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Pulmonary And Cutaneous Sarcoidosis in A Tertiary Health Facility In South-East Nigeria: A Case Report

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ABSTRACT:
Sarcoidosis is a chronic inflammatory disorder, of unknown aetiology affecting multiple systems, and characterized by non-caseating granulomas. It is characterized by a complex interplay of genetics, environmental exposures and aberrant immunologic response. The diagnosis of this condition is challenging because of its mimicry of tuberculosis, and other granulomatous disorders. It often involves multisystem involvement for a specific diagnosis. Pulmonary sarcoidosis is the commonest type of sarcoidosis.
In this article we present Mr AA, 45-year- old male who presented with a history of chronic cough of about 7 years, weight loss and skin lesions of about 5 years, and shortness of breath of about 6 months.
The index patient had clinical improvement over the duration of therapy, has remained clinically well; and on regular follow-up with routine investigations. This case highlights the need for clinical vigilance, familiarity with imaging features, and prompt treatment in selected cases; thus, helping in diagnosing, evaluating the extent of disease, and guiding optimal health care.
Effective regular follow-up is necessary to monitor changes in the disease, including extension, progression, remissions, flare-ups, and complications.

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I. INTRODUCTION

Sarcoidosis is a chronic multi-system inflammatory disorder characterized by non-caseating granuloma, usually of unknown etiology.[1,2]. It often involves the presence of at least two organs for a specific diagnosis.[3]Common organs involved include the lungs and hilar lymph nodes, skin, eyes, Musculo-skeletal and joint systems, as well as the heart, kidneys and the central nervous system.[4,5]Pulmonary sarcoidosis is the commonest type of sarcoidosis.[3]

Sarcoidosis is characterized by a complex interplay of genetics, environmental exposures and aberrant immunologic response.[6]

The current evidence suggests that environmental agents, including infectious and non-infectious, are able to trigger an exaggerated inflammatory response with non-necrotizing granulomatosis in genetically susceptible individuals.

Identified infectious agents include *Mycobacterium tuberculosis*, *Propionibacterium acnes*, reported within lymph nodes or granuloma of sarcoidosis patients.[3] Pathogenic components or DNA of mycobacteria have been implicated as potential antigenic triggers for Sarcoidosis.[3]

Other non-infectious agents implicated in Sarcoidosis include silica, aluminum, mold, bioaerosols and insecticides.[7,8]

Patients who have genetic susceptibility to sarcoidosis, such as patients with specific Human Leukocyte Antigen (HLA) class II alleles (HLA-DRB1, HLA-DQB1) or patients with genetic polymorphism in BTNL2, ANXA-II, manifest an aberrant inflammatory reaction to the above antigens due to dysregulated immune system.[3,9-12]

Antigen presenting cells such as macrophages and dendritic cells present the persisting antigens of these environmental agents to CD4+ Helper T cells which secrete immunostimulatory cytokines particularly interleukin 2, IFN-γ and TNF-a, which in turn propagate further macrophage activation, granuloma formation and a persistent inflammatory state in affected organs such as the lungs and intrathoracic lymph nodes.[4,6]

The clinical presentation and investigations that enable prompt diagnosis are varied, and require high index of suspicion in clinicians.

II. CASE PRESENTATION

We present a 45-year-old male, civil servant of south-eastern Nigerian descent and residence, who presented with a history of chronic cough of about 7 years, weight loss and skin lesions of about 5 years and shortness of breath of about 6 months.

The cough was non-productive, persistent, not worse at any time of the day, season of the year or position, not accompanied by haemoptysis or fevers. He admitted to occasional drenching sweats at the time. Following initial evaluation by chest radiograph and sputum evaluation, he was diagnosed presumptively as having pulmonary tuberculosis on radiological grounds as his sputum tests were negative for acid-fast bacilli. He completed a 6-month course of antituberculosis treatment using the 2-month initial four-drug combination of isoniazid, rifampicin, pyrazinamide and ethambutol and the 4-month continuation phase of rifampicin and isoniazid. However, the cough only reduced in intensity without entirely abating.

Thereafter, he noticed he was losing weight, in spite of a preserved appetite. He reported no excessive sweating, palpitations, hyper-defaecation, increasing urination or thirst at the time. He lost about 18kg over a 3- to 5-year period, dropping from his usual weight of 80kg to about 62kg on presentation.

