Original Article

A Case Report Of Binswanger's Disease

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DOI: 10.47750/pnr.2022.13.S02.252

Abstract

Background: Binswangers disease is a progressive neurological disease caused by atherosclerosis and thrombo-embolism affecting the white matter and blood vessels affecting the deep brain structures (basal ganglia and thalamus). Depending on the vascular etiology, the symptoms and physical effects as-sociated with Binswanger stroke may suddenly worsen, stabilize, and then improve briefly, but the patient's general condition continues to improve as they continue to enter contact with the occluded vessel. High blood pressure, smoking, high cholesterol, heart disease, and diabetes are risk factors for Binswanger's disease. Rare genetic disorders such as autosomal dominant cerebral artery disease (CADASIL) and subcortical infarct and leukoencephalopathy also cause Binswagger disease. Diag-nosing Binswangers disease necessitates using a multimodal approach. More specific tests will be-come accessible in the coming years when novel pathophysiological mechanisms are revealed. It is a complex syndrome caused by variety of factors rather than a single disease.

Methods: Seventy years old male presented with complaints of inability to walk, dysarthria, dysphagia, abulia and apathy. He was a known case of systemic hypertension, CAD and Parkinsonism on treatment little supportive for the diagnosis of Binswangers disease.

Results: This case describes a clinical approach to Binswangers disease with the finding helpful in consistent with MRI brain findings, CSF flow study and new novel biomarker.

Conclusion: A Rare disease with nil significant approach still but a new biomarker is available to come into a conclusion of Binswangers disease. Further investigations will become available in the years to come in order to better treat these patients

Keywords: Binswangers Disease, Neuroinflammation, white matter, Dementia, Leukoencephalopathy

INTRODUCTION

White matter destruction causes Binswangers disease, a kind of small artery vascular dementia. Subcortical leukoencephalopathy and subcortical arteriosclerotic encephalopathy are other names for it (SAE). A multitude of conditions, including chronic hypertension and advanced age, can cause white matter atrophy. Memory and intellectual function loss, as well as mood fluctuations, are hallmarks of this condition. These alterations have an impact on the brain's executive functioning. It usually appears between the age of 54 and 66, with mental impairment or stroke as the initial signs. Evaluating and treating Binswangers disease is challenging. Otto Binswangers originally characterised it in 1894, and Aloiz Alzheimer coined the term "Binswangers sickness" in 1902. Jerzy Olszewski, on the other hand, is credited with much of the modern-day research into this disease, which began in the 1960s.

CASE REPORT

70 years male presented with complaints of inability to walk for 2 years, but able to feel sensation associated with no numbness, burning sensation, breathlessness at rest, grade IV on and off, relieved on lying down position, no diurnal, seasonal variation, dribbling of urine, urgency, frequency for 6months.Patient also presented with dysphagia, dysarthria, apathy and abulia for 6 months. Patient was found to have no headache, loss of consciousness, seizures, trauma, ENT bleed, memory loss, no family history of Parkinsonism with no known comorbidities. Patient is a known alcoholic for 35years, discontinued last 2years, Known case of CAD for 5years on antiplatelets, Known case of dyslipidemia for 2years on statins, Patient had a previous history of TIA 3 years back and was on medications. Known case of systemic hypertension for 2years on antihypertensives, Known case of Parkinsonism for 2years. On examination- pt was conscious, oriented, afebrile. On Systemic examination,

No notable findings observed on examination of cardiovascular, respiratory and per abdominal examination. On Central nervous system examination, Bradykinesia, Magnetic gait, dysarthria with extrapyramidal signs. ECG showed normal sinus rhythm. T wave inverson in ll, lll, avf, v2-v6.Viral serology done and Elisa for HIV turned out to be positive. 2D echo showed ejection fraction 50% (mild LV dysfunction), coronary artery disease, Hypokinesia of anterior, anterolateral, anteroseptal wall. Cardiology opinion sought, advised to start on beta blocker, and diuretics.

