Case Report

Cutaneous candidiasis mimicking inverse psoriasis lesion in a type 2 diabetes mellitus patient

Steven Philip Surya¹, Kardiana Purnama Dewi², Regina Regina²

¹. Medical Profession Study Programme, School of Medicine and Health Sciences, Atma Jaya Catholic University of Indonesia
². Department of Dermatology and Venereology, School of Medicine and Health Sciences, Atma Jaya Catholic University of Indonesia / Atma Jaya Hospital Jakarta

Email: regina@atmajaya.ac.id

Abstract

Background: Type 2 diabetes mellitus (T2DM) condition could affect the skin. The disease is among the greatest risk factors of skin infection such as cutaneous candidiasis and is also associated with autoimmune skin diseases such as psoriasis, which lesion morphologies and predilection areas are quite similar. These similarities are a source of confusion to clinicians.

Case illustration: We present a 60-year-old female patient with uncontrolled T2DM and multiple erythematous plaques in the form of shiny lesions at the intertriginous area that developed 1 week prior to her hyperglycemic state. The lesions had recurred at the same site for approximately 10 years and were initially diagnosed as intertriginous candidiasis. Bedside testing revealed a positive Auspitz sign, while the patient’s fungal culture was unremarkable. She was diagnosed with inverse psoriasis (IP) and treated with 0.1% mometasone furoate cream twice a day. The lesions improved but were not completely resolved.

Discussion: Inverse psoriasis is a subgroup of psoriasis characterized with thin, non-layered scales, and various predilection areas. The features of the primary lesion may be altered by a patient’s activities, such as use of cleansing soap, which may cause lesions to dry out and ooze, similar to cutaneous candidiasis. Additional examination, such as bedside testing and laboratory work, could help obtain a proper diagnosis.

Conclusion: The similar morphologies and predilection areas of IP and cutaneous candidiasis lesions may confuse clinicians. In some limited cases, the correct diagnosis may be obtained by complete history taking, physical examination, and other simple tests.

Keywords: inverse psoriasis, intertriginous cutaneous candidiasis, type 2 diabetes mellitus

Background

Type 2 diabetes mellitus (T2DM) is the most prevalent metabolic disorder worldwide. The World Health Organization has predicted that 15% of the global population will develop T2DM, impaired fasting glucose, or impaired glucose tolerance by 2025.¹ In Indonesia, over 21 million patients are expected to be diagnosed with diabetes mellitus by 2030.² Diabetes mellitus caused by genetic or clinical disturbances manifests as suboptimal or absence of insulin secretion, resulting in increased blood sugar levels.³ An experimental study has revealed an association between blood sugar level and an immunosuppressed state.⁴ A prolonged hyperglycemic state and insulin deficiency could render a patient prone to infection, neuropathy, suppression of cytokine production, defective phagocytosis, immune cell dysfunction, and suboptimal immune response against bacteria.⁵ Thus, T2DM patients are prone to infection. One of the most common infections affecting T2DM patients is cutaneous candidiasis.⁶ A recent study demonstrated an association between T2DM and autoimmune diseases and it is called as immune-mediated inflammatory disease of the skin.⁷-¹⁰ Alterations in the immune system under a high blood glucose state render T2DM among the factors influencing the incidence of oral or cutaneous candidiasis and psoriasis.⁷,¹¹
While a diagnosis of psoriasis is usually made upon the observation of classical plaque lesions and history taking, other examinations, such as dermatopathology, may sometimes be necessary to confirm this diagnosis.

Inverse psoriasis (IP) is a very rare variant of psoriasis and affects only approximately 3%–7% of all psoriasis patients. IP is characterized with well-demarcated, shiny, erythematous lesions. The lesions may be moist and smooth and feature a fissure at their center. The predilection areas of IP include the axillae and inguinal and inframammary folds. Given their similar predilection areas and lesion morphologies, IP and cutaneous candidiasis may confuse clinicians.

In our case report, a 60-year-old female patient with uncontrolled T2DM in a hyperosmolar hyperglycemic state was referred to our department for exacerbation of multiple erythematous lesions 1 week before admission. The patient's skin condition had persisted for 10 years but often resolved by itself. Relapses occurred without any known exacerbating factor. During a relapse, the lesions would appear in the same locations, such as the scalp, trunk, extensor area of the extremities, and inframammary, lower abdominal, and inguinal folds. The lesions had been previously diagnosed by a general physician as cutaneous candidiasis, which the patient usually treated with 4 mg of methylprednisolone during relapse. The lesions would heal occasionally but mostly persisted. No other family members had a history of scaly erythematous plaques.

During physical examination, besides an elevated blood pressure, the patient's other vital signs were within normal limits. Shiny, linear, erythematous plaques were noted in the inframammary, lower abdominal, and both inguinal folds. The patient's Psoriasis Area Severity Index score was 3.3. No satellite lesions were found around the main lesion (Figure 1–2).

**Case Illustration**

A 60-year-old female was referred by the internist inpatient department to the dermatovenereology department in the ward because of multiple itchy scaly and non-scaly erythematous plaque lesions on her inframammary fold, groin area, and extremities; the patient reported no pain or burning sensation. The lesions developed 1 week prior to her admission to the hospital.

