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Infective Endocarditis Presenting With Scalp Allodynia and a Giant Cell Arteritis–Like Vasculitic Phenotype

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Funds Collection G

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Patient: Male, 60-year-old
Final Diagnosis: Infective endocarditis
Symptoms: Fever • generalized stiffness • low back pain • scalp allodynia
Clinical Procedure: —
Specialty: Infectious Diseases • Rheumatology

Objective: Unusual clinical course

Background: The clinical manifestations of infective endocarditis (IE) are highly varied, and diagnostic delays are common and potentially life-threatening. Due to their overlapping clinical presentations, IE may be misdiagnosed as giant cell arteritis (GCA). Although IE mimics GCA, scalp allodynia associated with IE has not previously been reported.

Case Report: A 60-year-old man with mitral valve prolapse presented with fever, generalized stiffness, and low back pain refractory to oral antibiotics and low-dose glucocorticoids. He also reported a stinging sensation on his scalp while washing his hair, consistent with scalp allodynia. Prednisolone was initiated for presumed GCA. However, blood cultures subsequently grew *Streptococcus parasanguinis*, and imaging studies showed hematogenous dissemination. Ultimately, IE was confirmed based on the modified Duke Criteria. All symptoms, including scalp allodynia, resolved completely following the initiation of antibiotic therapy.

Conclusions: This case highlights that scalp allodynia, while characteristic of GCA, can also occur in IE, and underscores the limitations of relying solely on clinical symptoms when differentiating GCA from infectious etiologies. In patients with fever and scalp allodynia—particularly those with predisposing cardiac conditions—IE warrants strong consideration. Blood cultures should be obtained before initiating glucocorticoid therapy, and early clinical re-assessment is critical to avoid the potentially catastrophic consequences of missed or delayed IE diagnosis.

Keywords: Diagnosis, Differential • Endocarditis, Bacterial • Giant Cell Arteritis • Hyperalgesia • Scalp • Viridans Streptococci

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Introduction

Infective endocarditis (IE) is an infection of the endocardium and cardiac valves associated with substantial mortality. Its clinical manifestations are diverse, ranging from an acute course complicated by heart failure and systemic embolization to an indolent, subacute presentation with nonspecific symptoms such as low-grade fever, malaise, chills, sweats, dyspnea, back pain, arthralgias, and weight loss [1]. Although microembolic and immunologic phenomena such as Osler nodes, Janeway lesions, splinter hemorrhage, and conjunctival hemorrhage are well-known characteristic physical findings of IE, they are present in only 5% to 10% of patients, making IE challenging to suspect and diagnose [1,2].

IE can present with musculoskeletal symptoms, including morning stiffness, shoulder pain, and hip pain, through embolic or immunological mechanisms, thereby resembling the manifestations of giant cell arteritis (GCA) and polymyalgia rheumatica (PMR) [3,4]. Moreover, although uncommon, jaw claudication and amaurosis fugax, which are characteristic clinical features of GCA, have been reported in IE [4]. A partial response of these symptoms to glucocorticoid therapy has also been described [4]. For these reasons, IE is often misdiagnosed as GCA/PMR [4-10].

IE can also cause scalp tenderness [4], which is a well-recognized manifestation of GCA, occurring in 39.1% of patients [11]. This overlap may further obscure the distinction between IE and GCA, adding to the diagnostic challenge. In GCA, scalp tenderness frequently worsens with touching or brushing the hair, fulfilling the definition of allodynia: pain provoked by stimuli that do not normally cause pain [12]. Although scalp allodynia in the setting of IE has not previously been reported, its presence should not be regarded as excluding an infectious etiology. Here, we report a patient who presented with polyarthralgia, fever, and scalp allodynia in whom GCA was initially suspected, but IE was ultimately confirmed.

