

Photoclinic



Figure 1. A & B) Indurated and ulcerative satellite plaques with a violaceous base and sharp but irregular borders, surrounded by petechiae in several areas on both his knees and the anterior aspect of his right thigh.

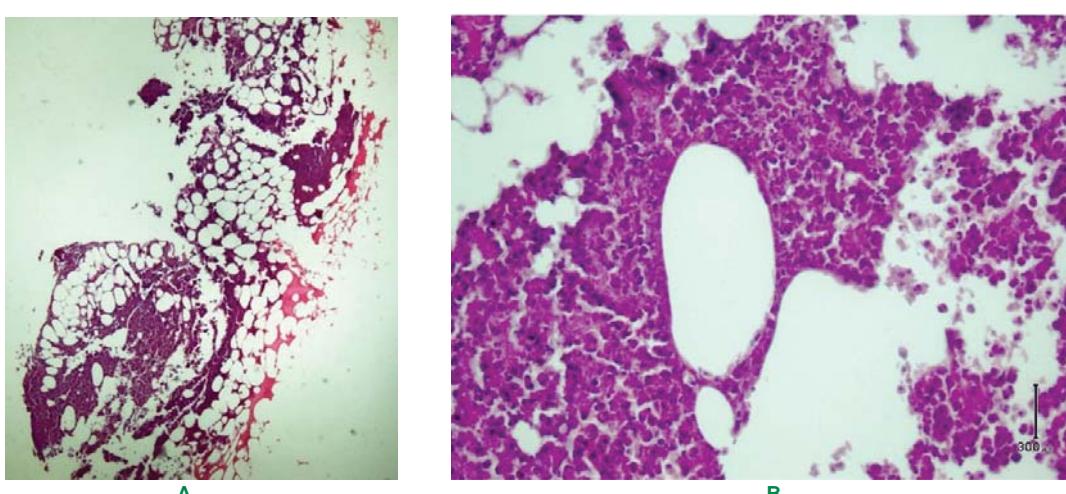


Figure 2. A & B) Necrosis of subcutaneous fat with paraseptal lymphocytic infiltration and mixed septal/lobular panniculitis were seen. (Magnification x30 and x400 respectively)

Cite this article as: Mortazavi H, Soori T, Azizpour A, Goodarzi A, Nikoo A. Photoclinic. *Arch Iran Med*. 2014; **17(9)**: 649 – 650.

A 61-year-old opium addicted male admitted to the hospital with a history of a fairly good health, until four weeks before his admission, when he became febrile with an axillary temperature of 38.4 °C. Then he developed tender, erythematous and indurated plaques over the front of his both knees, which became ulcerative in a few days. Thereafter, the lesions spread to his right thigh. Physical examination at the time of admission revealed two indurated and ulcerative satellite plaques with a violaceous base and sharp, but irregular borders, surrounded by petechiae in several areas on both his knees and the anterior aspect of his right thigh. The lesions measured from 5 to 18 cm in diameter. Both feet were edematous (Figure 1 A&B). Axillary temperature was 38 °C, heart rate was 104, and blood pressure was 124/78 mmHg.

Hossein Mortazavi MD¹, Tahereh Soori MD¹, Arghavan Azizpour MD¹, Azadeh Goodarzi MD¹ Azita Nikoo MD²

Author's affiliations: ¹Department of Dermatology, Razi Hospital, Tehran University of Medical Sciences, Tehran, Iran, ²Department of Pathology, Razi Hospital, Tehran University of Medical Sciences, Tehran, Iran.

•Corresponding author and reprints: Azita Nikoo MD, Razi hospital, Tehran University of Medical sciences, Tehran, Iran. Tel: +98-21-55159988, E-mail: azinik@yahoo.com.

Accepted for publication: 22 June 2014

Laboratory evaluation revealed a normal cell blood count and normal urine analysis. The patient's erythrocyte sedimentation rate was 13 mm/hour (normal, < 20 mm/hour). Viral markers (hepatitis B and C and HIV) as well as VDRL were negative. The complement levels of C3, C4 and CH50 were in the normal range and anti-nuclear antibody (ANA) was negative. Liver function tests were within normal limits. Amylase and lipase levels were 51 IU/L (up to 100) and 18 IU/L (up to 60IU/L), respectively. Carbohydrate antigen 19-9 (7.2 U/ml, normal < 3.7 U/ml) and alpha1-antitrypsin antibody (51.4 mg/dl, normal 62-169 mg/dl) were within normal limits.

