Hidradenitis Suppurativa of the Vulva in a Retroviral Positive Postmenopausal Woman: A Case Report

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Abstract

Hidradenitis suppurativa (HS) is a painful and chronic condition commonly occurring in women in the reproductive age group. We report the case of a postmenopausal woman who was being treated by highly active antiretroviral therapy (HAART) who presented to our clinic with a 3-month history of a discharging ulcer on the left labium majus with ipsilateral non-tender inguinal lymphadenopathy. Histopathological examination of the surgically excised labial lesion tissue showed features of HS. The patient was treated with oral antibiotics, oral steroids, and anti-inflammatory medications. Following these treatment interventions, the lesion healed well with no recurrence over two years of follow-up.

Keywords: Hidradenitis Suppurativa; Postmenopausal Women; Excision Biopsy; Nigeria

Introduction

Hidradenitis suppurativa (HS) is a chronic suppurative and inflammatory disorder of the terminal follicular epithelium in the apocrine gland–bearing parts of the skin such as the axilla, groin, perianal and infra-mammary areas and has a pronounced impact on patients.[1,2] This condition is three times more common in females than in males.[1,3,4] It is rare in healthy individuals, prepubertal children, and post-menopausal women.[3] A recent meta-analysis estimated an overall HS prevalence of 0.4% in the Euro-American and Australian populations.[5] Several studies have suggested that the prevalence may rise to around 4% in women during their prime childbearing ages of 20–40 years.[1,6,7] The assumption that HS symptoms would decline after menopause was challenged by a North American survey in which 39.5% of post-menopausal women reported that their HS symptoms had worsened after menopause, while 44.2% reported no change.[7] However, there are not many cases of HS in postmenopausal women in the literature, and very few from Nigeria.

The treatment modalities of HS include topical and systemic antibiotics, topical antiseptics, systemic and intra-lesional corticosteroids, systemic retinoids, anti-androgen drugs and hormonal manipulations. Severe HS may require surgical excision of the involved tissue and radical therapy with good outcome. Recently, biologics such as adalimumab have been advocated.[1,2,8,9]

Case Report

A 53-year-old woman was referred to our gynecology clinic with a three-month history of swelling in the left labium majus. The swelling was insidious and had gradually increased in size. It was associated with pain, ulceration, and a yellowish discharge without foul smell.

She had a history of five pregnancies (all resulting in live births), was HIV positive for six years, and was three years postmenopausal. She was hypertensive but not diabetic. A cervical cytology (Pap smear) test conducted two years earlier had yielded normal results. She had no history of postmenopausal vaginal bleeding.

General examination revealed a healthy-looking woman with a body mass index (BMI) of 30.5 kg/m^2. There was no evidence of weight loss, and she had no cough. Her blood pressure was 150/95 mmHg. The left inguinal lymph nodes were enlarged, discrete, and were not tender. Other systemic examinations were normal. Pelvic examination revealed healthy looking clitoris, right labia majus and minus. In the lower third of the left labium majus, there was an ulcerative, hard, nodular, and non-tender mass of about 20 × 20 mm. The vaginal capacity appeared normal and there were no nodular masses. The cervix was healthy looking. The uterus was approximately 8 weeks gestation size and both adnexa were free.

Our initial clinical diagnosis was chronic indurated vulvar ulcer secondary to syphilis, assuming there was no vulvar malignancy. The other differential diagnoses included lymphogranuloma venereum, chancroid, donovanosis, aphthous ulcers and tuberculosis. Thus, local excision with a wide margin (excision biopsy) became necessary.

Laboratory results showed hemoglobin concentration of 13.3 g/dL, total white blood cell count of 6,900 / mm^3 (granulocytes: 42.1%, lymphocytes 49.9%, platelet count: 264,000 /mm^3. The results of urinalysis, liver function test, and serum levels of electrolyte, urea, and creatinine were all within their normal ranges. Tests for syphilis, hepatitis B and hepatitis C yielded negative results. No microbial growth was detected in the ulcer. CD4 Count was 452 cells/mm^3 and the viral load was < 20 copies/mm. Chest X-ray revealed mild cardiomegaly (cardiothoracic ratio at 54%) with mild unfolding of the aorta. There was no active lung parenchymal disease, and the surrounding bony thorax was normal. Abdominopelvic ultrasonography also revealed normal findings.