Case Report

The patient was a 60-year-old Japanese man with a history of moderate mitral regurgitation due to mitral valve prolapse, obstructive sleep apnea treated with continuous positive airway pressure for 10 years, and dyslipidemia. Mitral regurgitation was diagnosed 5 years earlier and was monitored with serial transthoracic echocardiography (TTE), with no progression observed, including on a study performed 6 months before admission. Regular medications included pitavastatin calcium and ethyl eicosapentaenoate. He noticed frequent gingival bleeding related to vigorous interdental brushing over the preceding month. He developed left lateral neck and left sternoclavicular joint pain 20 days before admission. Nineteen days before admission, a local orthopedic clinic noted erythema and warmth over the left sternoclavicular joint and provided analgesics. Sixteen days before admission, because the pain persisted, cefcapene pivoxil was initiated on suspicion of sternoclavicular joint arthritis. Fourteen days before admission, the development of stiffness in the left side of the neck, shoulder, back, and the anterior chest prompted evaluation at our hospital. Oral antibiotic therapy was discontinued, and 2 sets of blood cultures were obtained, both of which were negative. The symptoms persisted, and 10 days before admission, prednisolone 15 mg daily was administered for suspected PMR or crystal-induced arthritis, which resulted in mild improvement in pain. Eight days before admission, he developed fever. Prednisolone was discontinued, and colchicine was initiated 3 days before admission. Two days before admission, he noticed a stinging sensation on the scalp while washing his hair. On the day before admission, generalized stiffness and low-back pain developed; progressive pain and difficulty ambulating prompted presentation to the emergency department on the day of admission.

On presentation, he was alert, and vital signs were as follows: body temperature 38.8 °C, heart rate 108 beats/min, blood

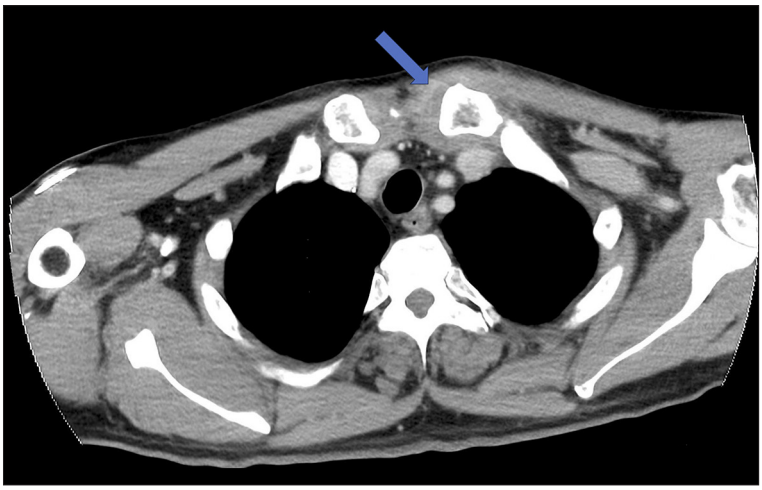

Figure 1. Contrast-enhanced computed tomography findings. A low-attenuation lesion at the left sternoclavicular joint was noted (arrow).

