Adepoju ST, et al., J Clin Stud Med Case Rep 2024, 11: 252
DOI: 10.24966/CSMC-8801/1000252

HSOA Journal of Clinical Studies and Medical Case Reports

Case Report

Multiple Sclerosis in a 40 Year Old Woman in LAUTECH Teaching Hospital, Ogbomoso, South West Nigeria: a case report

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Abstract

Multiple sclerosis is an autoimmune demyelinating disease with affectation of the nervous system. The disease is rare among Africans, partly due to lack of widespread investigative tools.

Methodology

This is a case report of a 40-year-old Yoruba female clergy, who has never left Nigeria who presented with recurrent headache and progressive loss of vision with intermittent improvement of symptoms. No weakness in the limbs or loss of splintering control.

Results

Magnetic Resonance Imaging (MRI) showed hyperactive intensity in the corona radiata, centrum ovale and periventricular areas, with bilateral optic nerve atrophy and idiopathic intracranial hypertension.

This calls calls for increased index of suspicion and availability of adequate investigative tools in making such diagnosis and prompt management.

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Citation: Adepoju ST, Orugun ST, Kolo-manma K, Oyedepo VO, Alabi A, et al. (2024) Multiple Sclerosis in a 40 Year Old Woman in LAUTECH Teaching Hospital. Clin Stud Med Case Rep 11: 252.

Received: November 04, 2024; **Accepted:** November 20, 2024; **Published:** November 30, 2024

Keywords: Multiple Sclerosis; Autoimmune; Demyelinating; Idiopathic; Nigeria; Neurology; Clinic

Introduction

Multiple Sclerosis is a demyelinating autoimmune disease affecting the nervous system with debilitating course and significant impact on patient's life. It is believed multiple sclerosis occurs more commonly in the western world or in people who have emigrated to the west and quite rare in black Africa & Africans [1]. This report however considers Multiple sclerosis in a 40-year-old woman of Yoruba extraction from a suburban town in southwest Nigeria who has never travelled beyond the shores of Nigeria.

Case Summary

40 year old Yoruba female clergy who resides in Ogbomoso, Oyo State, Nigeria, who presented with loss of vision of 4 weeks and recurrent headaches of 3 weeks duration. She was in her usual state of health until 4 weeks prior to presentation when she developed blurring of vision, which started insidiously and progressively worsened after about 1 week. There was associated inability to see far objects and writings initially. This however did not impair her daily activities. She had not had similar occurrence previously. No redness in the eyes, no itching, no excessive tearing from the eyes. Her eyesight worsened 6 days prior to presentation and she could not view texts on her cell phone and was noticed to miss her steps. At presentation, she could not perceive light in either eye. No prior history of transient loss of vision. No history of trauma to the eyes. No eye pain, no eye discharge, no history of photophobia or diplopia.

She also developed recurrent headaches 3 weeks prior presentation. It was generalized, non-throbbing, no feeling of bands around the head, no tearing. Headache was said to be relieved by Tabs Acetaminophine.

No history of neck pain, no history of vomiting, no differential limb weakness, no facial deviation, no drooping of the eye lids.

She was diagnosed of hypertension in pregnancy in her first confinement about 11 years prior to presentation. No history suggesting hypertension after that pregnancy. She lost a pregnancy (her third confinement) at 8 months and was delivered of a still born. Her last confinement was adversely uneventful and wasn't told she had high blood pressure. She was not diagnosed of Diabetes Mellitus.

No previous history of transient ischaemia attack (TIA)or stroke, no history of fever. No history of chest pain or palpitations.

She had presented to several clinics where she had some medications. At presentation, her BP was 230/180mmhg.

There was no family history of hypertension or diabetes mellitus.

No family history of blindness or stroke. She neither smoked cigarette nor took alcohol. There was no known drug allergy.

General Examination

A young woman, conscious, afebrile, not pale, not icteric, not dehydrated, not cyanosed, no peripheral lymph node enlargement, no finger clubbing, and no pedal oedema.

Nervous System

Fully conscious, oriented in time place and person,Speech was coherent not slurred,

Pupils were bilaterally round 6mm dilated, unreactive to light or accommodation. There was cranial nerve (CN) III palsy,no facial nerve palsy. No signs of meningeal irritation, no asterixis .Tone was normal across all joints. Power was full across all limbs. Deep tendon reflexes were normal. Plantar responses were extensor bilaterally.

Cardiovascular System

Pulse rate 120bpm, regular, normal volume thickened arterial wall.

BP- Right arm 202/120mmhg left arm 220/132mmhg (the patient had taken 30mg of Nifedipine few hours before this measurements were taken).

Jugular Venous Pressure was normal. The apex beat was at the 5th intercostal space on the left, lateral to the mid clavicular line and it was heaving. Heart Sounds were S1 and S2 with a loud A2.