Lipocalin 2, a bio marker is done and found to be elevated. Neurologist opinion sought in view of MRI brain report showing white matter pallor and rarefaction, [FIG1B AND 1C]multiple chronic lacunar infarcts in right thalamus, B/L ganglio capsular, B/L fronto parietal and right temporal occipital regions with mild exvacuo dilatation of left ventricle and

enlarged ventricles.[fig 1A] Multiple tiny areas of blooming focus in B/L cerebellar, midbrain, left occipital and bilateral frontal to parietal region - microbleed suggestive of hypertensive encephalopathy. CSF flow study to rule out normal pressure hydrocephalus. In CSF Analysis increased albumin index without oligoclonal bands was evident .Thyroid profile found to be normal for this patient. Differential diagnosis can be vascular Parkinsonism, Binswangers' disease, non pressure hydrocephalus, Parkinson plus syndrome. Radiologist opined that, the diagnosis of normal pressure hydrocephalus can be ruled out after evaluating the MRI. Lipocalin-2 marker sent to rule out Binswangers disease and it turned out to be elevated. Hence arrived at a diagnosis of vascular dementia (Binswangers disease). Urologist opinion sought in view of overactive bladder. Advised to start on Tab mirabegron 25 mg only at night for 1 month. Neurologist review done. UPDRS study scale found out to be165 score (before syncopa) and 126 (after syndopa) .Patient was followed up for 10 days with syndopa and found dementia symptomatically better.

DISCUSSION

Based on clinical symptoms, risk factors, general and neurological examination, computed tomography and MRI findings, and a thorough differential diagnosis, [1]Binswangers illness was proposed as a diagnosis for our patient. More than 20 years ago, Benett and Caplan established a diagnostic criteria for Binswangers disease. Since then, the pathophysiology of the disease has improved, and in complex instances, auxiliary tests can be employed in addition to the normal clinical features and imaging. [2]Changes in CSF biochemistry may indicate the neuroinflammation that occurs in small vessel disease.[5] Neuroinflammation induces a breakdown in the blood-brain barrier, resulting in increased permeability on the one hand and dramatic alterations in glial cell protein and cytokine expression patterns on the other. Increased albumin levels in the blood may reflect these