At about the same time, he noticed dark skin lesions appearing over his limbs. These were painless, raised, non-itchy, non-blanching and appeared to cluster mostly over the extensor surfaces of the extremities with minimal truncal, facial and truncal involvements.

Six months prior to presentation, he developed breathlessness noticed initially on moderate exertion but progressively worsened to interfere with his usual activities of daily living. There was no leg, facial or abdominal swelling, reduction in urine output, orthopnoea or paroxysmal nocturnal dyspnoea. It was predominantly on account of this symptom that he sought expert pulmonology care.

He had negligible smoking history (<0.5 pack-years). He drank socially and reported no use of recreational substances. He had no medical history of hypertension, asthma, epilepsy, diabetes mellitus or sickle cell disease. He was single and lived alone. He had no occupational exposures to organic or inorganic dusts, solvents, fumes, and did not keep any pets or birds.

On examination, he was noted to be a middle-aged male, who was mildly breathless, not pale, febrile, cyanosed, jaundiced or dehydrated; he had no leg oedema, significant peripheral lymphadenopathy or digital clubbing.

His respiratory rate was 26 cycles/min, even and regular, Oxygen saturation (SpO2) was 92% on room air. His trachea was central and percussion notes were resonant with preserved cardiac and hepatic dullness. There were scattered crackles in both lung fields, worse in the left upper zone. No wheezes were heard.

He had a pulse rate of 98 beats/min and a blood pressure of 128/74mmHg measured on the right arm in the seated position. His first and second heart sounds only, were heard and there were no murmurs.

He had diffuse patches of nontender, partially scaly, hyperpigmented lesions, with central atrophy over the extensor surfaces of his upper and lower limbs, with the lower limb lesions showing a wider distribution (figure 1a). A few lesions were noted on the face and scalp as well.

His abdomen was soft, nontender and moved with respiration and no palpable organomegaly was detected.

There were no abnormalities from his central nervous system examination.

An earlier chest radiograph done had showed diffuse reticular lesions with right upper lobar streaks suggestive of pulmonary tuberculosis (figure 2).

Following the clinical history, examination findings and review of the chest radiograph, an impression of possible sarcoidosis was made and a high-resolution computed tomography (HRCT) of the chest, spirometry, full blood count, serum electrolytes plus calcium, urea and creatinine, urinalysis and fasting blood glucose tests were requested. The possibility of a skin biopsy was kept in view where diagnostic uncertainty remained after the initial evaluations.

The HRCT of the chest demonstrated diffuse interstitial lung changes bilaterally, but worse on the left; hilar and perihilar consolidative changes bilaterally also worse on the left with calcified peri-bronchial nodes.Other than marginally enlarged aortic and cardiac sizes, no other abnormalities were noted. A conclusion of sarcoidosis with differentials of pulmonary tuberculosis and interstitial lung disease entertained (figure 3).

The spirometry test revealed reduced Forced Expiratory Volume in 1 second (FEV1) and forced vital capacity (FVC) but preserved FEV1/FVC ratio consistent with restrictive ventilation.

His blood count was essentially normal but for mild anaemia (Hb of 11.8g/dL) and he had no renal or glycaemic abnormalities or hypercalcaemia.

The diagnosis of sarcoidosis was upheld; and following shared decision making, a course of oral corticosteroid therapy was commenced as Prednisolone 40mg once daily under mucosal protection of proton-pump inhibitor therapy for the first 3 months.

Symptoms of cough, breathlessness and weight loss had significantly resolved at the time of reassessment 3 months later. His resting pulse had dropped to 78/min and his SpO_2 was 97% in room air. He had gained 7kg in the time and his skin lesions were involuting (figure 1b). His steroid therapy was subsequently tapered over the coming months and additional medications prescribed, viz Tab. Azathioprine 50mg twice daily and Tab. Hydroxychloroquine 200mg daily.

The latter medication was prescribed after satisfactory baseline ophthalmological assessment.

He continues his follow up with progressive sustained improvement in respiratory and skin symptoms after 8 months of therapy and is due for re-evaluation by imaging and functional lung testing in the coming months.

