

Case Report

Leukoaraiosis in a 54 Year Old Nigerian Male: A Case Report

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Abstract: Background: Leukoaraiosis is a chronic atherosclerotic disease with an abnormal periventricular and cerebral white matter disease. Hypertension and old age were reported as the most-important risk factor for leukoaraiosis. Other risk factors like diabetes mellitus, abdominal obesity, hyperlipidaemia, tobacco use, alcohol abuse and chronic kidney disease has been reported. The aim of this case report is to highlight leukoaraiosis in a 54 year old Nigerian who presented with neurological manifestation. Case information: we report a case of a 54 year old male with long standing hypertension presenting with features of leukoaraiosis. Result: the diagnosis was confirmed with MRI which revealed scattered nodular white matter T2 and FLAIR hyperintensities in the cerebral cortices with fluid restriction on diffusion weighted imaging. Conclusion: detailed identification and control of risk factors are the main stay of treatment for leukoaraiosis. Hypertension was managed via proper education on dietary and life style modifications, medications and regular follow-up in clinics.

Keywords: Leukoaraiosis, Old Age, Hypertension, Inflammation, Brain MRI, Antihypertensive Drugs

1. Introduction

Leukoaraiosis also known as cerebral white matter disease, is a pathological appearance of the brain white matter, which has long been believed to be caused by perfusion disturbances within the arterioles perforating through the deep brain structures [1].

The term "leukoaraiosis" was coined in 1986 by Hachinski, Potter, and Merskey as a descriptive term for rarefaction ("araiosis") of the white matter, showing up as decreased density on Computed Tomography (CT) scan and increased signal intensity on T2/FLAIR sequences (white matter hyperintensities) performed as part of MRI brain scans [3, 4].

Leukoaraiosis is a chronic atherosclerotic disease, and hypertension is the most-important risk factor. Hypertension is a common cause of micro and macro-vascular related adverse cardiovascular complications globally [2]. Neurological disorders including encephalopathy, stroke and dementia have been reported as common complications of hypertension. Leukoaraiosis has been noted as an important prognostic factor for patient with stroke, cognitive impairment, dementia, and also impart survival of these patients [2].

Other risk factors of leukoaraiosis include advancing age, especially those 60 years and above, type 2 diabetes mellitus, abdominal obesity, hyperlipidaemia, hyperhomocysteinaemia, carotid stenosis, history of stroke, tobacco use, alcohol abuse, female gender and chronic kidney diseases have been reported [5, 7]. Studies have also demonstrated a relationship between inflammation and leukoaraiosis [16].

Clinical features may include worsening headache, loss of balance and coordination, dizziness, confusion, difficulty with speaking, hemiparesis, and loss of vision. Depression, restlessness and cognitive impairment can occur [5].

Diagnosis is confirmed with neuroimaging showing as periventricular and bilateral patchy white matter hypodensities on Computed Tomography Scan or hyperintensities on T2/FLAIR sequence on Magnetic Resonance Imaging Scans [3].

Management begins with early identification of abnormal increase in blood pressure and treatment of various risk factors that contribute to small vessel damage in the brain. This helps to prevent complications such as ischaemic stroke, cognitive impairment, and established dementia.

The aim of this case report is to highlight leukoaraiosis in a

54 year old Nigerian who presented with neurological manifestation.

2. Case Summary

Mr O. U a 54year old male trader, who is known to live with hypertension for 5year, however not adherent to either prescribed medications and follow up care. He presented to our emergency room with a headache of 4 days duration and irrational talk of 2 days duration; and an impairment of consciousness of 12hrs duration.

Headache was insidious in onset, generalized, throbbing, initially it was mild but later became severe, associated with neck pain, blurring of vision and tinnitus. There was no aggravating factor but is relieved temporally by analgesics. He did not have fever, photophobia, vertigo, nausea, vomiting, seizures or weakness of the limbs.

Two days prior to presentation, he started talking irrationally, subsequently became restless, confused with worsening conscious state the following day. He did not have faecal or urinary incontinence. Neither him nor anyone in the family had similar illness or stroke, transient ischaemic attack or diabetes mellitus. He is married and has 4 children who are alive and well. He does not take recreational drugs, alcohol or tobacco.

Examination showed a middle aged man, restless, afebrile, not pale, anicteric, acyanotic, no peripheral lymphadenopathy, mildly dehydrated and no pedal edema.

He was drowsy but easily roused with a Glasgow coma scale of 13. He had nuchal rigidity but kernigs and Brudziski's signs were equivocal. Both pupils were equal, constricted and reacted sluggishly to direct light. There was no differential abnormality detected in both the cranial or peripheral motor innervations assessed.