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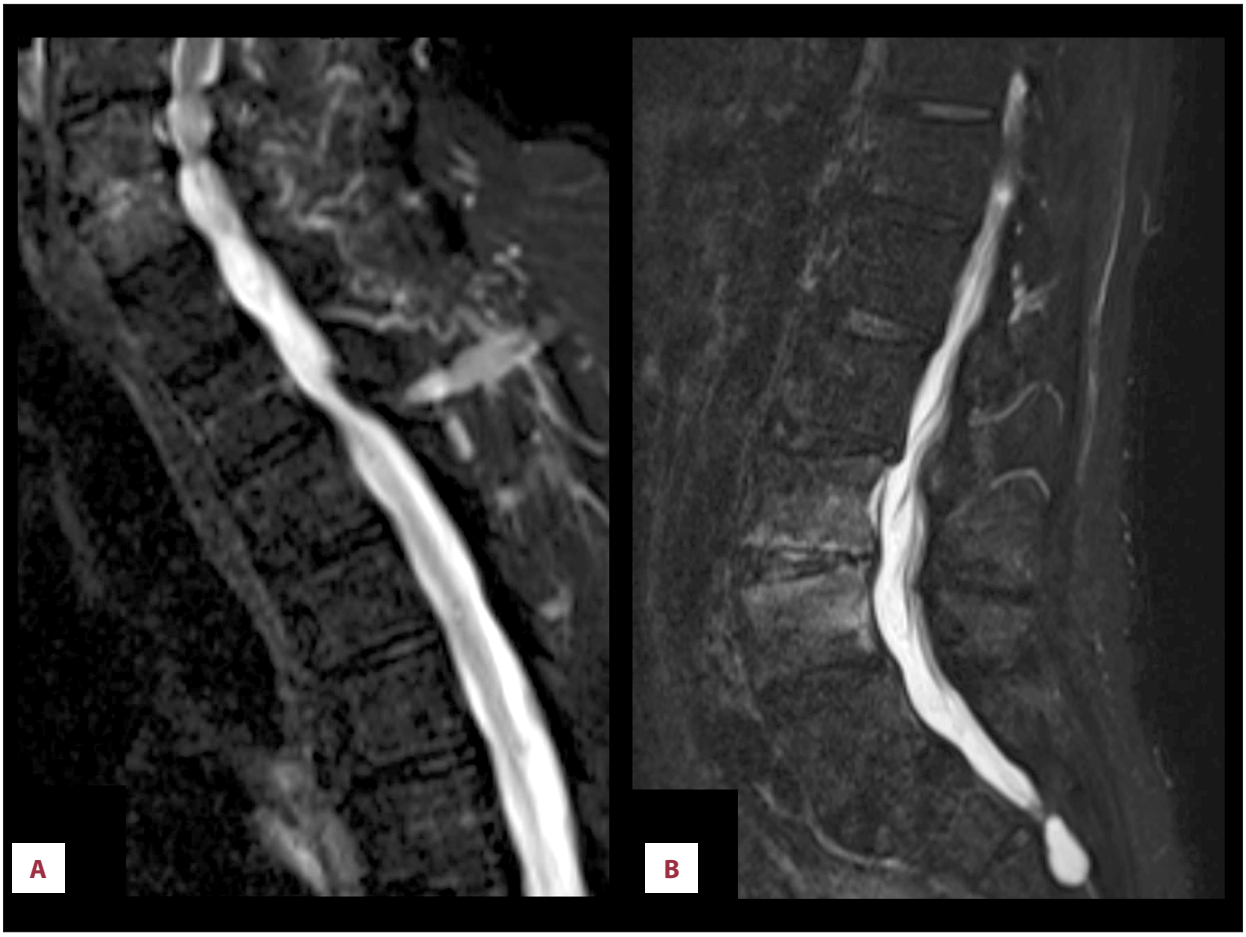

Figure 2. Spine magnetic resonance imaging findings. On short TI inversion recovery imaging, high-intensity lesions were revealed at the C6/7 (**A**) and L3/4 (**B**) intervertebral discs with adjacent vertebral bodies.