FIGURES

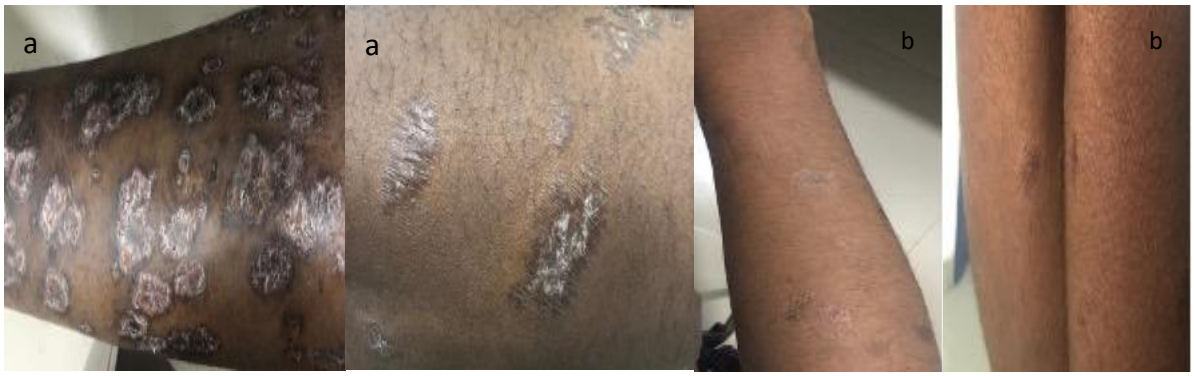

Figure 1a. Skin lesions prior to treatment; **b**. lesions after 3 months of treatment

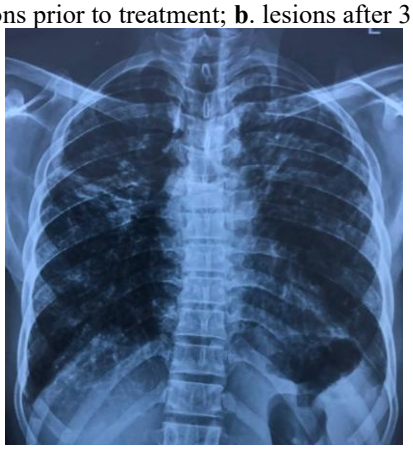

Figure 2. Chest radiograph showing predominantly upper zone streaks and reticulonodular opacities suggestive of pulmonary tuberculosis

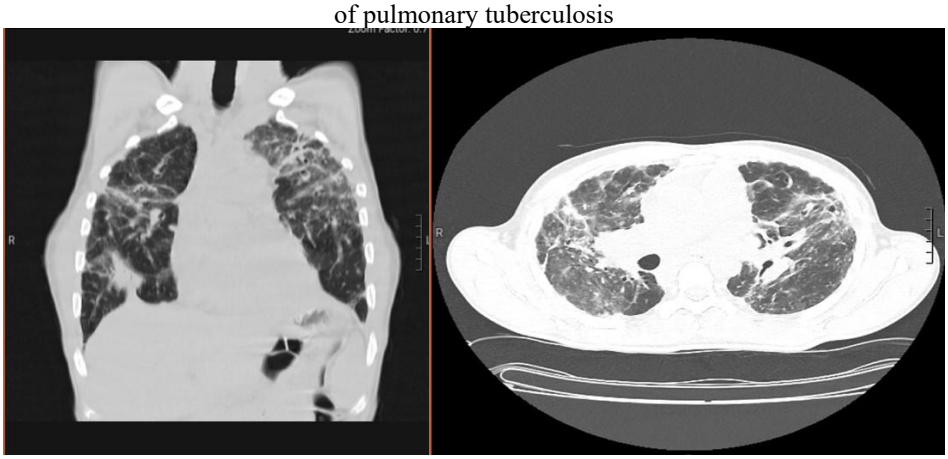

Figure 3. Coronal and axial HRCT chest scans showing diffuse interstitial changes, perihilar consolidative changes and calcified peri-bronchial nodes.

III. DISCUSSION

Sarcoidosis demonstrates great variability in its racial, geographic, age and sex distribution.