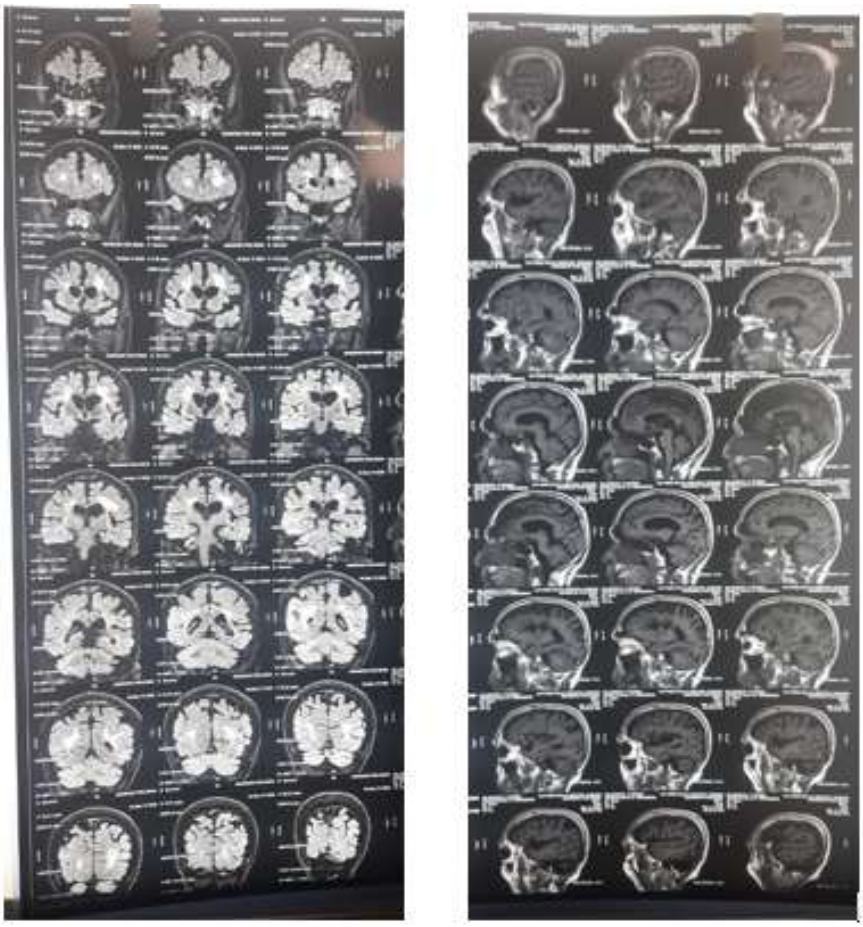

Fig 1: A and B showing multiple chronic lacunar infarct and mild blooming focus

changes. Greater amounts of inducible matrix metalloproteinases such as MMP-3 and MMP-9, as well as CSF (due to altered protein expression).[4,7] Lipocalin 2 is a new biomarker that has been found to have increased levels in Binswangers disease patients (LCN2).[5] LCN2, is a glycoprotein that plays a role in neurovascular damage in patients with vascular disease. It showed results, with patients with

