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CUTANEOUS POLYARTERITIS NODOSA: A CASE REPORT

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ABSTRACT
Cutaneous polyarteritis nodosa is a rare type of vasculitis that involves inflammation in small to medium-sized blood vessels, mainly targeting the skin. It affects people of all ages and significantly impacts both the skin and other organ systems. This case report aims to describe the clinical presentation, diagnostic approach, and therapeutic response. A 38-year-old man came with a two-week history of a red, non-itchy rash on both legs. A week before the rash appeared, patient experienced fever, sore throat, and stomach pain. His general condition was stable. Dermatological examination showed multiple palpable purpura on both legs, ranging from miliary to lenticular in size. Diascopy revealed non-blanching purpura, and dermoscopy showed a homogeneous pattern of multiple erythematous spots. Histopathology indicated a proliferation of blood vessels with enlarged endothelial cells, concluding with a diagnosis of polyarteritis nodosa. Patient was diagnosed with cutaneous polyarteritis nodosa and treated with methylprednisolone at 1 mg/kg/day, divided into three doses of 32 mg each, with tapering off every week. Clinical improvement was observed, and the skin lesions progressively resolved. This case highlights the importance of early recognition and corticosteroid therapy in the successful management of mild cutaneous polyarteritis nodosa.

Keywords: corticosteroid; cutaneous polyarteritis nodosa; vasculitis

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INTRODUCTION

The inflammation of blood vessels is characteristic of vasculitis. Polyarteritis nodosa (PAN) is a type of systemic vasculitis that primarily impacts medium and small-sized arteries. Cutaneous polyarteritis nodosa (CPAN) primarily impacts the skin, but extracutaneous symptoms can include fever, fatigue, muscle pain, joint pain, neuropathy, and involvement of organs like the kidneys, liver, and heart (Munera et al., 2020; Okazaki et al., 2017). Cutaneous polyarteritis nodosa occurs in individuals of all ages, from 3 days to 81 years old, and is associated with immune complex-mediated disease. One of the most commonly identified causes is Group A beta-haemolytic Streptococcus. Furthermore, CPAN has been linked to inflammatory bowel disease and hepatitis B infection (Pineider et al. 2020; Wang and Tsai, 2021; Wolff et al., 2023).

Cutaneous polyarteritis nodosa is characterized by painful, red spots such as purpura, papules, and swelling, which can merge to form large, hardened patches of skin. Other symptoms include livedo reticularis, skin ulceration, myalgia, arthralgia, and non-erosive arthritis. Cutaneous symptoms are mainly limited to the lower extremities (Furukawa, 2012; Matteoda et al., 2015). Mild CPAN usually improves with rest and treatment using nonsteroidal anti-inflammatory drugs (NSAIDs). In patients who do not respond to NSAID therapy and those with severe CPAN, moderate doses of systemic steroid therapy are recommended (Chung et al., 2021; Micheletti, 2022). This study aims to evaluate and describe the clinical characteristics, diagnostic strategies, and treatment responses in adults patient diagnosed with

cutaneous polyarteritis nodosa, thereby contributing to a better understanding of its management and outcomes.

METHOD

This reports presents a descriptive case study of a 38 year old male diagnosed with cutaneous polyarteritis nodosa. Clinical data were obtained through detailed patient interviews, physical examination, and supportive investigations including diascopy, dermoscopy, and histopathological analysis. The diagnosis was based on the combination of characteristic skin finding and histological. Case reports designed to highlight rare presentations that may contribute to clinical knowledge and improve diagnostic accuracy. In this study, clinical and pathological data were systematically compiled to document the course of the disease and its response to therapy. An evaluative approach was also applied to assess the clinical significance of the findings, acknowledge the report's limitations, and ensure the reliability of its conclusions.

RESULT

Case description, diagnosis, management, and evaluation

A 38-year-old male presented to the Dermatology and Venereology Clinic of Adam Malik Hospital Medan with the main complaint of a reddish rash without itching on both legs for the past two weeks. Initially, the rash appeared on the left leg and then spread to both legs without itching or pain. The patient experienced pain in both knee joints and ankles along with the appearance of the rash. About one week before the rash appeared, the patient had a fever, sore throat, and stomach pain but did not seek treatment, assuming it was a common cold. The patient had never experienced such complaints before and did not have a history of drug or food allergies. The patient has a history of a Hepatitis B diagnosis for five years.