Following cardiologist's review and appropriate counseling, the patient underwent wide local excision biopsy of the vulval lesion with 2 cm free margins. The histopathological report on the specimen revealed focus of ulceration in the epidermis with extensive areas infiltrated by a mixed population of lymphocytes, plasma cells and neutrophils underlying the dermis. Skin adnexa, hair follicles and apocrine glands were surrounded by these cells with obvious luminal contents in the apocrine glands. There was mild acanthosis of the epidermis. There was no evidence of malignancy. Together, these features strongly supported a diagnosis of hidradenitis suppurativa, ruling out our initial clinical suspicions of syphilitic chancre or vulvar malignancy.

The histopathological slides are shown in Figures 1 to 5:

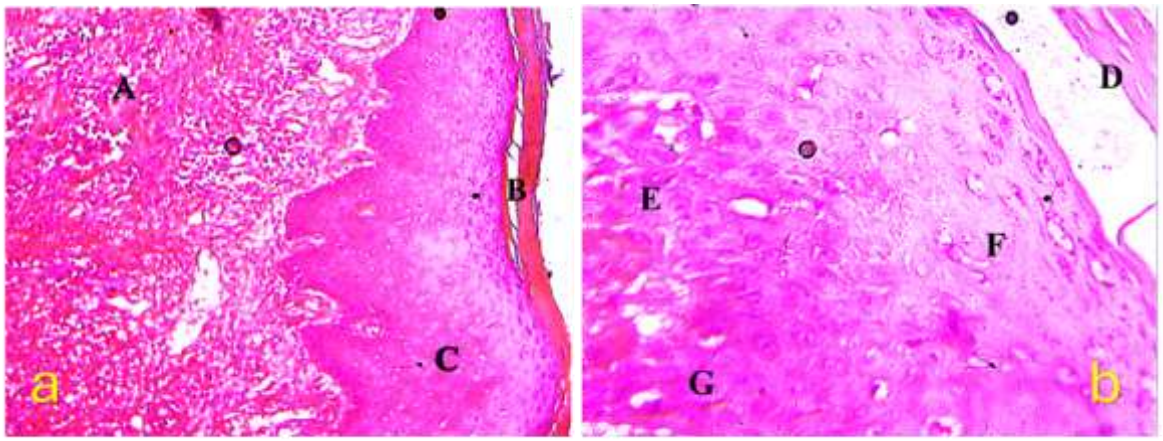

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Figure 1: Sections of the hematoxylin & eosin-stained dermal lesion at different magnifications. 1a: At 100× magnification, the dermis (A), stratum corneum (B), and epidermis (C) are visible, revealing the tubular and acini structures lined by epithelial cells with an outer layer of myoepithelial cells. 1b: The same section at 400x magnification shows the stratum corneum (D) and the epidermis (E, F, G) in a higher magnification.

