

Infection-Induced Panniculitis Associated with a Cardiac Abscess

Kyoung-Ae Jang, M.D., Jee-Ho Choi, M.D., Kyung-Jeh Sung, M.D.,
Kee-Chan Moon, M.D., Jai-Kyoung Koh, M.D.

*Department of Dermatology, College of Medicine, University of Ulsan,
Seoul, Korea*

Infection-induced panniculitis develops either through direct inoculation or as a manifestation of sepsis. However, it has rarely been considered as a disease entity within broader context of panniculitis. Moreover, panniculitis associated with a cardiac abscess without the evidence of sepsis has not been reported. We describe a case of infection-induced panniculitis associated with a cardiac abscess. We suggest that infection should be considered as a potentially important etiology of panniculitis, especially in the case of immunosuppression, and in such a case, meticulous efforts should be done to find the focus of infection.

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Key Words : Infection-induced panniculitis, Cardiac abscess

Infection-induced panniculitis develops either through direct inoculation or as a manifestation of sepsis. However, it has rarely been considered as a disease entity within broader context of panniculitis¹. Moreover, panniculitis associated with a cardiac abscess without the evidence of sepsis has not been reported. We describe a case of infection-induced panniculitis associated with a cardiac abscess.

CASE REPORT

A 19-year-old Korean man was referred from the department of infection. He had suffered from fever of unknown origin and skin lesions for 2 months. A skin examination revealed several thick crusted nodules and erythematous subcutaneous nodules on both the shins and fingers (Fig. 1). He complained of pain and tenderness from these skin lesions. They began as painful subcutaneous

nodules, later ruptured and became thick crusted nodules. Also, pityriasis versicolor on the anterior chest and striae distensae on both the shoulders were noticed. A histopathological examination of a subcutaneous nodule with an intact surface from the right shin showed mixed septal-lobular panniculitis (Fig. 2), accompanied with an infiltration of numerous neutrophils, histiocytes, and lymphocytes with vascular proliferation (Fig. 3). Another biopsy specimen from a crusted nodule on the shin showed necrosis of the epidermis and dermis with the formation of a crust. Fat tissue could not be seen. Stains for Giemsa, Ziehl-Neelsen and periodic acid-Schiff were all negative. However, Gram staining showed Gram-positive cocci in the cytoplasm of the histiocytic cells (Fig. 2). We concluded that the diagnosis of the skin lesions was infection-induced panniculitis. The patient had had a long-standing history of medication of systemic corticosteroids for facial palsy for 8 months before admission to our hospital. Laboratory studies revealed elevated levels of white blood cells (21,000/ μ l) (neutrophils 68%, lymphocytes 28%) and an increased erythrocyte sedimentation rate (55mm/hr). Organisms were not detected in the bacterial cultures from blood, urine, and sputum. However, methicilline-resistant *Staphylococcus*

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Reprint request to : Kyoung-Ae Jang, M.D., Department of Dermatology, Asan Medical Center, College of Medicine, University of Ulsan, 388-1, Poongnap-Dong, Songpa-Gu, Seoul, 138-736, Korea
Tel)(02)2224-3460/Fax)(02)486-7831

Fig. 1. Several black colored, thick crusted nodules and erythematous subcutaneous nodules on the right shin. The peripheral orange color was ascribed to the application of betadine.

Fig. 2. Photomicrograph showing mixed septal-lobular panniculitis (Hematoxylin-eosin stain, $\times 40$). Inset showed Gram-positive cocci (Gram stain).

Fig. 3. A number of inflammatory cell infiltrations in the septa in subcutaneous fat tissue (Hematoxylin-eosin stain, $\times 200$).

aureus (MRSA) grew from both of the crusted skin lesions and erythematous nodular ones. There

was no evidence of human immunodeficiency viral infection. The patient was started on vancomycin, 1.0g, intravenously twice a day. All the work-ups for the origin of the fever had failed until transesophageal echocardiography (TEE) was done, which was attempted due to an acute attack of ventricular tachycardia. Because TEE showed an abscess in the aortic valve area, he was operated on and a homograft replacement of the root of the aortic valve was performed. MRSA also grew on the culture from the abscess of the aortic valve. Gradually, his skin lesions as well as his general condition improved.

DISCUSSION

Patterson *et al.*¹ evaluated the histopathological findings in 15 cases of panniculitis due to infection. They concluded that the distinctive features associated with infectious panniculitis of diverse etiologies included: epidermal alterations such as acanthosis and parakeratosis; dermal edema with a diffuse or perivascular neutrophilic infiltrate; and mixed septal-lobular panniculitis with neutrophilic infiltration, vascular proliferation and hemorrhage, and necrosis.

In our case, histopathological examinations revealed mixed septal-lobular panniculitis with numerous cellular infiltrations, such as neutrophils, histiocytes, and lymphocytes, and vascular proliferation. Moreover, our case showed Gram-positive cocci on Gram staining and growth of MRSA from the skin lesion cultures. Our patient was in an immunosuppressed state owing to a long-standing history of medication of systemic corticosteroids. After the diagnosis of infection-induced panniculitis due to MRSA in our immunosuppressed patient, an accompanying cardiac abscess was detected. After treatment with vancomycin and a homograft replacement of the aortic valve, his skin lesions as well as his general condition gradually improved. Although we could not determine the precise pathway of the infection, we considered that the bacterial colonies in the skin lesions spread to the heart through the blood stream,

where they might have grouped into vegetating abscess.

Since the first description by Patterson et al.¹, no other studies have described this disease entity. We speculate that this might be due to its rarity or ignorance.

We suggest that infection should be considered as a potentially important etiology of panniculitis, especially in the case of an immunosuppressed state, and in such a case, meticulous efforts should be done to find the secondary focus of infection in internal organs.

REFERENCES

1. Patterson JW, Brown PC, Broecker AH. Infection-induced panniculitis. *J Cutan Pathol* 1989;16:183-93.