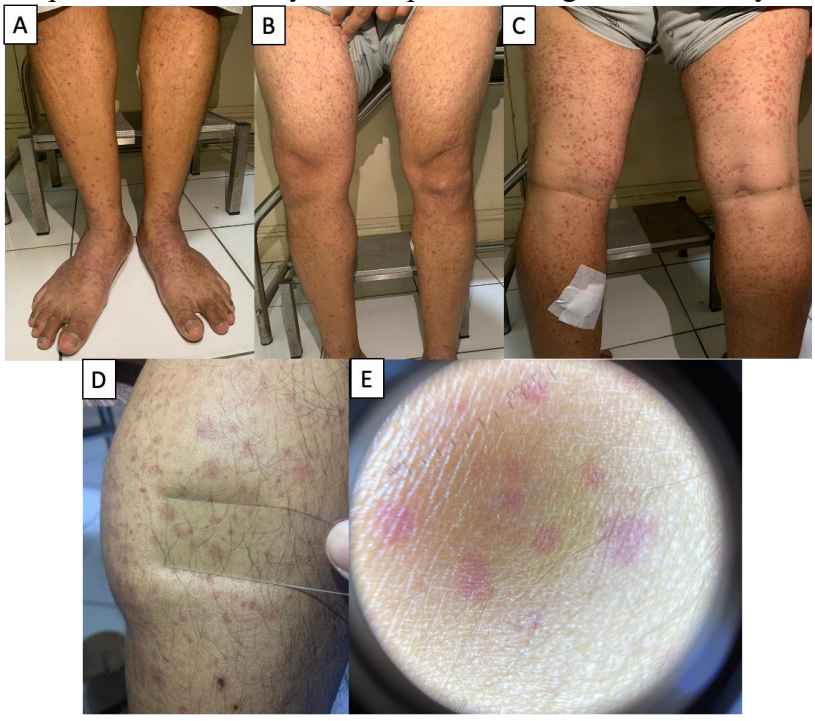

Figure 1. Dermatological examination found (A,B,C) Multiple palpable purpura, sizes varying from miliary to lenticular, circumscribed, spreading symmetrically on bilateral cruris regio. (D) Diascopy examination showed purpura was non-blanching whe pressure was applied. (E). Dermoscopy examination found homogeneous pattern of multiple erythematosus spots.

On physical examination, the patient appeared in good overall condition, fully conscious, blood pressure was 140/80 mmHg, pulse was 86 bpm, respiratory rate was 18 rpm, body temperature was 37.4ºC, body weight was 95 kg, and body height was 173 cm. Dermatological examination revealed multiple palpable purpura, varying in size from miliary to lenticular, circumscribed, and spreading symmetrically on the bilateral cruris region (Figure 1A-C). Diascopy examination showed that the purpura was non-blanching when pressure was applied (Figure 1D), while dermoscopy examination revealed a homogeneous pattern of multiple erythematous spots (Figure 1E).

Laboratory examination was carried out. A complete blood test showed hemoglobin of 13.6 g/dL, erythrocytes of 4.44 million/mL, hematocrit of 38.9%, MCV of 88 fl, MCH of 30.6 pg, MCHC of 35 g/L, platelets of 467,000/mm³, and leukocytes of 11,660/mm³. The differential count of basophils / eosinophils / neutrophils / lymphocytes / monocytes was 0.3% / 0.9% / 70.8% / 23.2% / 4.8%, respectively. Coagulation factors were within normal limits (PT: 10.5, INR: 0.94, APTT: 22.8, and TT: 15.3). Renal function was within normal limits (urea: 24 mg/dL and creatinine: 1.07 mg/dL). Liver function was within normal limits (SGOT: 32 U/L, SGPT: 52 U/L). Immunoserology tests showed HBsAg reactive, Anti-HCV non-reactive, anti-dsDNA level at 48.8 IU/mL, ANA test at 17.32 IU/mL, and rheumatoid factor (RF) <8 IU/mL. Urine analysis results were within normal ranges.

Histopathological examination of the skin biopsy revealed a proliferation of blood vessels of various sizes, with enlarged endothelial cell linings, round and oval nuclei, and smooth chromatin. In addition, the blood vessel walls showed thickening, along with infiltration by neutrophilic inflammatory cells. Within the blood vessel walls, inflammatory cells, lymphocytes, neutrophils, and macrophages were visible. Red blood cells were seen outside the blood vessels. These results led to the diagnosis of polyarteritis nodosa. (Figure 2).