Globally, it is difficult to establish the true epidemiology of sarcoidosis because of its initial asymptomatic course.[2] Its highest prevalence however is reported in the Nordic population, with highest per 100,000 population estimates in Sweden and Denmark (20 – 60 cases per 100,000 population).[1] 60 per 100,000 population prevalence is reported in the United States with a profound predilection for the African American population (18 per 100,000 compared to 8.1 per 100,000 in the white population).[1,2]

Lowest prevalence has been reported amongst the East-Asian population, often less than 5 per 100,000 population.[13]

The challenge of underdiagnosis is worse in Sub-saharan African and Nigeria, and is often complicated by delayed presentation, insufficient diagnostic tools and frequent misdiagnosis as pulmonary tuberculosis,[14,15]and this is demonstrated in our index case. Patient had been misdiagnosed for Pulmonary Tuberculosis, which is the closest differential of Pulmonary Sarcoidosis, and was only properly diagnosed seven years after onset of initial symptoms.

Awotedu et al in 1987 reported 12 cases within 2 years at the University College Hospital, Ibadan-Nigeria that appeared to discredit the erstwhile belief of its rarity in the region.[15]Despite underreporting, 47.6% of Interstitial Lung Disease cases in a health facility in Nigeria were attributable to Pulmonary Sarcoidosis,[16] and pulmonary involvement in Sarcoidosis remain the commonest presentation in Nigeria.[15]

Females are more commonly diagnosed with Sarcoidosis but current smoking appears to be associated with a lower risk of developing sarcoidosis, though the mechanism is unclear..[17-19] Our patient, a male, with a history of tobacco smoking deviate from this pattern. There are however reported studies that have shown a male preponderance in specific forms of sarcoidosis.[19,20]

There are variabilities both in the onset and presentation of sarcoidosis, and thus could be easily misdiagnosed, especially in resource-variable settings such as sub-saharan Africa, as was with the index patient. High index of suspicion is advised.

Presentation of Sarcoidosis will often depend on the organ system involved. It ranges from asymptomatic to organ failure.[3]Constitutional symptoms may include fever, night sweats and weight loss.[1,4] Pulmonary sarcoidosis which is the commonest form of sarcoidosis could also present with dry cough, chest discomfort and exertional dyspnea, and spirometry will reveal restrictive respiratory impairment.[21,22] Greater than 90% of patients with sarcoidosis will have pulmonary involvement.[3]

The case presented had dry cough and exertional dyspnea as well as constitutional symptoms (fever, weight loss, malaise and drenching night sweats). Exam findings in pulmonary sarcoidosis may reveal decreased breath sounds and crackles, and digital clubbing in the setting of pulmonary fibrosis.[13, 23]

20-30% of patients with sarcoidosis, as in our index patient, also present with cutaneous manifestations ranging from erythema nodosum, lupus pernio, and papular or plaque–like eruptions.[1,4,20] The skin is reported to be the second most commonly involved organ in sarcoidosis.[24]

Our patient had both pulmonary and cutaneous sarcoidosis and similar cases, though rare, have been reported in the literature.[25] Lupus pernio, which was present in our patient, is a distinct form of cutaneous sarcoidosis that disproportionately affects black people,[26]and is characterized by erythematous, indurated or violaceous plaques, mostly on the face. It is associated with increased risk of concomitant pulmonary sarcoidosis.[27]

Other extra-pulmonary manifestationsof sarcoidosis include the musculoskeletal system (myopathy, arthritis, arthralgia), heart (arrythmia, heart failure), central nervous system (leptomeningism. Cranial neuropathies and hypothalamic dysfunction).[4,23,28] Direct renal involvement and calcium dysregulation has also been reported.[3]These were not present in our index patient.

The diagnostic criteria as outlined by the 2020 American Thoracic Society (ATS), European Respiratory Society (ERS) andWorld Association of Sarcoidosis and Other Granulomatous Disorders (WASOG) include three essential components:[17-19]

1. A compatible clinical presentation and radiologic findings
2. Histologic evidence of non-caseating granulomatous inflammation
3. Exclusion of other causes of granulomatous diseases, particularly infections (e.g. mycobacterial, fungal) and occupational exposures (e.g. berylliosis, hypersensitivitypneumonitis).

Chest radiograph is the baseline imaging modality and has been used by the Scadding Staging System to provide framework for both classification and prognostication:[1,21, 23]

Stage 0: Normal Chest Radiograph

Stage I: Bilateral hilar lymphadenopathy (BHL) without pulmonary infiltrates.

Stage II: BHL with parenchymal infiltrates

Stage III: Parenchymal infiltrates without BHL

Stage IV: Pulmonary fibrosis with volume loss, architectural distortion, and cystic changes.

