also at the emergency department 30 minutes after the onset of symptoms. On admission, the systolic blood pressure was 60 mm Hg. Right hemiparesis, right homonymous hemianopia, complete aphasia, and a generalized maculopapular erythema were detected on physical examination. Intravenous fluids, epinephrine, antihistamines, and steroids were administered. The hypotension, fever, and rash resolved, but the complete aphasia and mild hemiparesis persisted. Blood cell counts and coagulation parameters were within normal limits. Cerebral MRI revealed a large ischemic infarct involving the left frontal, parietal, and temporal lobes. Doppler ultrasonography was performed and ruled out stenoses of the extracranial arteries, and echocardiography was normal. CSF parameters were also within normal limits. A serology for IgE antibodies to rifampin, from a blood sample obtained 4 days after the episode, was negative within normal limits. A serology for IgE antibodies to rifampin, from a blood sample obtained 4 days after the episode, was negative within normal limits. A serology for IgE antibodies to rifampin, from a blood sample obtained 4 days after the episode, was negative within normal limits. A serology for IgE antibodies to rifampin, from a blood sample obtained 4 days after the episode, was negative within normal limits. A serology for IgE antibodies to rifampin, from a blood sample obtained 4 days after the episode, was negative within normal limits. A serology for IgE antibodies to rifampin, from a blood sample obtained 4 days after the episode, was negative within normal limits. A serology for IgE antibodies to rifampin, from a blood sample obtained 4 days after the episode, was negative within normal limits.

Allergic or hypersensitivity reactions to rifampin occur occasionally, although the frequency of occurrence is low. In a recent large series, flu-like syndrome, rash, acute renal failure, thrombocytopenia, and hypotension occurred in 0.26%, 0.07%, 0.1%, 0.01%, and 0.01% of patients, respectively [1]. This type of toxicity is almost always associated with intermittent, high-dose rifampin administration (900–1,200 mg two-to-three times a week), and is rare with dosages of 600 mg daily or twice weekly [2, 3]. However, among patients with HIV infection, adverse reactions to antituberculous treatment are more common (~10% of cases) [4]. It is possible that this increased susceptibility is caused by the overproduction of IgE which results from the shift in CD4 cells from the Th1 to the Th2 type [5].

In an extensive search of the literature using MEDLINE (1966 to the present) and references from the selected articles, we found no published case of cerebral infarcts related to rifampin treatment. As is true for the patient we described, most reported cases of anaphylactic reactions to rifampin presented with prodromes, generally while receiving the drug on a daily regimen, and developed anaphylaxis upon re-exposure to rifampin. Our patient seemed to develop flu-like symptoms initially, an uncommon event apparently not related to anaphylaxis, and ultimately developed anaphylactic symptoms, suggesting that patients with a previous history of adverse reaction to rifampin who are re-exposed to the agent should be under medical supervision during re-exposure.

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References

**Propionibacterium acnes Sepsis in a Previously Healthy Man**

*Propionibacterium acnes*, an organism normally found on the skin, usually has a low level of virulence. However, it can cause serious infections, especially those associated with implanted cardiac or neurogenic prostheses or endocarditis on previously damaged valves [1, 2]. We describe a healthy man who developed sepsis caused by *P. acnes* that was isolated in cultures of blood and bone marrow; although endocarditis was suspected, it was not definitely documented.

A 35-year-old man presented to our hospital in November 1997 because of a 6-day history of intermittent fever (temperature as high as 40°C [104°F]), headache, and dry cough. He was born in Syria and had been there 3 months before admission. His medical history was unremarkable. At the time of admission, the patient appeared in good general health. His body temperature was 38°C (100.4°F), and his blood pressure was 120/80 mm Hg. The pulse rate was 76, and the respiratory rate was high at 28. Physical and neurological examinations showed no abnormalities. The WBC count was 5,000/mm³ (82% granulocytes).

Laboratory tests revealed the following abnormal values: serum iron, 18 mg/dL; iron-binding capacity, 1.82 g/L; ferritin, 380 ng/mL; platelets, 123,000/mm³; erythrocyte sedimentation rate, 31 mm/h; C-reactive protein, 111 mg/L; aspartate aminotransferase, 117 U/L; alanine aminotransferase, 168 U/L; lactate dehydrogenase, 825 U/L; γ-glutamyltransferase, 111 U/L; alkaline phosphatase, 498 U/L; prothrombin time, 1.23; and partial thromboplastin time, 1.27. Testing for hepatitis B surface antigen was negative, as was the VDRL (Venereal Disease Research Laboratory) test. Urinalysis revealed 50 RBCs per high-power field and >300 mg of protein/dL. Blood gas analysis showed the following: pH, 7.49; PO₂, 69 mm Hg; PCO₂, 27.1 mm Hg; and oxygen saturation of hemoglobin, 95.3%. Testing for antibodies to HIV, hepatitis A virus, hepatitis C virus, human cytomegalovirus, Epstein-Barr virus, *Toxoplasma gondii*, *Leishmania* species, *Leptospira* species, *Rickettsia conorii*, *Rickettsia typhi*, and *Borrelia burgdorferi* was...
negative, as was testing for cryptococcal antigens and *Mycobacterium tuberculosis* in urine.