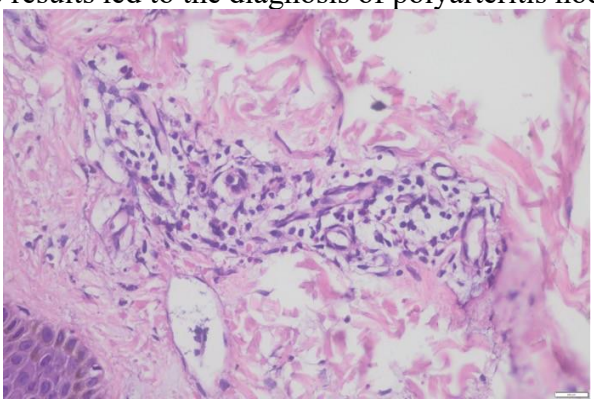

Figure 2. Histopathology examination by skin biopsy found proliferation of blood vessels with various sizes, enlarged endothelial cell linings, round and oval nuclei, and smooth chromatin.

Based on patient history, dermatological examination, and supporting examinations that had been carried out, patient was diagnosed with cutaneous polyarteritis nodosa which was established based on diagnostic criteria, namely physical examination found palpable purpuric skin lesions and other organ system disorders. The patient was administered methylprednisolone, a systemic corticosteroid, at a dosage of 1 mg/kg/day (96 mg/day), divided into three doses of 32 mg, with a gradual weekly reduction. The patient was advised to get plenty of rest at home and consume healthy, nutritious food. He was asked to return for a follow-up in one week. At the first follow-up control, conducted seven days after initiating treatment, the patient reported improvement in skin lesions and reduction in joint pain. Dermatological examination showed that the palpable purpura on both legs had started to fade and decrease in number (Figure 3). The oral methylprednisolone dose was tapered to

80mg/day, divided into three doses (32mg – 32 mg – 16mg). the patient was advised to return for a follow up visit one week later.

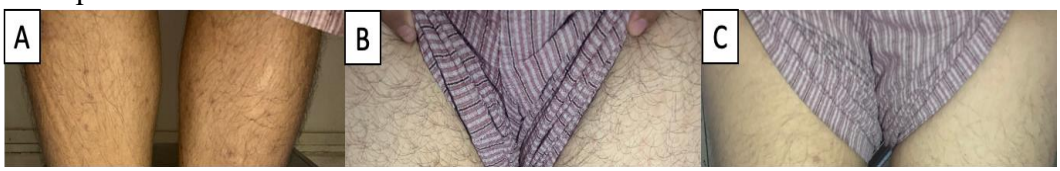

Figure 3. Palpable purpura on the bilateral crural region, varying in size from miliary to lenticular, which had begun to fade and diminish.

At the second follow-up, two weeks after the start of therapy, further improvement in the rash was observed (Figure 5). The dosage of oral methylprednisolone was reduced to 64 mg/day, administered in three divided doses (32 mg – 16mg – 16mg). The patient was scheduled for another follow-up one week later.

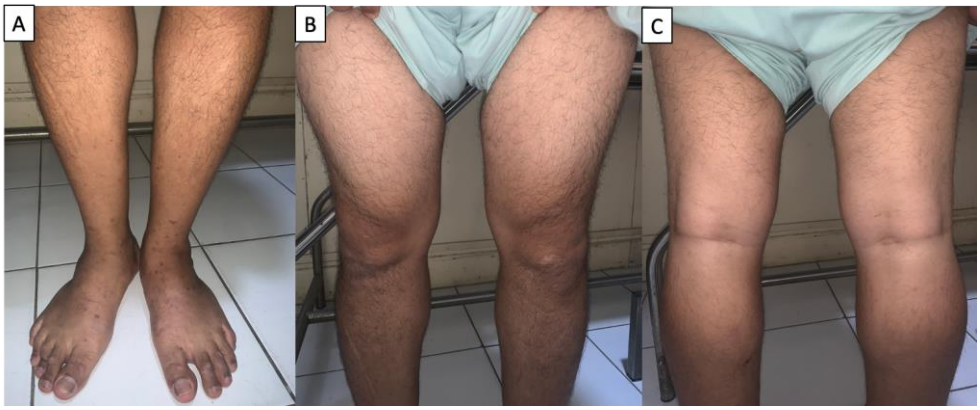

Figure 5. Palpable purpura on both legs showed marked reduction during the third clinical evaluation.

The patient's prognosis was good regarding life expectancy (quo ad vitam bonam), uncertain to good regarding functionality (quo ad functionam dubia ad bonam), and uncertain to good regarding recovery (quo ad sanationam dubia ad bonam).