Chest

RR – 18cycles per minute. SpO2 was 100% in room air with equal chest expansion. Percussion note was resonant. Breath sounds were Vesicular.

Abdomen

Abdomen was full, it moved with respiration. There was no area of tenderness. The liver was 6cm palpable below the right coastal margin. It was smooth, non-tender. The liver span was 14cm. The spleen was not palpably enlarged. The kidneys were not bimanually palpable. There was no ascites. Her bowel sounds were normoactive.

Fundoscopy

Blurred Disc margins (initially done by the physicians). Initial assessment – Hypertensive emergency likely malignant phase Hypertension with hypertensive retinopathy KIV intracranial Space occupying lesion with possible chiasmata compression.

Plan – FBC

- Electrolytes, urea and creatinine
- Urinalysis
- ECG
- Abdominal scan
- Cranial MRI

Blood pressure was controlled using IV labetalol 100mg in infusion and subsequently Tabs Nifedipine 20mg twice daily, Tabs Hydrochlorothiazide 25mg daily and telmisartan 80mg daily.

Ophthalmology Review

Ocular Examination

Right eye	Left eye
NPL	NPL
Dilated unreactive	Dilated unreactive
Clear media	Clear media
Pale disc, not cupped, indistinct nasal and superior disc margins	Pale disc, not cupped, indistinct nasal and superior margins
Silver wiring. Arteriovenous nicking and attenuated vessels on the disc	Wide spread exudates temporally localized retinal haemorrhages
	Macular oedema
Intraocular pressure- 15mmHg	Intraocular pressure 14mmHg

Assessment

Grade 4 hypertensive retinopathy KIV possible Optic nerves compression from an Intracranial space occupying lesion.

Results of investigation

Urinalysis

Bilirubin- negative

Urobilinogen – normal

Ketones negative

Glucose negative

Protein negative

Blood negative

Nitrite negative

pH 6.0

Specific gravity 1.025

Leucocyte negative

Full Blood Count

PCV 33%

MCV 92fl

MCH 35pg

WBC 14,800/cmm

NEUTROPHILS 86%

EOSINOPHILS 1%

LYMPHOCYTES 12%

MONOCYTES – 1%

PLATELET- 191,000/cmm

Electrolytes, Urea And Creatinine

HCO_3^- 26 mmol/l

Cl^- 97 mmol/l

Na^+ 132mmol/l

K^+ 3.2mmol/l

Urea 5.2mmol/l

Creatinine 88µmol/l

ECG at admission showed

- Sinus tachycardia
- Left atrial enlargement
- LVH with left ventricular strain pattern.

Random blood glucose(RBG)

at presentation - 4.6mmol/l

Report Of MRI

Multiple non-enhancing punctate T2/FLAIR hyperintense and T1 isointense foci bilaterally in the deep white mater of the cerebral cortex, but they were found majorly in the centrum semi-ovale and corona radiata. Similar widespread punctate and diffused hyperintense lesions were also seen within the basal ganglia, both thalami, midbrain, pons, the middle cerebellar peduncles and the cerebellar hemispheres on T2. The same lesions were hypointense on T1 and hyperintense on T2 FLAIR.

On T2 sagittal plane, ovoid-shaped high signal intensity lesions are seen distributed along the axis of the medullary veins, perpendicular to the body of the lateral ventricle (Dawson's fingers).

However, no intracranial mass was seen, and the optic chiasm was preserved.

Both optic nerves appeared thinned out measuring 2.3mm and 0.9mm on the right and left respectively (normal range: 4.8-6.2mm) with their intracranial parts surrounded by cerebrospinal fluid.

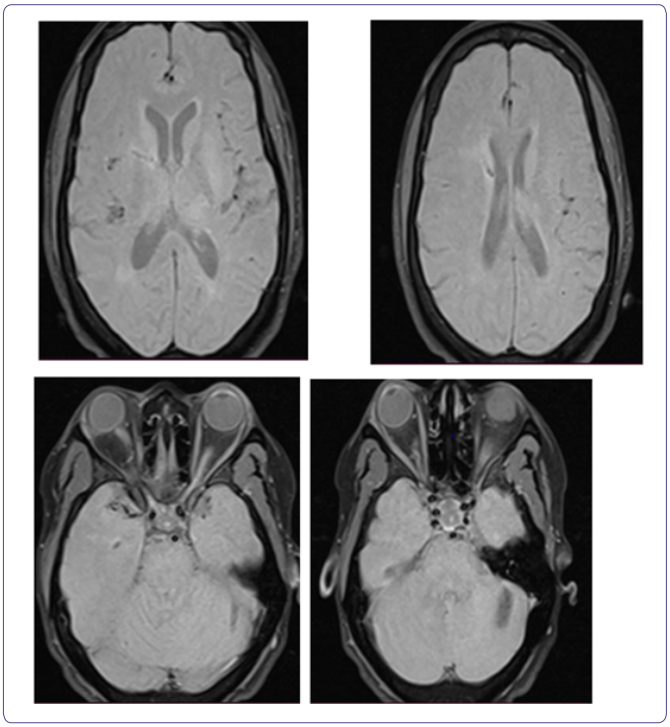
The sellar was enlarged and filled with CSF. The hypointense T2 and hyperintense T1 infundibulum was seen traversing within it giving the infundibulum the sign of an empty sella. The pituitary gland was seen displaced postero-inferiorly. The parasellar regions were normal. The cavernous sinuses were normal.

