**Case Report**

**A case of hypertension with dementia: Common but underdiagnosed**

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**Abstract**

We report a case of a 55-year-old known hypertensive female who presented with features suggestive of dementia, which, on further workup unveiled background dyslipidemia and type 2 diabetes mellitus. An magnetic resonance imaging of the brain revealed findings suggestive of Binswanger’s disease. This is a discussion of this unusual disease and its presentation and the differentials of this presentation which may be encountered in general clinical practice.

**Keywords:** Binswanger's disease, blood pressure, memory loss, neurology, subcortical dementia

**Introduction**

Binswanger’s disease is a type of subcortical vascular dementia, which is a myriad of neurological symptoms and hence, is underdiagnosed in lieu of more common/well-known etiologies such as Alzheimer’s and Parkinson’s. This syndrome-like presentation also poses a variety of challenges for diagnosis as well as treatment, which is just supportive because it is a noncurable entity but progression and complications (thalamic hemorrhage in our case) could, of course, be reduced with early diagnosis and appropriate management of risk factors.

**Case Report**

A 55-year-old hypertensive female from Pauri Garhwal in the Indian state of Uttarakhand presented to the outpatient department of Internal Medicine with forgetfulness for 3 years. The forgetfulness of the patient had progressed from subtle changes like forgetting where she kept her money or other things, to her daily chores such as eating and bathing at the time of presentation. She also had problems in articulation, though perception and comprehension seemed normal. She also had a history of clumsiness of movements, slowness in performing daily tasks, lack of concentration, decreased attention span, and depressed mood.

She had been experiencing unprovoked falls from the last 2 years, the frequency of which had been progressively increasing, with each lasting around 1 min and not associated with the symptoms suggestive of a seizure episode. The consciousness remained intact throughout. There was no history of confusion after the episodes. She had a significant history of minor trauma due to falling at inappropriate places. There was no particular side on which the patient fell.

There was no history of abnormal involuntary movements, cranial nerve involvement, blurring of vision, sensory deficit, muscular weakness, tremors, bowel or bladder incontinence, or head injury. The patient has been a known hypertensive (uncontrolled from 2 years), on and off medication. Family history was not significant.

On general examination, blood pressure of the patient was 180/100 mm of Hg in the right brachial artery in supine position. Blood tests revealed hypercholesterolemia and type 2 diabetes mellitus. Head computed tomography showed no abnormality. Magnetic resonance imaging of the brain revealed findings suggestive of Binswanger’s disease. This is a discussion of this unusual disease and its presentation and the differentials of this presentation which may be encountered in general clinical practice.

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position. Other vitals were stable. Body mass index (BMI) was 25.7 kg/m² (overweight according to the WHO BMI classification). The patient seemed depressed.

On neurological examination, the patient was cooperative, conscious, and responsive to commands. Glasgow coma scale - E 4 V 5 M 6.

Mini-Mental Status Examination revealed inference of Mild cognitive impairment (score: 20/30). The patient exhibited unsteady gait. All the tests for coordination were abnormal on the left side. Intentional tremors and rebound phenomenon were present. All remaining cerebellar signs and signs of meningeal irritation were absent. Cranial nerves, motor, and sensory evaluation was within normal limits. All other systems seemed normal.

On laboratory investigations, the patient was found to have dyslipidemia and type 2 diabetes mellitus [Table 1]. Her hemogram, liver, and kidney function tests were within the normal range.

On fundoscopy, papilledema, diabetic, or hypertensive retinopathy was ruled out. On lumbar puncture, cerebrospinal fluid pressure was found to be normal. Serum homocysteine and serum fibrinogen levels were well within normal range.

Magnetic resonance imaging (MRI) of the brain showed a hemorrhagic infarct in the right thalamus with extensive chronic ischemic white matter lesions with multiple microhemorrhages in cerebellar and cerebral hemispheres, brainstem, and deep nuclei with a right-sided (2.3 cm × 2.1 cm) infratentorial arachnoid cyst with prominent ventricular system, sulcal spaces, and basal cisterns [Figures 1-3].

The differentials which were kept in mind while investigating the patient were Alzheimer’s, Parkinson’s disease, dementia with Lewy bodies, frontotemporal degeneration (Pick’s disease), and normal pressure hydrocephalus (NPH). All of these causes were ruled out based on the characteristic radiological findings of Binswanger’s disease and slight variation in clinical presentation.