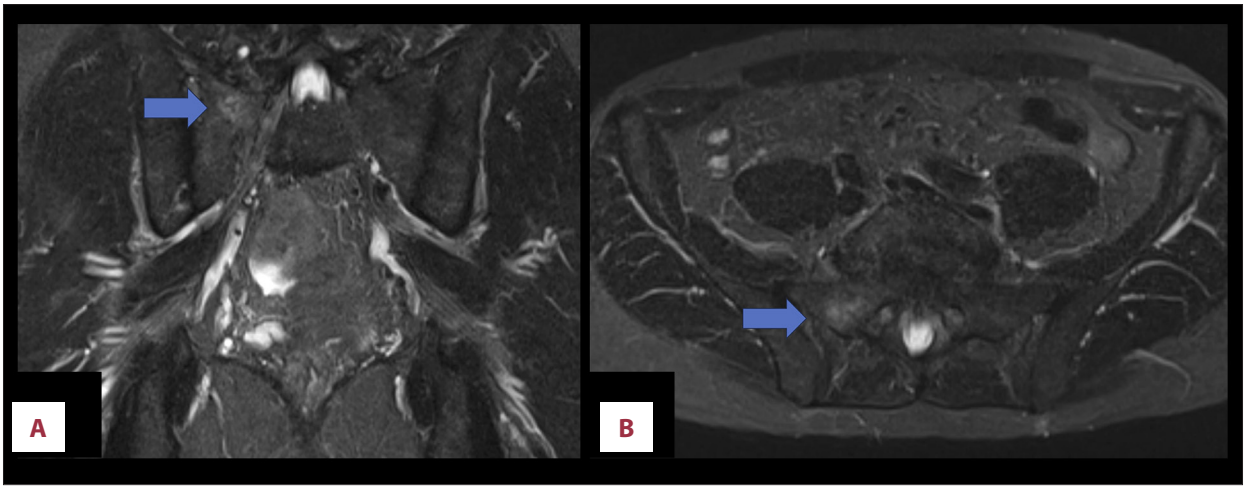

Figure 3. Pelvic magnetic resonance imaging findings. Arrows indicate high-intensity lesions in the right sacral ala on short TI inversion recovery imaging: (**A**) coronal, (**B**) axial.

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pressure 114/80 mmHg, respiratory rate 18 breaths/min, and oxygen saturation 96% on ambient air. Physical examination showed erythema, warmth, swelling, and tenderness of the left sternoclavicular joint, tenderness over the left posterior neck and the left side of L4-L5 vertebrae, and a grade 3/6 systolic murmur at the apex. A stinging sensation of the scalp was triggered by touching the hair, consistent with scalp allodynia. There was no thickening, induration, or engorgement of either temporal artery. On further review of systems, he reported headaches, chills, night sweats, malaise, and fatigue. Laboratory test results were unremarkable except for an elevated white blood cell count (11.4×10^3/μL), elevated C-reactive protein level (7.09 mg/dL), and elevated erythrocyte sedimentation rate (55 mm/h). Contrast-enhanced computed tomography (CT) revealed a low-attenuation lesion at the left sternoclavicular joint (**Figure 1**).

Differential diagnoses included GCA and IE. Oral prednisolone 1 mg/kg/day was promptly initiated as empirical treatment for possible GCA, considering the risk of development and progression of visual complications with delayed treatment. Additionally, a left temporal artery biopsy was planned. At the same time, although the initial blood cultures were negative, repeat blood cultures were obtained because the potential effect of prior antibiotic administration on the initial blood cultures was considered. All 4 blood culture bottles drawn at admission grew *Streptococcus parasanguinis*. The results, reported 16 hours after collection, became available 6 hours after glucocorticoid therapy had been initiated. Intravenous ceftriaxone was then started. Prednisolone was discontinued, and temporal artery biopsy was canceled. Further evaluation was performed on suspicion of IE. TTE revealed newly increased echogenicity on the posterior leaflet; however, it was difficult to determine whether this finding represented vegetation. The severity of mitral regurgitation remained unchanged. Spine and pelvis magnetic resonance imaging (MRI) revealed high-intensity lesions at the C6/7 and L3/4 intervertebral discs with adjacent vertebral bodies, as well as in the right sacral ala on short TI inversion recovery (STIR) imaging (**Figures 2, 3**), consistent with osteomyelitis. Brain MRI demonstrated a T2-weighted hypointense lesion in the right occipital lobe, consistent with intracranial hemorrhage (**Figure 4**).