Doppler ultrasound of arterial and venous flow, performed to rule out the possibility of deep vein thrombosis, was normal. Ultrasound of liver and biliary ducts, CT scans of the mediastinum, and x-ray of knees showed no significant change. An incisional skin biopsy was performed.

What is your diagnosis?

See the next page

Infectious-induced panniculitis has been occasionally reported, but it is important for pathologists and dermatopathologists to differentiate infectious etiology of panniculitis from more common etiologies that have frequently the same pathologic features.

In infection-induced panniculitis, patients develop local erythematous nodules. Some of these nodules may become fluctuant and subsequently ulcerated with draining. Lesions on the legs and feet are common, but other parts of the body might be involved as well.¹ Two forms of infective panniculitis have been described; primary infective panniculitis which occurs after direct inoculation of the pathogen into the fat tissue of subcutis, whereas secondary infective panniculitis occurs through hematogenous spread of the organism like during septicemia in the blood stream.^{2,3}

In primary infective panniculitis, infectious agents are considered to be directly responsible for the panniculitis. Examples of reported microorganisms to date are bacteria (like *Staphylococcus aureus*, Group A *Streptococcus*, and *Pseudomonas aeruginosa*), mycobacteria, borrelia, and fungi.⁴⁻⁷

The diagnosis of infective panniculitis is based on the histological finding of mostly lobular, mostly septal, or mixed septal/lobular panniculitis without vasculitis. Infiltration of neutrophils, vascular proliferation, hemorrhage, and necrosis of fat tissue is also reported. The isolation of a microorganism from the skin lesion or sometimes from blood specimen is required for a definitive diagnosis.^{2,8}

The clinical appearance of the skin lesions in our patient suggested the possible diagnoses of infective panniculitis or pyoderma gangrenosum, so to confirm the diagnosis a biopsy was performed. Pathologic examination revealed compact hyperkeratosis, parakeratosis and marked acanthosis. There were focal

dermal fibrosis with vascular proliferation and patchy infiltration of lymphocytes and some plasma cells. Necrosis of subcutaneous fat with paraseptal lymphocytic infiltration and mixed septal/lobular panniculitis were seen. According to figure 2A and 2B, wound culture at first showed growth of *Staphylococcus aureus*. A second culture from the wound revealed pseudomonas. The patient responded to appropriate antibiotic therapy. Clinical and pathological findings were in favor of the diagnosis of infectious panniculitis. The response of the lesions to antibiotics further confirmed our diagnosis and also ruled out other possible diagnosis such as pyoderma gangrenosum.

References

1. Patterson JW, Brown PC, Broecker AH. Infection-induced panniculitis. *J Cutan Pathol.* 1989; **16**: 183 – 193.
2. Morrison LK, Rapini R, Willison CB, Tyring S. Infection and panniculitis. *Dermatol Ther.* 2010; **23**: 328 – 340.
3. Johansson L, Thulin P, Low DE, Norrby-Teglund A. Getting under the skin: the immunopathogenesis of *Streptococcus pyogenes* deep tissue infections. *Clin Infect Dis.* 2010; **51**: 58 – 65.
4. Weedon D. Weedon's skin pathology. 3eds. China: Elsevier; 2010.
5. Kramer N, Rickert RR, Brodkin RH, Rosenstein ED. Septal panniculitis as a manifestation of Lyme disease. *Am J Med.* 1986; **81**: 149 – 152.
6. Ginter G, Rieger E, Soyer HP, Hoedl S. Granulomatous panniculitis caused by *Candida albicans*: a case presenting with multiple leg ulcers. *J Am Acad Dermatol.* 1993; **28**: 315 – 317.
7. Maiorillo RP, Merwin CF. North American blastomycosis presenting as an acute panniculitis and arthritis. *Arch Dermatol.* 1970; **102**: 92 – 96.
8. Diaz Cascajo C, Borghi S, Weyers W. Panniculitis: definition of terms and diagnostic strategy. *Am J Dermatopathol.* 2000; **22**: 530 – 549.