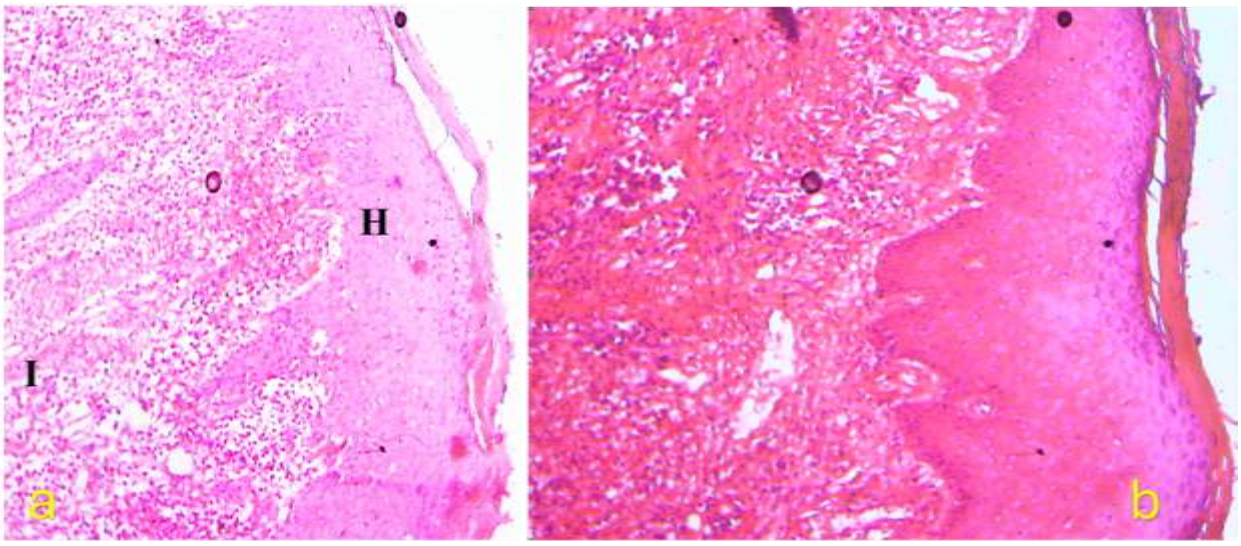


Figure 2. Sections of the hematoxylin & eosin-stained skin. 2a: At 100× magnification, the epidermis reveals a keratinized stratified squamous epithelium (H) overlying the papillary dermis (I). 2b: At 400× magnification, the same section shows the papillary dermis with the reticular dermis which is unremarkable in this slide.

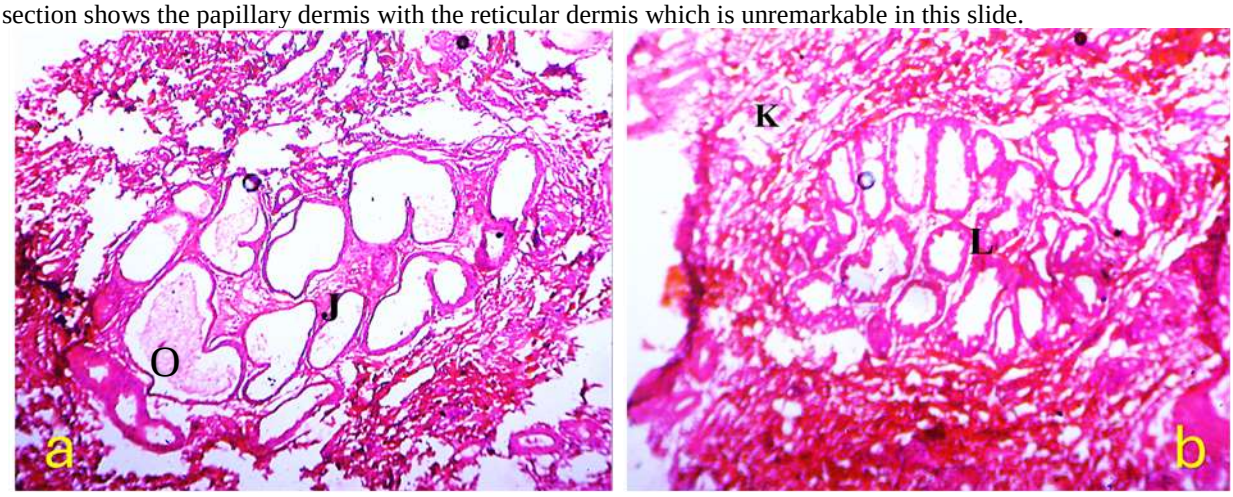
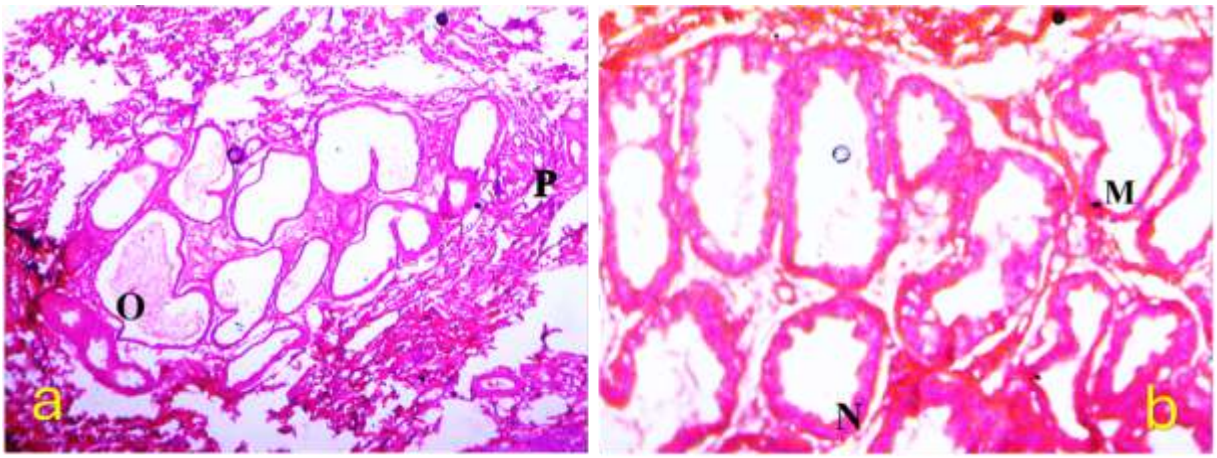


