Between Clinical Intuition and Serendipity: An Unusual Case of *Streptococcus Gordonii* Cholecystitis

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The most frequent pathogens isolated from bile cultures of patients with cholecystitis are anaerobes and *Enterbacterales* such as *E. coli, Klebsiella species*, and *Streptococcus species* [1].

Streptococcus gordonii belongs to the *Viridians streptococci* group of oral bacteria and is commonly associated with dental caries. *S. gordonii* has been previously reported as the causative pathogen in both endocarditis and spondylodiskitis [2]. However, it has rarely been associated with biliary infections. In this report, we presented a patient diagnosed with cholecystitis associated with *S. gordonii* infection.

PATIENT DESCRIPTION

A 77-year-old male presented to the emergency department due to severe fatigue and malaise over 3 days. Past medical history was notable for chronic renal failure, hypertension, and episodes of symptomatic bradycardia, for which a cardiac pacemaker was recently implanted.

On admission, the patient was alert. He had a temperature of 36.7°C, his blood pressure was 122/87 mmHg, and heart rate was 108 beats/minute. Physical examination was unremarkable.

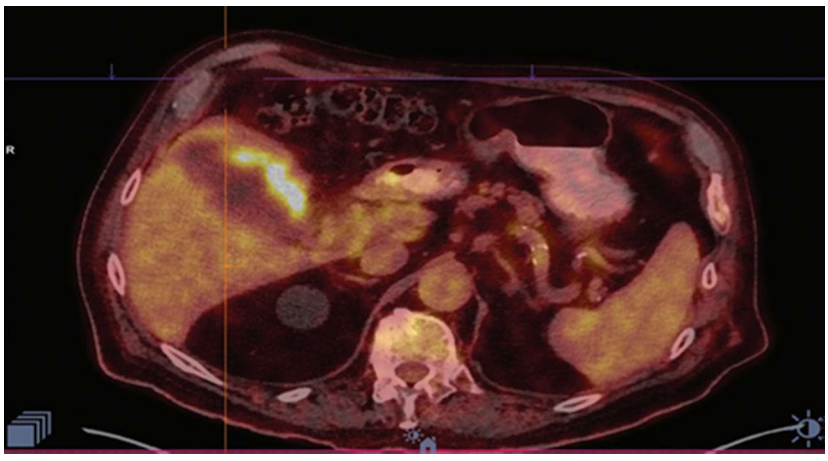
Laboratory tests showed high white blood cell (WBC) count of 19,800 cells/μL with neutrophil predominance (81%). Hemoglobin level was 12.9 g/dl (normal range 14-18 gr/dl), platelet count was 187,000/μL (normal range 150,000-450,000/μL). C-reactive protein (CRP) was markedly increased (24 mg/dl, normal range 0–0.5). Sodium level was 130 mEq/L (normal range 135–145), and mild hypokalemia was noted (3.1 mEq/L, normal range 3.5–5). Creatinine level was 2.16 mg/dl (normal range 0.66–1.25 mg/dl) and liver function tests were normal. Blood cultures were positive for *S. gordonii*, and ceftriaxone was initiated.

Detection of *S. gordonii* bacteremia in the presence of a cardiac pacemaker warranted consideration of pacemaker-electrode endocarditis. Transesophageal echocardiogram (TEE) was performed. However, no vegetations were demonstrated on the pacemaker electrodes or heart valves, and ^{18}F-fluorodeoxyglucose positron-emission tomography/computed tomography (^{18}F-FDG PET/CT) was performed. A strong pathological and irregular radiotracer uptake along the gallbladder wall was detected, with stranding of the pericholecystic fat and regional lymph node enlargement. No signs of heart valve or pacemaker electrode infection were noted [Figure 1].