In this patient, initial chest radiograph revealed diffuse reticulonodular lesions with right upper lobe streaks. A High-Resolution CT (HRCT) of the chest, as was done by the index patient promotes advantage in detecting upper lobe predominance, peri-lymphatic micronodules, broncho-vascular and subpleural distributions as well as detecting fibrosis or bronchiectasis.[21,23]

Other advanced imaging modalities include Positron Emission Tomography with Flourodeoxyglucose (FDG), Magnetic Resonance Imaging (MRI) and confers advantages such as detecting metabolically active granulomas, and detecting cardiac or neurologic involvements.

Flexible bronchoscopy with TBLB or EBUS-TBNA are minimally invasive ways of obtaining tissue for histology and though generally kept in view for index patients, one would expect the presence of tight, non-caseating granulomas comprised of multi-nucleated giant cells, epithelioid histiocytes with minimal lymphocytic cuffing.[4,19] Broncho-alveolar lavage (BAL) fluid analysis, endobronchial mucosal biopsies are additional supportive investigations that could be carried out. They were not necessary in our index patient.

Differential Diagnosis of PulmonarySarcoidosis include Pulmonary Tuberculosis, Granulomatosis with Polyangiitis (GPA), etc. Whilst often initially misdiagnosed for Tuberculosis on account of overlapping symptomologies and similarities in radiographic findings, as was seen in our index patient, cases of co-existence of both have been reported.[29] GPA is typically characterized by **necrotizing** granulomas and positive **c-ANCA** (PR3-ANCA) markers, whereas sarcoidosis is characterized by non-caseating granulomas.

Our patient was placed on oral prednisolone for 3 months as this is the first line treatment for sarcoidosis. The recommendation is to administer at a dose of 20 – 40mg daily for the first 4-6weeks, then taper at 5-10mg/day for a duration of 6-12months.[18]

Corticosteroids have been shown to significantly improve symptoms, and as was noted in our patient facilitates clearance of radiographic infiltrates and overall improvements in pulmonary function such as Forced Vital Capacity (FVC). Common steroid side effects to look out for include pathological fractures, diabetes and opportunistic infections.[2]

Other second line therapies include Methotrexate, Mycophenolate Mofetil, Leflunomide and Azathioprine, all of which are steroid-sparing agents. Methotrexate is given weekly at 10-15mg with folate supplementation. Azathioprine which was commenced for the patient at the end of the corticosteroid regimen is given at 1.5 to 2.5 mg/kg/day. Full blood count, renal and liver function tests must be done both as baseline and routinely, to assess for toxicity in patients who have been commenced on these agents.[19,20]

Third line biologic agents such as TNF-α inhibitors (Infliximab, Rituximab, Adalimumab) are reserved, per recommendation for Refractory Sarcoidosis.[20,23]These were not necessary in our index patient.

Follow up and serial monitoring is very important for patients on management for Sarcoidosis. This is important to assess response to treatment and identify drug toxicity early. It is advised that chest radiographs and spirometry be done every 3 to 6 months during treatment and annually after treatment.[18,19] Most patients with Scaling Grade 0-III will experience spontaneous remission while a third might progress to chronicity and its associated complication such as Pulmonary fibrosis and pulmonary hypertension.[23,28]Our index patient was placed on such follow-up, with scheduling of serial monitoring of aforementioned investigations.

The good clinical response of our patient to the initial corticosteroids indicates favourable prognosis, and is reported in a two-third of patients with pulmonary sarcoidosis.[18,19,23]

IV. CONCLUSION

Sarcoidosis is a chronic multi-system non-caseating granulomatous disorder. Pathogenesis involves an interplay of dysregulated immune response to persisting antigens of infectious and non-infectious environmental agents in a person who is genetically susceptible. In our region, high index of suspicion is advised for early detection and treatment of sarcoidosis as it is often misdiagnosed for Pulmonary Tuberculosis and other granulomatous diseases. This capacity for early detection is important as treatment modalities differ widely and advanced stages of sarcoidosis could carry risk of treatment failure and progression to debilitating complications. Histopathological confirmation remains the gold standard but advanced diagnostic tools such as HRCT of the chest, flexible bronchoscopy with TBLB or EBUS-TBNA is recommended to enhance the diagnostic yield and outcomes for patients in South-East Nigeria.

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