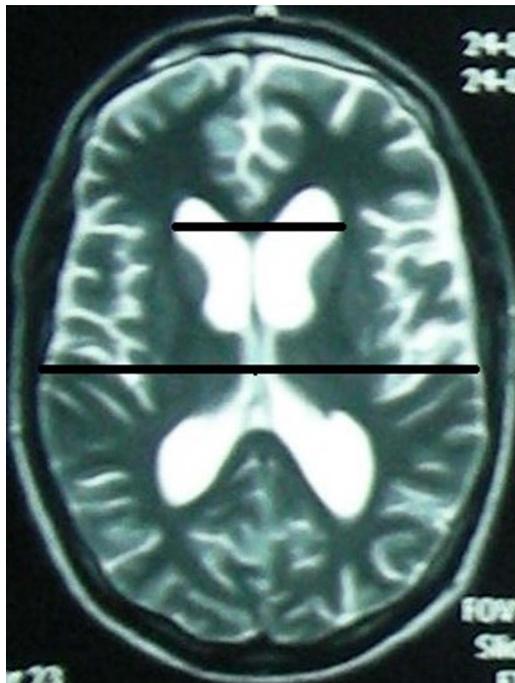


Figure 1 T2-weighted MRI of the brain depicting prominent ventricular enlargement out of proportion to sulcal atrophy, rounded frontal horn, temporal horn enlargement, periventricular signal changes and Evan's index of more than 0.3 suggestive of normal pressure hydrocephalus (NPH).

Learning points

- ▶ Physicians must always be alert to the adverse effects of polypharmacy in patients with multiple diagnosis and multiple prescriptions.
- ▶ The implications of polypharmacy are huge considering the rising number of young elderly with dementia syndromes and the diagnosis of such a reversible aetiology is rewarding.
- ▶ There is a desperate need to develop a comprehensive geriatric assessment so that a distressed patient does not fall in the trap of polypharmacy and its complications.

Competing interests None.

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