Figure 3. Sections of the hematoxylin & eosin-stained skin at 100x magnification. 3a: The skin section shows tubular and acini structures which are lined by epithelial cells and outer myoepithelial cells (O, J, L). 3b: The lesion is disposed within a stroma of connective tissue (K), which contain blood vessels.



Figures 4. Sections of the lesion stained with hematoxylin and eosin at different magnifications. 4a: At 100× magnification, cyst-like structures are seen in clusters and separated by connective tissue stroma (P). Eosinophilic fluid is present in some of the acini (O). 4b: At 400× magnification, the glandular lesions are lined by epithelial cells (N) with myoepithelial cells disposed to the periphery (M).

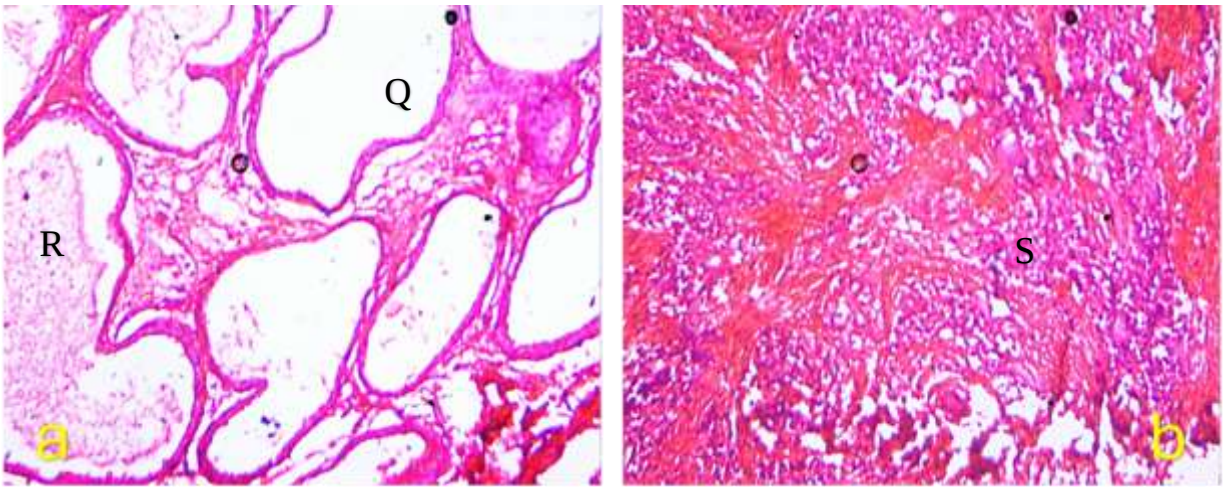


Figure 5. Both slides are stained with hematoxylin and eosin (H & E) and magnified 100×. They reveal cyst-like structures arranged in clusters and separated by connective tissue stroma. The glandular lesions (Q) are lined by epithelial cells with myoepithelial cells disposed to the periphery. Some of the acini contain eosinophilic fluid (R). Interconnecting tissue stroma is labeled 'S.'