The pulse was 76 beats per minute, full volume, regular and synchronous with other peripheral pulses, the arterial wall was thickened with presence of loco-motor brachialis. The blood pressure was 190/110mmHg supine, jugular venous pressure was not raised, apex beat was displaced but not heaving and only 1st and 2nd heart sound were present, no murmur. There was no abnormality detected on examination of other body system.

His brain MRI (figure 1) revealed scattered nodular white matter T2 and FLAIR hyperintensities in the cerebral cortices with fluid restriction on diffusion weighted imaging. No mass lesion or focus of haemorrhage was seen. There are inflammatory changes in the right maxillary sinus. Radiological diagnosis of bilateral cerebral ischaemic white matter disease (leukoaraiosis) and right maxillary sinusitis were made.

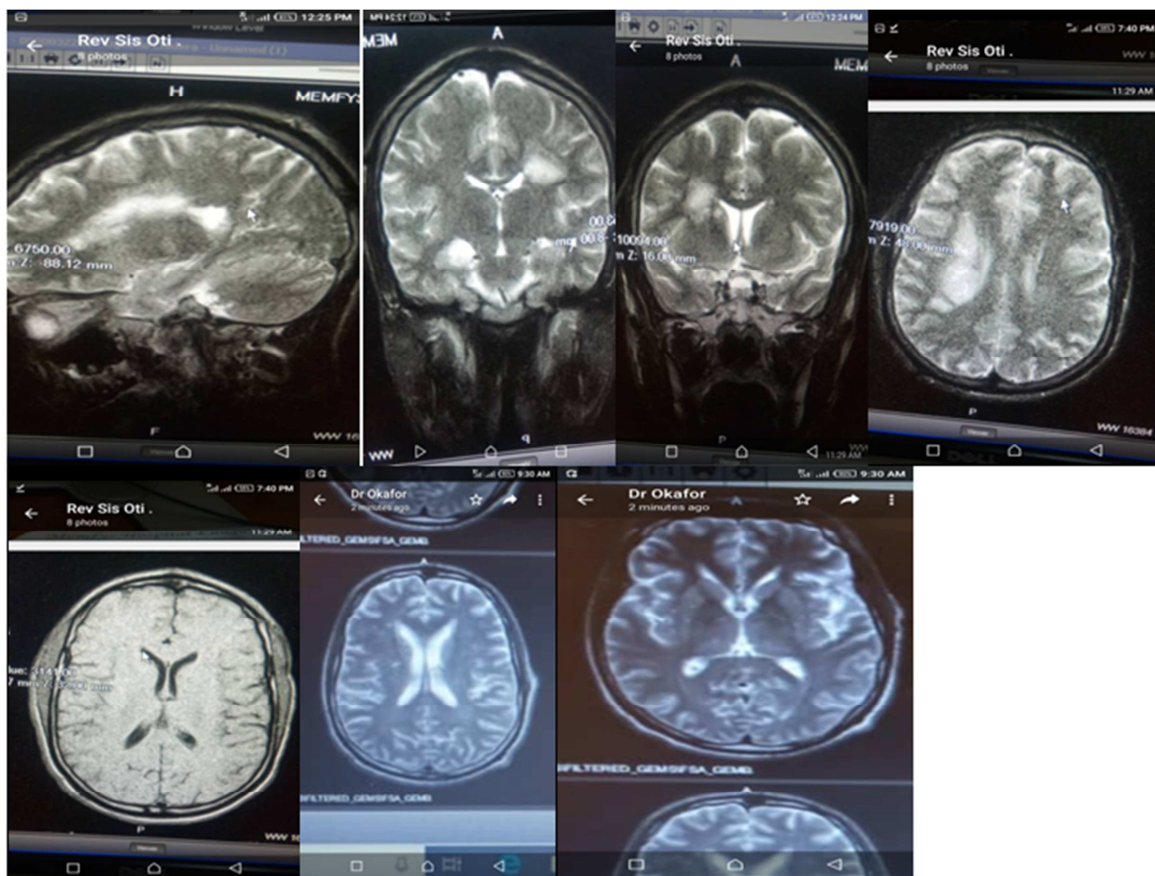


Figure 1. Brain MRI showing scattered nodular white matter T2 and FLAIR hyperintensities in the cerebral cortices with fluid restriction on diffusion weighted imaging.

Full blood count showed leukocytosis ($13.01 \times 10^9/L$) and neutrophilia (78%) with mild toxic granulation; Haemoglobin

was 14.7g/dl and peripheral blood film was normal. Serum biochemical analysis was normal, screening for hepatitis BsAg, hepatitis C antibodies and human immunodeficiency virus were negative. He had microscopic haematuria and urine culture yielded significant growth of *Pseudomonas aureginosa* and sensitivity was noted.

