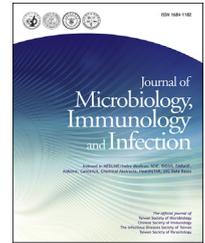




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Letter to the Editor

Thrombophlebitis of superior mesenteric vein with bacteremia of *Gemella sanguinis* and *Streptococcus gordonii*



KEYWORDS

Mesenteric thrombosis;
Pylephlebitis;
Odontogenic infection;
Sepsis

Abstract Pylephlebitis is a condition with thrombophlebitis of the portal mesenteric venous system. Herein, we report a patient suggesting odontogenic bacteremia as a risk factor of pylephlebitis. He was diagnosed as superior mesenteric vein thrombophlebitis, and blood cultures grew *Gemella sanguinis* and *Streptococcus gordonii*.

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Dear Editor,

Pylephlebitis, or suppurative thrombophlebitis of the portal mesenteric venous system has been known to result from an infection in the regions drained by the portal system.¹ The common predisposing infections include diverticulitis, appendicitis, pancreatitis, and inflammatory bowel disease. Hypercoagulable states including malignancy were also reported as additional risk factors.² Herein, we report a case of superior mesenteric vein (SMV) thrombophlebitis with bacteremia of *Gemella sanguinis* and *Streptococcus gordonii*.

A 49-year-old man visited complaining of fever and chills. He also had abdominal pain of 2-week duration, which had shown no improvement with medications. He was a non-smoker and his past medical history was unremarkable. His initial blood pressure was 113/74 mmHg, pulse was 91 beats per minute, and temperature was 38.3 °C. There was generalized tenderness with palpation of his abdomen, yet no rebound tenderness. His chest was clear without murmurs.

Laboratory studies disclosed the following values: leukocyte count of 15.15×10^9 cells/L with 92% neutrophils, platelet count of 114×10^9 cells/L, C-reactive protein level of 27.45 mg/dl (reference range 0.00–0.50 mg/dl), total bilirubin of 3.4 mg/dl, aspartate aminotransferase of 55 IU/L, alanine aminotransferase of 51 IU/L, amylase of 26 IU/L,

lipase of 27 IU/L, and d-dimer of 4.04 µg/ml. Contrast-enhanced computed tomography (CT) revealed hypo-attenuated thrombosis in SMV with increased fatty infiltration in mesentery (Fig. 1). There were no abnormal findings in the liver, biliary tract, pancreas, or bowels. Empirical treatment with intravenous piperacillin-tazobactam started with an unfractionated heparin infusion.

A transthoracic echocardiogram showed no evidence of endocarditis. Two blood cultures on admission were shown to be *G. sanguinis* and *S. gordonii*. Detailed history was retaken and the patient reported having had a mandibular tooth extraction followed by dental implant 8 months prior to the onset of his symptoms. Thereafter, he had regular dental follow-ups including intermittent oral hygiene procedures. Based on in vitro activity, the patient was switched to penicillin G intravenously. He was switched to oral amoxicillin, bridged to coumadin, and discharged on hospital day 24. At outpatient follow-up, the patient reported no symptoms, and laboratory results were normalized.

The bacterial flora of the mouth could lead to a bacteremic state ranging from 13% to 96%,³ when there is tissue trauma induced by dental procedures. On occasions, this may lead to seeding of organisms in different target organs, resulting in acute or chronic infections. Bacterial endocarditis is a well-known complication of such bacteremia. Yet, multiple severe complications of odontogenic infections have been reported, such as descending necrotizing

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Figure 1. Contrast-enhanced CT scan. Sagittal and coronal view of thrombosis in the superior mesenteric vein.

mediastinitis, Lemierre's syndrome,⁴ septic cavernous sinus thrombosis,⁵ and vertebral osteomyelitis.

We assume that *S. gordonii* could evade the immune system of the host, play an integral role by creating surfaces for *G. sanguinis* to adhere to, and cause thrombophlebitis in SMV in our patient. To our knowledge, this is the first case report of SMV pylephlebitis with a bacteremia of *G. sanguinis* and *S. gordonii* along with antecedent dental surgery, suggesting odontogenic bacteremia as a risk factor of pylephlebitis.

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Ji Hye Kim

Department of Emergency Medicine, Inha University School of Medicine, Incheon, South Korea

Hea Yoon Kwon

Department of Internal Medicine, Inha University School of Medicine, Incheon, South Korea

Areum Durey*

Department of Emergency Medicine, Inha University School of Medicine, Incheon, South Korea

*Corresponding author. Department of Emergency Medicine, Inha University Hospital, 7-206, Shinheung-Dong, Jung-Gu, Incheon, 22332, South Korea. Fax: +82 32 890 2307.

E-mail address: areum.durey@gmail.com (A. Durey)

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