The histopathological findings were discussed with the patient**,** who was also counseled on the possible modes of further management. She received the following systemic antibiotics: intravenous cefuroxime 750 mg 8-hourly for 72 hours and oral cefuroxime 500 mg 12-hourly and oral metronidazole 400 mg 8-hourly, both for 10 days. Steroid treatment comprised tablet prednisolone 10 mg 12-hourly for 5 days, later scaled down to 5 mg 12-hourly for 5 days and finally 5 mg daily for another 5 days. She also received anti-inflammatory tablet diclofenac sodium 50 mg 12-hourly for 7 days. Her antiretroviral and antihypertensive medications continued as before. She was discharged after three days on the above treatment. At two weeks follow up, the histopathological findings were reviewed and the antibiotics modified to metronidazole, clindamycin, and rifampicin for 16 weeks. There was good clinical response, and she had no recurrence of the vulvar lesion during a two-year period of follow-up.

Discussion

Hidradenitis suppurativa is a chronic inflammatory and debilitating disease affecting the apocrine gland bearing skin and causes painful abscesses and nodules that can eventually progress to interconnected sinus tracts, scarring and contractures of the skin.[10]

The etiopathogenesis of hidradenitis suppurativa remains unclear. Some studies have shown an association between HS and obesity, genetic predisposition, cigarette smoking and family tendency.[1] These were not found in our patient. Others have shown sex predilection.[11] Available reports show that HS is a multifocal disease where lifestyle, immunological processes, genetics, and hormonal predisposition may play roles in promoting follicular hyperkeratosis, dilatation and rupture, leading to chronic tissue inflammation.[12] The atrophy of the sebaceous glands is followed by an early lymphocytic inflammation and hyperkeratosis of the pilosebaceous unit, with eventual hair follicle destruction and granuloma formation, with increased vulnerability to secondary superimposed bacterial infections.[13] While HS is commoner in reproductive-aged women, it may also occur at the extremes of age in immune-compromised individuals,[1] as exemplified in the current case. People with HIV are reported to be particularly susceptible to developing HS with the characteristic involvement of atypical sites, such as face or thigh.[14] Deng et al. found a six-fold higher rate of HS diagnosis in patients with HIV compared with those without HIV.[15]

Clinical examination is the mainstay of diagnosing HS. Its hallmark is inflammation of the apocrine gland-bearing regions causing painful boils. The severity is determined by the degree to which the lesions progress to abscesses, sinus tracts, and scarring, and is based on Herley's category 1–3.[16] Differential diagnosis of this clinical condition depends on the anatomic location. In the vulva, these include, vulvar carbuncles, syphilitic ulcer, lymphogranuloma venereum, Crohn's disease, vulvar acne, keloidalis nuchae cyst, and vulvar malignancy.

In our patient, a postmenopausal woman, hidradenitis suppurativa presented as a hard, ulcerated, painful vulvar nodule, and her atypical clinical condition posed a diagnostic challenge with an initial suspicion of malignancy. Therefore, bacteriological studies and biopsy of the suspected lesion were resorted to, leading to the diagnosis of HS.

The treatment modalities depend on the stage of the disease, ranging from lifestyle modifications to medical, surgical, and radiotherapy approaches. Recommended lifestyle changes include weight reduction, reduction of alcohol consumption, and avoiding rubbing of the affected skin.[9]

Medical management includes topical, systemic and intralesional therapies using anti-inflammatory, antibiotics, steroids and anti-androgens. These have been shown to improve the quality of life and reduce the recurrence of symptoms.[10] Additionally, recent reports have shown favorable outcomes from using systemic immunosuppressive agents in patients with severe form of the disease. Surgery is reserved for patients who have shown poor response to medical treatment and/or patients with late-stage disease or extensive scarring, with little or minimal option of incision and drainage due to high rate of recurrence.[10]

Our patient was treated with systemic antibiotics, steroids, and anti-inflammatory agents leading to good clinical response without recurrence for two years of follow up without resorting to immunosuppression. This probably shows that surgical excision may be superior to simple incision and drainage in the care of such patients.

Conclusion

Hidradenitis suppurativa cases may pose diagnostic dilemmas in low resource settings and require a multi-modal approach especially when it occurs in immune-compromised postmenopausal women. Though it is commoner in women in the reproductive age group, in the face of an immune-compromised condition, HS may occur at the extremes of age, as exemplified in this case, and as reported elsewhere.

Disclosure

The authors declare no conflicts of interest. Informed consent was obtained from the patient for publication of the case.

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