He was admitted as a case of subarachnoid haemorrhage with cerebrospinal meningitis as differential diagnosis.

He was nursed as unconscious patient and antibiotics, antihypertensive and mannitol were commenced. He responded well to management, and was discharged to outpatient care after 10 days of hospitalization on antihypertensives, antiplatelets, statins and multivitamins. Patient and caregiver were also properly educated on diet, life style modification including regular exercise, adherence to his medication and to be regular to follow up.

3. Discussion

The descriptive term leukoaraiosis, refers to white matter ischaemic lesion which causes an abnormal periventricular and cerebral white matter disease (CWMD). It may be bilateral and either patchy or diffuse areas of hypodensities on CT scan or hyperintensities on T2-weighted MRI [4]. These findings were consistent with our patient whose brain MRI showed scattered nodular white matter T2 and FLAIR hyperintensities in the cerebral cortices with fluid restriction on diffusion weighted imaging.

Hypertension and age especially middle age and elderly were reported as the main risk factors for development of cerebral WMD [6]. Our patient is hypertensive which was poorly managed and in his middle age.

Hypertension is one of the leading causes of mortality and morbidity worldwide. Its prevalence increases with age and is estimated to affect 65% of those ≥ 60 years-old [8]. Recently, the 2013 European Society of Hypertension/European Society of Cardiology Hypertension Guidelines defined a universal target of $<140/90$ mm Hg for all patients, except the elderly population with target of $<150/90$ mm Hg for those ≥ 80 years-old [9]. The American Heart Association, American College of Cardiology, Centers for Disease Control and Prevention, American Society of Hypertension, and the International Society of Hypertension also supported the treatment goals associated with this guideline [10, 11]. Therefore, good blood pressure control is vital to preventing complications and mortalities from hypertension. Our patient blood pressure control was not optimal with poor adherence to medication and follow up regime. Health education and improvement in patient socioeconomic status will have positive outcome on hypertensive patient including prevention of complications like leukoaraiosis.

Other reported risk factors associated with the development of leukoaraiosis such as type 2 diabetes, abdominal obesity, hyperlipidaemia, hyperhomocysteinaemia, history of stroke, smoking, alcohol abuse, and chronic kidney diseases were not present in our patient [5, 7].

Also, poor control of risk factor and possibly a subtle brain

inflammation may have contributed to early presentation of the disease in our patient. Laboratory evidence of systemic inflammation in our patient was typified by elevated white cell count with neutrophil predominance. Lammie G. A in a study of Hypertensive cerebral small vessel disease and stroke reported that systemic inflammation is a risk factor for development and progression of cerebral white matter disease [16]. Our index patient presented with features of inflammatory response demonstrated by the presence of fever and neutrophilic leukocytosis, this could explain the clinical presentation of the patient.

Although the pathogenesis of leukoaraiosis is not well understood, however, the current hypothesis concerning the association between hypertension and leukoaraiosis (cerebral white matter lesion) shows that long-standing hypertension causes lipohyalinosis of the media and thickening of the vessel walls, with narrowing of the lumen of the small perforating arteries and arterioles that nourish the deep white matter. The perforating vessels, which originate in the cortical and leptomeningeal arteries, have a relatively poor anastomotic system, which makes the white matter particularly vulnerable to cerebral ischaemia [12]. The lesion is usually bilateral like in our index patient.

Our patient had marked elevation in blood pressure at presentation which was controlled with combination of antihypertensive drugs. Studies have shown that poorly controlled hypertension plays a significant role in development and extension of cerebral white matter lesion [13, 14]. A magnetic resonance imaging (MRI) sub-study of the randomized PROGRESS trial of blood pressure control with ramipril versus placebo in normotensive and hypertensive subjects with cerebrovascular disease found that the mean total volume of new white matter ischaemic lesion was significantly reduced in the active treatment group compared with placebo [15]. Thus controlling blood pressure of the index patient contributed to the notable recovery of the patient.

Leukoaraiosis has been reported as an independent and an important prognostic factor in patients with stroke, cognitive impairment, dementia, and also their survival [16].

4. Conclusion

Leukoaraiosis though rare occurs in our populace especially in the late middle age and elderly hypertensive. Diagnosis is mainly by neuroimaging especially CT scan or MRI demonstrating characteristics intra-cerebral white matter lesions.

Early identification and control of risk factors especially hypertension and cerebral inflammatory diseases will help to prevent disease development and subsequent progression.

Antihypertensive medications are highly recommended as various studies have shown that it slows down disease progression. Preventive measures such as dietary and life style modification, including exercise and weight loss for obese patients are very beneficial.

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