The diagnosis of IE was confirmed in accordance with the modified Duke Criteria (1 major criteria: typical microorganism from 2 separate blood cultures; 3 minor criteria: predisposing heart condition, fever, and vascular phenomena [intracranial hemorrhage]), with metastatic involvement of left sternoclavicular arthritis, C6/7 and L3/4 vertebral osteomyelitis, and right sacral osteomyelitis. The need for transesophageal echocardiography (TEE) was discussed in consultation with an infectious disease physician and a cardiologist. Because TTE showed no definitive vegetations and the severity of mitral regurgitation remained unchanged from baseline, the likelihood of detecting surgical indications, such as large vegetations (>10 mm), periannular abscess, or valvular destruction, was considered low. Therefore, TEE was not performed. The patient was treated with antibiotics alone. After confirmation of susceptibility, antibiotic treatment was switched to intravenous ampicillin. All symptoms at presentation, including scalp allodynia, gradually resolved after treatment. Follow-up blood cultures taken 2 days after initiating ceftriaxone were negative. Intravenous antibiotic therapy was continued for 4 weeks after the clearance of bacteremia and then switched to oral clindamycin to complete a total of 6 weeks of therapy.

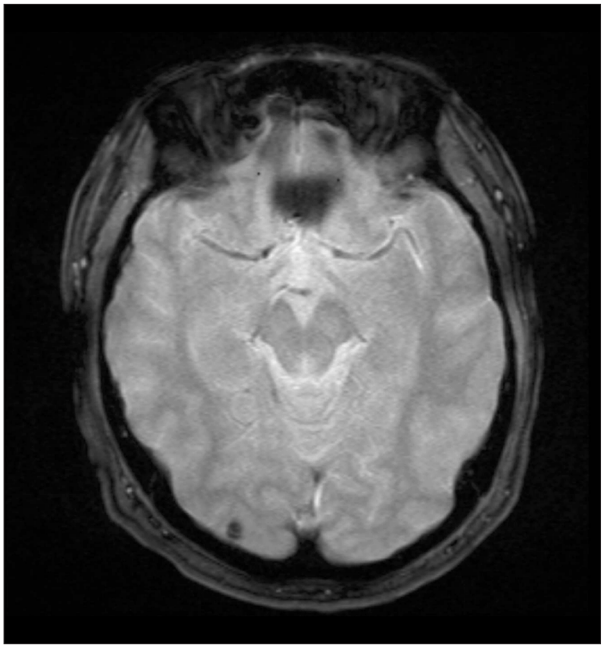

Figure 4. Brain magnetic resonance imaging findings. A T2-weighted hypointense lesion is present in the right occipital lobe.

Discussion

We present a patient with polyarthralgia, fever, and scalp allodynia in whom GCA was initially suspected but IE was ultimately diagnosed. The presumed pathological sequence in this case involved bacterial entry through gingival injury, followed by bacteremia and hematogenous dissemination, resulting in inflammatory involvement of the vertebral bodies and sternoclavicular joint, although microbiological evidence of infection at each site was lacking. Consequently, systemic infection secondary to IE can produce a constellation of findings that perfectly mimics GCA and PMR.

IE can involve the temporal artery and mimic GCA clinically and pathologically. Scalp tenderness has previously been

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reported in a patient diagnosed with IE, although neither the extent of tenderness nor the presence of scalp allodynia was specified [4]. A similar case has been reported of a patient who presented with transient monocular blindness and a painful right temporal nodule, was initially treated as GCA, and eventually received a diagnosis of IE caused by *Streptococcus mutans*. In that case, temporal artery biopsy revealed neutrophilic and mononuclear infiltrates involving the entire vessel wall, accompanied by intraluminal thrombus. Also, gram-positive cocci were observed in the perivascular connective tissue, suggesting dissemination of infection [6].

