We postulate that the use of empirical oral corticosteroids in this patient attenuated an already impaired host response to HSV-1 infection and allowed progression of ulceration through the esophageal wall. This case illustrates the need for aggressive evaluation of esophageal symptoms in HIV-infected patients, rather than empirical treatment for presumed aphthous ulceration. Appropriate therapy may then be directed toward the identified pathology.

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References

Figure 1. Meglumine diatrizoate swallow demonstrating extravasation of contrast into the pleural space (arrow) in an AIDS patient with Boerhaave’s syndrome due to herpes simplex virus type 1 esophagitis.

Polymicrobial Endocarditis with Haemophilus parainfluenzae in an Intravenous Drug User Whose Transoesophageal Echocardiogram Appeared Normal

Polymicrobial endocarditis with Haemophilus parainfluenzae in intravenous drug users constitutes a distinct clinical syndrome [1] characterized by initially positive blood cultures that yield a common gram-positive organism, subsequent microbiological identification of H. parainfluenzae, prominent pleuritic chest pain due to septic pulmonary emboli, salivary contamination of needles, and large tricuspid valve vegetations seen on an echocardiogram. This constellation of findings has permitted the prospective diagnosis of H. parainfluenzae endocarditis and the initiation of therapy before the organism is identified by culture. We describe a patient with this specific syndrome but without tricuspid valve vegetations on a transthoracic echocardiogram.

A 34-year-old pregnant female (gravida 3, para 2) presented to the hospital at 30 weeks’ gestation with fever, chills, and night sweats of 2 weeks’ duration. She was known to use intravenous cocaine and to clean her needles with saliva. She was febrile...
(temperature to 104°F) and tachypneic. Physical examination revealed a pleural friction rub, and a chest radiograph revealed a wedge-shaped opacity in the left lung base. Cultures of blood drawn on admission yielded *Staphylococcus aureus*. Antibiotic therapy with vancomycin was started. A lung perfusion scan showed multiple segmental perfusion defects consistent with multiple pulmonary emboli (figure 1). Doppler ultrasonographic studies and a venogram were negative for lower extremity and pelvic vein thrombosis. Findings on a transesophageal echocardiogram were normal.

The patient’s condition appeared to improve, but 1 week later she developed spiking fevers and chills. Antibiotic therapy was changed to cephalazidime, but her symptoms continued. Cultures of blood drawn on admission yielded delayed growth of *Haemophilus parainfluenzae* on chocolate agar. She received treatment with intravenous ampicillin for 4 weeks. The patient became afebrile and was discharged. She was well 6 months after discharge and was polymicrobial endocarditis with *Haemophilus parainfluenzae*.

Raucher et al. [1] called attention to the unique clinical and microbiological characteristics of polymicrobial endocarditis involving *H. parainfluenzae* in intravenous drug users. These investigators described 10 patients with strikingly similar clinical presentations: prominent pleuritic chest pain, septic pulmonary emboli, salivary contamination of needles, and delayed growth of *H. parainfluenzae* in culture. All of the patients had one or more large tricuspid valve vegetations. Salivary contamination of needles appears to be an important factor in acquiring this organism. In all the previous reports of this syndrome, initial blood cultures yielded a common, easily cultured gram-positive organism [2–5]. Therapy has typically been directed against the organism first isolated in culture, and the results have been equivocal, as was true for our patient. Therapy was never completely successful unless it included antibiotics that were effective against *H. parainfluenzae*. Because of the typical features, a diagnosis of *H. parainfluenzae* endocarditis never was determined for three of the patients in Raucher’s series before the results of cultures were available.

The echocardiographic characteristics of *H. parainfluenzae* endocarditis are large, easily recognized tricuspid valve vegetations. Although our patient had all the characteristics of previously described patients with this clinical syndrome, and cultures yielded delayed growth of *H. parainfluenzae*, repeated echocardiographic studies, including transesophageal echocardiography, failed to demonstrate vegetations on the tricuspid valve. The clinical value of this novel observation (Raucher’s syndrome without echocardiographically evident vegetations) lies in recommending early presumptive therapy directed at *H. parainfluenzae* while awaiting the results of cultures, even if findings on an echocardiogram are normal, as long as the other characteristics of this syndrome are present.

For intravenous drug users, the clinical syndrome of polymicrobial endocarditis with *H. parainfluenzae* is well described and sufficiently distinct to permit therapy directed against this organism before culture results provide confirmation of the diagnosis. Prior reports calling attention to this syndrome have described patients with echocardiographically demonstrable tricuspid valve vegetations. The findings in this report demonstrate that a negative transesophageal echocardiogram should not deter physicians from making a diagnosis of *H. parainfluenzae* “endocarditis” and instituting appropriate therapy when the classic features of this syndrome are present.

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