The ventricular system and subarachnoid spaces were within normal limits. Both cerebello-pontine angle cisterns were unremarkable.

Marrow of the cranial vault appears normal. Intracranial vessels display expected flow voids. There was no evidence of intra / extra axial fluid collection or mid line shift. The paranasal sinuses mastoid air cells are were normal.

Summary of radiological report

1. Widespread T2/FLAIR hyperintense plaques within the deep white matter of the centrum semi-ovale and the corona radiata - consistent with Multiple Sclerosis
2. Enlarged sellar turcica, filled with CSF, and demonstrating positive infundibulum sign associated with increased CSF around the optic nerve which demonstrates atrophic changes - consistent with idiopathic intracranial hypertension



Discussion

Multiple sclerosis (MS) is a demyelinating disease which affects the central nervous system. It is not a common diagnosis in Nigerian Hospitals. The reasons for this are the rarity of the disease condition among Blacks [1], the presence of few Magnetic Resonance Imaging (MRI) facilities and the general paucity of knowledge about the disease entity.

MS, also called Disseminated scleroses, due to distribution in space and time of this disease entity is common among Caucasians compared to Blacks; prevalence is highest in North America and Europe, but also higher in the middle east and North Africa (MENA) compared to sub Saharan Africa [1,2] and East Asia. Also, it is more common in Blacks who have emigrated to environments where it is more common compared to those in Africa, supporting the thoughts of environmental factors playing significant role in the pathogenesis of the disease entity. This is further buttressed by the higher prevalence in countries with higher latitude, colder climate and viral infections [3] Okubadejo in 2014 in a hospital based study showed incidence of 1.26 per 1000, with a preponderant female to male ratio of 3 to 1. Patients were mostly in the 4th decade of life, all of who stayed in Nigeria. These findings correspond with the findings in our patient who had just had her 40th birthday [3].

In a case report in Enugu, South East Nigeria, it was reported that the occurrence of multiple sclerosis was in a male farmer who ingested alcohol and smoked significantly, about the same age as our patient, who had no visual affectation, but he had recurrent difficulty with walking [4].

Despite the usefulness of imaging to determine probability of MS, a strong index of suspicion still remains the more accessible resource especially where MRI is not available. Based on the Mc Donald's criteria, there can be probable, possible or definitive MS based on a set of criteria which include dissemination of disease in time and space of

at least 2 episodes with MRI confirmation. Cerebrospinal fluid(CSF) examination also shows increased protein content and CSF oligoclonal band [5].

The index patient did not have the benefit of CSF examination due to papilloedema. However, there were recurrent visual defects and MRI features.

In this patient, there was associated bilateral optic nerve atrophy both clinically as evidenced by optic disk pallor despite normal ocular pressure and bilateral thinning of the optic nerve.

Optic nerve involvements are not uncommon in MS, optic neuritis, and not optic atrophy is said to be the commonest[7]. At the initial stages of the disease, it may be difficult to differentiate between it and Devic's disease; however, in Devic's disease, also known as, Neuromyelitis Optica, MRI findings are usually normal.

In most of the case reports around Nigeria, visual manifestations have not been well reported as we see in this patient [6-8]. The earlier cited case report in Enugu and another in South-south Nigeria who had locked in syndrome were also devoid of ophthalmic involvement[8]. Availability of genetic testing would have helped to provide better insight into this patient's case especially considering disease entities such as Leber's Hereditary Optic Neuropathy resulting from mitochondrial point mutations in mtDNA 11778G>A, 14484T>C, or 3460G>A mutations which is quite rare and tends to run a familial course being mitochondrial in inheritance [9].

Conclusion

Multiple sclerosis is an immune mediated demyelinating disease of the central nervous system, which runs a debilitating cause. Prevalence is lowest in sub-Saharan Africa and East Asia, however perceived to be rising worldwide. A high index of suspicion still remains a priced possession of clinicians in environments where adequate imaging modalities are not readily available. Also there is need to pay attention to possible ophthalmic manifestation especially as first or predominant features as in this patient.

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