To the best of our knowledge, there are no previous reports that have specifically described scalp allodynia in IE. Although temporal artery biopsy was not performed in the present case and direct data on cranial cutaneous allodynia in IE are lacking, the mechanism of scalp allodynia in IE can be speculated by analogy to GCA as follows: (1) inflammatory infiltrates and emboli produce ischemia and tissue injury within vascular territories shared with cutaneous sensory fibers [13,14]; (2) vasculitic or embolic disorders in vasa nervorum result in neuropathic allodynia via peripheral sensitization or ectopic firing [15]; and (3) direct extension of inflammation from the vessel walls to adjacent nerves sensitizes nociceptive fibers [15]. In IE, septic emboli and inflammatory small-vessel vasculitis are well-recognized mechanisms of neurologic involvement, including ischemic infarcts and inflammatory vascular lesions in the brain and other organs, providing an example of infection-driven vascular injury that can affect neural tissue through embolic and vasculitic pathways [16]. Furthermore, temporal arteritis resembling GCA has been reported not only in IE but also in the setting of sepsis and bacteremia [17,18].

Scalp allodynia occurs in a restricted number of conditions, including migraine, herpes zoster, trigeminal neuralgia, temporomandibular disorders, and GCA. Among these conditions, the combination of scalp allodynia, fever, and elevated inflammatory markers (C-reactive protein and erythrocyte sedimentation rate) is particularly suggestive of GCA. In the present case, the subacute presentation of fever, generalized stiffness, pain, scalp allodynia, and elevated acute phase reactants led clinicians to suspect GCA. Involvement of the sternoclavicular joints and vertebral bodies, as observed in this case, has been reported in patients with GCA and PMR, further supporting this diagnostic consideration [19-22]. Because delayed treatment of GCA increases the risk of irreversible visual loss, guidelines recommend initiating glucocorticoid therapy before a definitive diagnosis is confirmed by temporal artery biopsy [23,24].

Clinicians often face a dilemma regarding the optimal timing of glucocorticoid initiation, as it may exacerbate IE [25]. Glucocorticoid therapy in patients with unrecognized IE can mask the symptoms of IE and lead to diagnostic delay. Also, it can possibly result in extensive valvular involvement and poorer outcomes by altering host defense mechanisms [26]. In the present case, a delay in the diagnosis of IE might likewise have allowed further valvular damage to progress, potentially necessitating surgical intervention or even resulting in a fatal outcome. A pragmatic approach is to consider the history, physical examination, and investigations available, and to weigh the probability of GCA against that of IE on a case-by-case basis. When the clinical probability of GCA outweighs that of IE, as in the present case, prompt initiation of glucocorticoids is reasonable. However, IE can present even in patients with scalp allodynia; therefore, blood cultures must be obtained before glucocorticoid administration, and patients should be closely monitored, with IE and other infectious mimics kept in the differential diagnosis. Prior antibiotic exposure is a well-recognized cause of false-negative blood cultures in IE [27]. Thus, when IE is suspected in patients with recent antibiotic use, repeat blood cultures after an appropriate antibiotic-free interval should be strongly considered if initial cultures are negative.

IE can present with a wide range of systemic manifestations. Our report highlights that clinical symptoms, physical examination findings, and routine laboratory tests are often insufficient to reliably distinguish GCA from IE. Therefore, clinicians should include IE in the differential diagnosis even when GCA is strongly suspected.

Conclusions

In conclusion, we report a case of IE presenting with scalp allodynia. Although the presence of scalp allodynia and fever generally leads clinicians to suspect GCA, IE should always be included in the differential diagnosis. When initiating glucocorticoids for suspected GCA, blood cultures should be obtained, and patients should be monitored closely for evidence of IE or an alternative diagnosis.

Institution Where Work Was Done

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Informed Consent

Informed consent for publishing this case report was obtained from the patient.

We used ChatGPT (OpenAI) to improve English phrasing and readability. The authors reviewed and verified all edits and take full responsibility for the content of the manuscript.

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Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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