In Alzheimer’s disease, the patient has a progressively severe memory loss and little or no gross locomotor disturbances until very late in the disease course.

Patients with Parkinsonism present with bradykinesia, rigidity, tremors, and shuffling gait. The patients have mild memory loss and cognitive impairment. Motor disturbances occur early in the course of the disease.

Both NPH and Binswanger have a similar clinical presentation, but NPH progresses gradually, and Binswanger disease progresses in a stepwise fashion with more commonly associated vascular complications.

Table 1: Relevant laboratory investigations

| Test                        | Patient value | Reference range |
|-----------------------------|---------------|-----------------|
| Lipid profile               |               |                 |
| Total cholesterol           | 280 mg/dl     | <200 mg/dl      |
| Serum triglyceride          | 187 mg/dl     | <150 mg/dl      |
| HDL                         | 48 mg/dl      | 40-60 mg/dl     |
| LDL                         | 148 mg/dl     | 100-129 mg/dl   |
| Plasma glucose              |               |                 |
| Random                      | 229 mg/dl     | <140 mg/dl      |
| Postprandial                | 155 mg/dl     | <140 mg/dl      |
| Fasting                     | 101 mg/dl     | 70-106 mg/dl    |
| HbA1C                       | 6.6%          | 4%-6.2%         |

LDL: Low-density lipoprotein; HDL: High-density lipoprotein; HbA1c: Glycated hemoglobin

The patient was advised lifestyle modifications along with antihypertensives (Amlodipine 5 mg), oral hypoglycemic agents, lipid-lowering drugs for control of her risk factors for preventing any further similar episode of complication on a preexisting debilitating disease. In addition to it, cholinesterase inhibitors (Donepezil 10 mg) and multivitamin supplements.

The patient came for a follow-up visit after 15 days. She improved symptomatically for giddiness and depressed mood, but the remaining symptoms persisted. Her blood pressure levels were still on the higher side (150/90 mm Hg), therefore, dose of Amlodipine was increased to 10 mg. Blood glucose and lipid
profile were within normal range. Hence, all remaining drugs were continued in same doses. She was referred to psychiatry for counseling regarding her depression. She was explained about the nature of her disease, prognosis and was encouraged to control the risk factors for preventing future complications.

**Discussion**

Binswanger disease, also known as subcortical arteriosclerotic encephalopathy,[2] is a form of vascular (small vessel) dementia. Vascular dementia is the second-most common type of dementia.[1] Binswanger disease usually affects males and females in equal numbers[3] and occurs mostly after 50 years of age.[1]

The entity is defined by widespread, microscopic incomplete areas of infarction in the deep layers of white matter in the brain due to critical stenosis of medullary arterioles and hypoperfusion.[1] The damage in this particular disease is irreversible since it is caused by thickening and narrowing of arteries (Arteriosclerosis) all over the body but especially in the brain.

Risk factors include advanced age, hypertension, atherosclerosis, smoking, obesity, diabetes mellitus, hyperhomocysteinemia, hyperfibrinogenemia, hereditary disease such as cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy, cerebral amyloid angiopathy and other conditions that can cause cerebral hypoperfusion.[1,4] Thus, it is actually a multifactorial clinical syndrome of vascular dementia with no specific cause.[4]

The symptoms associated with it are related to the disruption of subcortical neural circuits which cause cognitive and personality changes.[1] Due to the vascular etiology, the symptoms and signs associated with Binswanger’s disease may worsen suddenly due to stroke or hemorrhage, stabilize and then improve for a brief time, but the patient’s overall condition continues to worsen as the blood vessels become increasingly obstructed.[6]

The disease can be diagnosed with radiological imaging like MRI/computed tomography showing changes in white matter, multiple infarcts in deep cortical and subcortical structures.

Till now there has been no specific treatment for this disease. The treatment is more or less symptomatic, aiming at the reduction of risk factors to slow the disease progression.[1,5] Recent drug trials with Donepezil have shown improved cognition and stabilization of global functioning and behavior.[1] Patients with depressed mood may be put upon selective serotonin reuptake inhibitors.[5] Atypical antipsychotics like risperidone can be used in agitated, aggressive patients.[5]
Conclusion

This particular case may broaden the horizons for signs and symptoms of Binswanger’s disease which would reduce the chances of underdiagnosis of this entity at primary care level.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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