Abdominal ultrasonography revealed a severely inflamed gallbladder with multiple bile stones obstructing the cystic duct. Percutaneous drainage of the gallbladder was performed. Bile fluid cultures were positive for *S. gordonii.*

The patient's condition gradually improved, and a follow-up ultrasound and a non-contrast CT scan 5 days later demonstrated decreased gallbladder wall enhancement, and reduced thickening of

Figure 1. The patient's ^{18}F-fluorodeoxyglucose positron-emission tomography/computed tomography showing strong pathological uptake along the gallbladder wall



the gallbladder wall. Ceftriaxone was administered for 12 days and repeat blood cultures were negative. The patient was discharged after an elective cholecystectomy was scheduled.

COMMENT

Acute cholecystitis accounts for as many as a third of all admission to surgical wards. Patients typically present with unremitting right upper quadrant pain, anorexia, nausea, vomiting, and fever. Cholecystitis is associated with female sex, pregnancy, obesity, old age, and hemoglobinopathies. Diagnosis relies on physical examination, laboratory tests and different imaging modalities such as abdominal ultrasound, CT, or hepatobiliary iminodiacetic acid (HIDA) scan.

Pathogens commonly associated with acute cholecystitis originate from the patient's indigenous bile flora and include *Enterbacterales* such as *E. coli*, *Klebsiella species*, *Enterococci*, *Streptococcus species*, and anaerobes [1]. *S. gordonii*, a member of the *viridians* group, is a facultative-anaerobic gram-positive bacterium that typically resides in the human oral cavity. It is an important etiological agent of dental caries, where it can cause the dissolution of tooth enamel using acidic end-products of carbohydrate metabolism [3].

Although most commonly isolated from the oral flora, *S. gordonii* can cause bacteremia, which may be complicated by infective endocarditis, spondylodiskitis, or septic arthritis [2].

Acute cholecystitis attributed to *S. gordonii* is an exceedingly rare phenomenon. To the best of our knowledge, ours is the second case of *S. gordonii* cholecystitis reported in the literature. The first report was in a study investigating the spectrum of pathogens identified in bile samples of patients with cholecystitis using next generation sequencing [4]. Interestingly, the findings of acute cholecystitis detected by ^{18}F-FDG PET/CT scan and *S. gordonii* as the causative pathogen, were both surprising.

Indeed, ^{18}F-FDG PET/CT scan is a useful imaging modality in the detection and follow-up of oncologic and hematologic malignancies as well as fever of unknown origin. It may help distinguish malignant from benign gallbladder lesions based on the intensity of radio-tracer uptake [5]. Although an infectious process involving the gallbladder may mimic a neoplastic lesion, The pericholecystic fat stranding, in addition to the strong irregular gall bladder uptake, suggests infectious cholecystitis rather than malignancy.

In this case report, we highlight the dissonance between clinical intuition and the usefulness of ^{18}F-FDG PET/CT. Clinical intuition led to the pursuit of endocarditis in a patient with a known pathogen and presence of implanted cardiac pacemaker, but ^{18}F-FDG PET/CT pointed to the accurate source of infection, despite an unusual pathogen and subtle clinical and laboratory findings. This finding is especially important in geriatric patients where *S. gordonii* has been shown to be associated with a wide array of clinical manifestations with non-specific presenting symptoms, such as in our case.

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Capsule

Make your own neoantigens

Immunotherapies activate the body's own immune system to fight cancer, but their effectiveness is limited by their ability to distinguish cancer cells from healthy cells. Chemical inhibitors are a mainstay of cancer therapy, but they too have limitations because cancer cells frequently become resistant to drug treatment. Putting the two together, **Hattori** and co-authors developed an approach that draws on the strengths of both drug therapy and immunotherapy. The authors first treated lung cancer with the drug sotorasib, which targets *KRAS*, a mutant gene involved in cell signaling pathways that control cell growth, cell maturation, and cell death. Engineered antibodies were then administered that specifically recognize neoantigens created by mutant *KRAS* protein bound to sotorasib. The antibodies killed even sotorasib-resistant cancer cells but spared normal cells that do not have mutant *KRAS*.

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