Examination of a blood smear was negative for *Plasmodium* species. Widal’s test was negative, as was Wright’s staining. Two stool specimens were negative for parasites. Eight aerobic and eight anaerobic blood cultures were negative after 5 days. Intradermal testing with PPD (5 IU) was negative.

Over the following days, high-grade fever persisted, testing revealed worsening of liver function, and hepatosplenomegaly was found; therefore, other tests were performed for rheumatoid factor, antinuclear antibodies, antibodies to extractable nuclear antigens, and antineutrophil cytoplasmic antibodies, and the results were negative. Complement components C3 and C4 and lymphocyte subpopulations were normal.

An ultrasound scan of the abdomen showed an enlarged spleen (bipolar diameter, 185 mm) with homogeneous structure and low-grade hepatomegaly. A chest roentgenogram demonstrated elevation of the right hemidiaphragm; the heart appeared normal. A CT of the abdomen revealed hepatomegaly and splenomegaly. Transthoracic echocardiography showed thickening of mitral valve leaflets consistent with the presence of endocardial vegetations. Transesophageal echocardiography demonstrated only minimal thickening of the mitral valve leaflets without vegetations. A CT scan of the brain was unremarkable. A high-resolution CT scan of the thorax showed coarseness of the right posterior pleural margin with thickening of lung parenchyma, findings consistent with fibrosis.

Aspiration biopsy of bone marrow was then performed. Cultures of bone marrow were positive for *P. acnes* after 7 days. Histological studies of a transcutaneous liver biopsy specimen showed an area with nonspecific inflammatory infiltrates. Culture of the biopsy specimen was negative. Blood specimens for aerobic and anaerobic cultures were obtained again. After 10 days, three anaerobic and two aerobic cultures yielded *P. acnes*.

The patient was treated with ampicillin (3 g iv q.i.d. for 5 weeks), netilmicin (200 mg iv b.i.d. for 15 days), and doxycycline (100 mg b.i.d. orally for 21 days). Defervescence occurred, and the laboratory values returned to normal; hepatosplenomegaly resolved.

In conclusion, our patient met the 1992 ACCP/SCCM (American College of Chest Physicians/Society of Critical Care Medicine) Consensus Conference Committee criteria for sepsis (SIRS [systemic inflammatory response syndrome] plus infection) [3]. A definite diagnosis of endocarditis was not made. To our knowledge, this is the first description of sepsis and bone marrow infection due to *P. acnes* in a previously healthy individual. Our case report points out the importance of incubating cultures of blood and tissue samples, even those from patients without cardiac or neurogenic protheses, for at least 14 days to isolate *P. acnes*.

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**References**


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**Superior Vena Cava Syndrome Secondary to Syphilitic Aneurysm of the Ascending Aorta in a Human Immunodeficiency Virus–Infected Patient**

Superior vena cava syndrome (SVCS), a well-known entity resulting from obstruction of the superior vena cava, usually occurs because of mediastinal tumors [1]. We describe a patient with previously undiagnosed HIV infection who developed SVCS because of a syphilitic aneurysm of the ascending aorta.

A 48-year-old man was admitted to our hospital (Santander, Spain) because of progressive swelling and redness of his face, neck, and arms of 4 days’ duration. He was a nonsmoker, and he did not give a history of venereal disease. He denied having any homosexual relationships, but there was a history of sexual contact with prostitutes.

His blood pressure was 130/80 mm Hg in each arm. Arterial pulses were brisk in all extremities. His face and neck were diffusely swollen, and his jugular veins were distended bilaterally to the angle of the mandible while he was examined in an upright position.

Laboratory findings included a hemoglobin concentration of 10.6 g/dL, a normal mean corpuscular volume, a WBC count of 5,200/mm³ (20% lymphocytes), and an erythrocyte sedimentation rate of 74 mm/h. Chest radiographs showed a large anterior mediastinal mass (figure 1). A CT scan of the chest showed an aneurysm of the ascending thoracic aorta, with partial thrombosis, compressing the superior vena cava. Venereal Disease Research Laboratory (VDRL) and *Treponema pallidium* hemagglutination (TPHA) tests on serum were positive (titers, 1:256 and 1:1,280, respectively). Evaluation of a CSF sample with use of VDRL and TPHA tests was negative. Serologies for antibodies to HIV were positive, both ELA and Western blot. The CD4⁺ lymphocyte count was 475/mm³, and the plasma viral load was 17,000 copies/mL.

During a thoracotomy, a large saccular aneurysm involving the entire ascending aorta and compressing the superior vena cava was resected. Histological evaluation of the resected aneurysm showed miliary gummatus in the tunica media and scant multinucleated cells between the epithelioid cells of the granulomas. Postoperative re-