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![Figure 1. Erythematous Plaques with Thick Layered Scales in the Groin Area](Image)
Figure 2. Shiny, Linear, Erythematous Plaques in the Inframammary Fold

Figure 3. Auspitz Sign in the Lower Abdominal Fold

Figure 4. Koebner Sign on the Left Elbow
Figure 5. Gram Smear from the Patient’s Groin Area

We found pinpoint bleeding when we scraped the skin of the inframammary and inguinal folds, which we concluded as a positive Auspitz sign (Figure 3). History taking and physical examination revealed a positive Koebner sign on the right elbow (Figure 4). The differential diagnosis of the patient’s skin condition was intertriginous candidiasis coincidental with psoriasis vulgaris or IP. We performed Gram smearing with samples obtained from the inguinal and inframammary folds but did not find any fungal element (Figure 5). No fungal growth was observed in cultures of Sabouraud dextrose agar and Sabouraud dextrose broth. We did not perform histopathological examination because of the patient’s refusal.

Blood examination showed an elevated erythrocyte sedimentation rate (61 mm/hour; normal values: 0–20 mm/hour), blood glucose (492 mg/dL; normal values: 60–140 mg/dL), and HbA1c (11.6%; normal values: 4.0–5.6%). Urinalysis showed +4 glucose, +1 protein, +2 nitrite, and +4 leucocytes. Sediment examination for urinalysis revealed elevated leucocytes (20–40/high field microscope; normal values 0–2/high field microscope) and bacterial and fungal elements. On the basis of the patient’s history and our physical examination, we diagnosed the patient’s skin condition as IP with lesions on the inframammary and inguinal folds mimicking intertriginous cutaneous candidiasis in diabetic patients.

We treated the patient with 0.1% mometasone furoate cream applied twice daily. The lesions visibly improved but had not resolved completely at the time the patient was discharged from the hospital. The prognosis of the patient was poor because of her failure to understand the severity of her skin condition and uncontrolled T2DM, which may exacerbate IP.

Discussion

T2DM is correlated with various skin issues ranging from infectious diseases to autoimmune diseases. Psoriasis is a chronic, low-degree inflammatory disease that could affect systemic conditions, such as cytokine production and metabolic regulation. A cohort study including 75,599 adults divided into the psoriasis and non-psoriasis groups and followed for 4 years showed that the psoriasis group has a higher incidence of diabetes than the non-psoriasis group (3.44% vs. 2.44%). An animal model suggested that systemic psoriasis may alter glucose metabolism and induce a hyperglycemic state similar to hyperglycemia-induced viral infections. A case–control study demonstrated significantly higher total cholesterol (p = 0.028), low-density lipoprotein (LDL) (p = 0.015), triglycerides (p = 0.006), and glycemic state (p = 0.021) in the psoriasis group compared with the control group.

Cutaneous candidiasis could increase morbidity and even mortality, especially among ill patients, and T2DM may predispose patients to cutaneous candidiasis on account of their immunosuppressed state. Cutaneous candidiasis usually affects intertriginous areas, such as the groin, abdominal skin fold, inframammary skin fold, and interdigital spaces. The disease typically appears as thin, bright-red plaques that can be erosive, dry, scaly, oozing, or macerated and often presents with satellite vesiculopustular lesions. A diagnosis of cutaneous candidiasis is mainly achieved through clinical examination followed by a potassium hydroxide smear and, in some
cases, fungal culture. A systematic study including 1,500 samples revealed candidiasis (not limited to cutaneous candidiasis) as a main exacerbating factor in psoriasis patients. Psoriasis is a chronic inflammatory disease that is closely related to genetics and characterized with alterations in epidermal growth and differentiation. A recent study showed that T cells may mediate autoimmune reactions against speculated self-antigens within the skin and cause chronic inflammation. Psoriasis causes increased production of interferon gamma and interleukin-17, which interact with dermal cells, macrophages, mast cells, and neutrophils; this phenomenon is called the psoriasiform reaction. IP generally yields lower amounts of CD16+ than common psoriasis.

IP is usually observed as erythematous, pruritic, well-demarcated, wet, and non-scaly lesions; a fissure may sometimes appear at the center of a lesion. The lesions have predilection areas of the axillae and inguinal and inframammary skin folds and sometimes cause a pruritic sensation. Diagnostic tools for psoriasis include history taking, physical examination, and skin biopsy. Examination goals include the ruling out of other look-alike diagnoses, such as cutaneous candidiasis.

IP is a genetic disease that often affects first-degree relatives with the same condition. In the present case, our patient presented with chronic recurrent multiple erythematous, dry, well-demarcated, and non-scaly lesions in the inframammary and inguinal folds and scaly oozing lesions in the abdominal fold. These lesions were suspected to develop because of the patient’s use of cleansing soap. Although we found multiple erythematous, erosive plaques on the patient’s skin folds, the lesions were generally dry, and no satellite lesion was observed. Our findings of positive Auspitz and Koebner signs supported the diagnosis of IP. We performed potassium hydroxide smear and fungal culture, and both examinations yielded negative results for fungal elements. Hence, we excluded the diagnosis of cutaneous candidiasis. However, urinalysis indicated fungal elements, indicating a fungal infection in the urinary tract that could have triggered the current exacerbation episode.

This case report presents some limitations. Histopathological examination could not be performed because of the patient’s refusal. Thus, our diagnosis was based on the successful exclusion of cutaneous candidiasis. Moreover, we cannot report a satisfactory treatment result for the patient due to lack of follow up.

Conclusion

IP is a rare subgroup of psoriasis. IP lesions sometimes mimic those of cutaneous candidiasis but could also be triggered by the latter. Psoriasis and candidiasis are highly correlated with metabolic conditions, such as T2DM. Psoriasis is a chronic, low-inflammation disease that may promote a hyperglycemic state, especially in diabetic patients. Clinically, the lesions of IP and cutaneous candidiasis may show similar morphologies and predilection areas; thus, these conditions may sometimes confuse clinicians. In limited cases, complete history taking and thorough physical examination may help obtain the correct diagnosis.

References

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