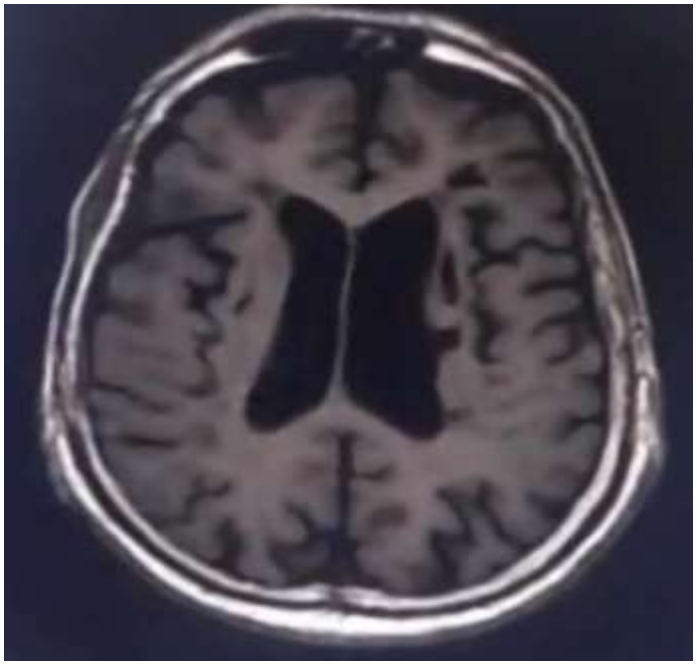

Fig 2: C showing exvacuo dilatation of left ventricle

vascular dementia having higher levels than those with huntings disease or other types of dementia. The clinician can use MRI diffusion tensor imaging (to assess white matter tract integrity) or dynamic contrast enhancement MRI (to detect blood-brain barrier disruption) to make a diagnosis. These imaging techniques are unproven and untested in the real world. It's vital to remember that a diagnosis can't be made merely on the basis of CT or MRI imaging; it needs to be backed up by a comprehensive clinical evaluation. Binswangers disease requires a multi modal approach to diagnosis. None of the biomarkers are sufficient to diagnose the condition on their own, but in patients with [8] cognitive impairment and neurological indications of questionable or unknown aetiology, using clinical data in conjunction with imaging and auxiliary testing can be helpful. There is a scarcity of information about the disease's prognosis. Binswanger's disease is currently incurable, and there is no cure. There are no particular clinical research addressing therapeutics for Binswangers disease, despite the American Heart Association (AHA) publishing therapy guidelines for people with vascular cognitive impairment. Blood pressure control, antiplatelet medication, and statins all aid in the prevention of secondary strokes and the slowing of white matter lesions. Dietary adjustments (salt reduction), physical exercise, physical therapy, and rehabilitations all play a key role in the treatment of these people, resulting in improved quality of life, as well as health advantages. It is often difficult to differentiate between Binswangers disease and Alzheimer's disease clinically. Memory impairment and dysfunction in cognition takes place in both, but predominantly in the initial phase of the disease strokes, hypertension, and asymmetric motor and sensory deficits, points to BD. Predominant impairment in memory without associated apathy, confusion, or character change was uncommon in analysis of our case report. It is helpful in differentiating Alzheimer's disease and BD by looking for the presence of hypodensity in white matter with infarcts in cornices and lacunar region.[3] Normal pressure hydrocephalus[NPH] presenting along with mental instability, when not associated with incontinence and gait disorder, simulates BD. Mild white matter low density, sometimes seen surrounding the enlarged frontal horns in NPH,46 simulates the white matter hypodensity of BD. Nevertheless, enlargement of ventricles is generally less pronounced and hypodensity in white matter more extensive in BD.A more detailed [6] clinicopathologic correlation is required to find out the exact cause of the characteristic white matter lesions, with serial sectioning of small vessels supplying the gliotic regions, experimental investigation of chronically hypertensive animals, and perhaps PET scan of patients with postulated BD.

CONCLUSIONS

Binswanger's disease is a complex neuropsychiatric disease, and its pathophysiology is only partially understood. Treatment options available are only symptomatic which includes antidepressant to treat depression, anti platelet to eradicate thromboembolism, statins to reduce atherosclerosis, anti hypertensives to treat hypertension. As new pathophysiological mechanisms are revealed, other tests will become available in the years to come and also novel therapies will specifically target these mechanisms (inflammation, arterial stiffness, and clearance of cerebral waste) in order to better treat these patients.

ACKNOWLEDGMENTS

The authors are thankful to the Department of General Medicine, Sree Balaji Medical College, Chennai, Tamilnadu, India for the continuous support towards this research study.

ETHICAL CONSENT

Patient included in the study provided informed consent

FUNDING

No funding was used to conduct current study.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

REFERENCES

1. Binswanger's disease: biomarkers in the inflammatory form of vascular cognitive impairment and dementia Gary A Rosenberg 1 PMID: 28902409 PMCID: PMC5849485 DOI: 10.1111/jnc.14218
2. Binswanger's disease: toward a diagnosis agreement and therapeutic approach Branko N Huisa 1, Gary A Rosenberg PMID: 25201523 PMCID: PMC4545265 DOI: 10.1586/14737175.2014.956726
3. Jacobs L, Kinkel W: Computarized axial transverse tomography in normal pressure hydrocephalus. Neurology 1976;26: 501-507
4. Biomarkers identify the Binswanger type of vascular cognitive impairment Erik Barry Erhardt 1 2 , John C Pesko 1 , Jillian Prestopnik 3 , Jeffrey Thompson 3 , Arvind Caprihan 2 PMID: 29513153 PMCID: PMC6681525 DOI: 10.1177/0271678X18762655
5. Binswanger's disease: toward a diagnosis agreement and therapeutic ap-proachBranko N Huisa 1, Gary A Rosenberg PMID: 25201523 PMCID: PMC4545265 DOI:10.1586/14737175.2014.956726
6. Clinico-pathology and differential diagnosis of Binswanger's disease Tibor Kovács 1, Imre Szirmai, Mátyás Papp PMID: 15887411
7. Lipocalin 2 regulates iron homeostasis, neuroinflammation, and insulin resistance in the brains of patients with dementia: Evidence from the current literature Daejin Lim 1, Jae-Ho Jeong 1, Juhyun Song 2 PMID: 33945675 PMCID: PMC8265939 DOI: 10.1111/cns.13653
8. Vascular cognitive impairment and dementia Philip B Gorelick 1, Scott E Counts 2, David Nyenhuis 3 PMID: 26704177 PMCID: PMC5232167 DOI: 10.1016/j.bbadis.2015.12.015