DISCUSSION

The cause of most CPAN cases is still unclear. Hepatitis B virus (HBV) infection is thought to be linked to 10–54% of CPAN cases, especially in European countries. However, in Japan, only a small number of polyarteritis nodosa cases have been linked to HBV. Moreover, the incidence of HBV-related polyarteritis nodosa has declined with the introduction of vaccines for viral hepatitis. The correlation between hepatitis B virus and CPAN occurs due to virus replication, which causes damage to the blood vessel walls. This damage is also caused by circulating immune system factors that activate the complement cascade, attracting and activating neutrophils (Wang and Tsai, 2021; Wolff et al., 2023).

Cutaneous polyarteritis nodosa is diagnosed through skin lesions (subcutaneous nodules, livedo, purpura, ulcers), histopathological findings (fibrinoid necrotizing vasculitis in small and medium arteries), and extracutaneous symptoms (fever, weight loss, hypertension, renal failure, infarctions, heart issues, pleuritis, intestinal problems, peripheral neuropathy, and abnormal arteriography showing microaneurysms, stenosis, and obliteration) (Hiraiwa et al., 2023).

In these cases, patients also suffer from fever and joint pain. CPAN can present with extracutaneous symptoms, including fever, arthralgia, peripheral neuropathy, and myositis,

affecting 22%–66% of patients (Furukawa, 2012). Additionally, hypertension can occur due to renal artery involvement, painful sensations in the abdomen due to vascular lesions in the GI tract, and peripheral neuritis, especially in motor nerves. According to the literature, common laboratory findings in CPAN cases include mild anemia, moderate leukocytosis, and negative serological tests for syphilis, ANA, and rheumatoid factor (Ordoñez-Parra et al., 2021; Hiraiwa et al., 2023).

Histopathological examination revealed thickened blood vessel walls infiltrated by neutrophilic inflammatory cells. Within the vessel walls, inflammatory cells such as lymphocytes, neutrophils, and macrophages were observed, along with the presence of red blood cells outside the vessels. According to the literature, the histologic changes in CPAN progress through four stages. The first stage, degeneration, features arterial wall damage with fibrinoid deposits and destruction of the elastic layers. The acute inflammatory stage follows, showing neutrophil and eosinophil infiltration around and within the vessel wall. Next, the granulation stage is marked by lymphocytes, macrophages, intimal thickening, and arterial thrombosis, potentially causing ulceration. Finally, the healed stage is characterized by fibroblast growth extending around the vessel (Furukawa, 2012; Hiraiwa et al., 2023).

The differential diagnosis in this case includes Henoch-Schönlein Purpura and Immune Thrombocytopenic Purpura (ITP). Clinical findings of Henoch-Schönlein purpura include palpable purpuric skin lesions accompanied by at least one of four symptoms or signs: digestive tract disorders, immunoglobulin A deposition found on biopsy, arthritis or arthralgia, or kidney disorders (proteinuria or hematuria) (Reamy et al., 2020). In this case, biopsy results did not find immunoglobulin A deposition, and laboratory results indicated normal renal function. Immune thrombocytopenic purpura is a bleeding disorder characterized by a decrease in total platelets (thrombocytopenia) that most commonly affects children, with clinical symptoms such as epistaxis, gingival bleeding, hematomas and petechiae. The absence of thrombocytopenia in this patient also eliminates the differential diagnosis of ITP (Thakur et al. 2024).

The treatment of CPAN is determined by the disease's severity (Stanton and Tiwari, 2023). Mild disease is characterized by constitutional symptoms, renal function is normal, and the absence of organ injury. Patients with mild symptoms and localized skin involvement can be treated with glucocorticoids. Prednisone is typically prescribed at an initial oral dose of 1 mg/kg per day, with a maximum daily dose ranging from 60 to 80 mg. The stable steroid dose is maintained for one month before gradually being decreased over six to eight months (tapering off). Patients with mild disease who do not respond to or cannot tolerate glucocorticoid treatment may be prescribed immune modulators like azathioprine or methotrexate. Renal insufficiency, ischemic, artery stenosis or aneurysm suggest moderate to severe disease. Patients with moderate to severe disease are treated with glucocorticoids along with other immunosuppressive drugs, such as cyclophosphamide (Chung et al., 2021; Stanton and Tiwari, 2023). The best prognosis is for CPAN patients with isolated skin lesions, but they have a high risk of relapse. Without any treatment, the risk of mortality in CPAN patients within 5 years is estimated to be about 10–20%. With corticosteroid therapy, the survival rate up to 5 years increases to around 50–60% (Chung et al., 2021; Stanton and Tiwari, 2023).

CONCLUSION

This case report describes the successful treatment of mild cutaneous polyarteritis nodosa in a 38-year-old man diagnosed based on clinical signs and histopathological examination, without any abnormalities in other organ systems. The patient was treated with systemic corticosteroid therapy.

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