Sir,

Cutaneous polyarteritis nodosa (CPAN) is a self-limiting cutaneous vasculitis characterized by *livedo reticularis* and painful nodules that mostly affect the lower limbs.\(^1\) It is limited to the skin, and involves no major organ systems. Mild fever and involvement of muscles, joints, and the peripheral nervous system may be observed. A definitive diagnosis is established by histopathologic evidence of necrotizing inflammation of the medium and small-sized arteries. We report one such case here.

An 18-year-old boy presented with 10 months history of increasing erythema, vesicle formation, and lower limb ulceration. Ten days prior to the appearance of cutaneous lesions, he had sustained high grade fever with temperature ranging from 38°C to 40°C, acute sore throat, joint and muscle pain, nausea, and vomiting. He had no other cardiovascular, respiratory, or gastrointestinal symptoms, and denied any illicit drug use.

Physical examination revealed symmetrically distributed, finger nail-sized red papules on the soles and red reticular spots at the medial border of the feet. The papules faded on pressure. Ulcers of varying sizes with dark crusts on the surface and surrounded by erythemas were visible in the calves (Figure 1). Large and small subcutaneous nodules were visible in the dorsum of the feet, the lower calves, and the upper extremities, with pain experienced by the patient on palpation. Some papules exhibited a beaded arrangement. The red papules were found to be scattered over the waist and buttocks. The top of each papule was necrosed. The right axillary and inguinal lymph nodes, in addition to the right epididymis, were swollen.

The hemoglobin level of the patient was 11.1 g/dL. His white blood cell and platelet counts were 13.8 x 10\(^9\)/L and 49.8 x 10\(^4\)/dL, respectively. The percentage of neutrophil granulocytes was 77.1%, while that of eosinophil granulocytes was 10.1%. No bacterial growth was observed in the blood culture. The erythrocyte sedimentation rate of the patient was 71 mm in the first hour. The C-reactive protein level was 118.43 mg/L. The electrocardiogram, echocardiogram, and head and chest computed tomography showed normal results. Serum antinuclear antibody, anti-double-stranded DNA and antineutrophil cytoplasmic autoantibody titers of the patient were negative. Serum complement C3 and C4 concentrations were 0.87 g/L and 0.48 g/L, respectively. The routine urine test showed normal results, with the 24-hour urinary protein level measuring 100 mg. The Bence-Jones protein test was negative, and the routine liver and kidney function tests were normal. The Hepatitis B surface antibody (HBsAb) test was positive, while the Hepatitis B surface (HBs) antigen, HBe-antigen, HBe-antibody, and HB core (HBc) antibody tests were negative. Bone marrow aspiration revealed predominantly mature granulocytes and increased numbers of rod nuclear cells, reactive plasma cells (6.5%), and eosinophils (11.0%), suggesting the presence of infection.

Histopathological examination revealed infiltration of neutrophil granulocytes, eosinophil granulocytes, and lymphocytic cells around the dermal tissue. The arterial wall was disintegrated, with exuded fibrin and dermal necrosis. The subcutaneous tissues showed small arteries with necrotizing changes. Fibroplasia, along with vascular wall proliferation and obstruction were also observed. A part of the muscular layer of the blood vessel and the internal elastic lamina fracture were replaced by hyperplasia of the fibrous connective tissue (Figure 2). Based on these findings, a diagnosis of CPAN was established.

**Figure 1:** The figure shows calves with small- and large-sized ulcers with dark crusts on the surface and surrounded by erythemas.

**Figure 2:** Photomicrograph of the histopathological examination of the patient (Hematoxylin and Eosin, 200 x).
The patient received oral prednisone at a dose of 1 mg/kg/day for 22 days. At discharge on day 23, the dose was reduced to 0.4 mg/kg/day, as the cutaneous lesions had resolved and the color of the local lesions and the crusts had faded. Subsequently, the prednisone dose was reduced by one table (5 mg) every week. After 12 weeks of treatment, the patient was discharged on oral prednisone. He was followed as an outpatient for a period of 6 months. No recurrence of symptoms or exacerbation was observed during this period.

CPAN typically advances in a course of repeated episodes of increasing severity. Ulcerative CPAN shows a slower progression and is associated with peripheral neuropathy. The clinical features of ulcerative CPAN may resemble those of Erythema induratum of Bazin (EIB; nodular vasculitis), papulonecrotic tuberculid and cutaneous leukocytoclastic vasculitis (CLV). EIB was excluded in this case based on the absence of lobular panniculitis. Papulonecrotic tuberculid was excluded based on the absence of central necrosis, depression, crusts, and the chief lesion in the upper dermis. CLV was excluded, as the small blood vessels located in the upper- and mid-dermis are affected in this condition.

Necrotizing arteritis of the small- and medium-sized vessels is a common histological feature of systemic and cutaneous PAN. CPAN affects the skin, muscles, joints, and the peripheral nervous system while visceral involvement is observed in systemic PAN. This can be confirmed clinically (central nervous system, pulmonary, cardiac, gastrointestinal, or renal), radiographically (abnormal angiography), or histologically (visceral biopsy).

In this case, the patient was positive for HBsAb and negative for HBs and HBe antigens, and HBe and Hbc antibodies. Hepatitis B virus (HBV) infection could have been the etiology of CPAN in this case. Despite a dramatic decline in its incidence over the last decade, HBV-associated PAN remains a life-threatening condition and a therapeutic challenge.

Since CPAN has variable clinical features, dermatologists should be aware of symptoms such as painful nodules and livedo reticularis. Early detection and treatment of the disease could help improve the treatment outcome.

REFERENCES