A 50-year-old ex-smoker has been complaining of a 5-month history of morning cough that is productive of small amounts of discolored phlegm, fever, a 10-pound weight loss, fatigue, and a lesion on his face (Figure 1). The lesion drains, tends to heal over for a few days, then breaks down and discharges pus containing sand-like yellow-to-white particles. The patient, who had undergone coronary artery bypass graft surgery 5 years before, takes no medications, admits to drinking two glasses of wine each night, and has no other complaints other than that his gums occasionally bleed when he brushes his teeth. Physical examination reveals a temperature of 38.2°C, blood pressure 140/80 mm Hg, pulse 84 beats/min, respirations 26 breaths/min, and crackles heard over the anterior portion of the right chest. A chest radiograph is obtained (Figure 2). The metal clips in the mediastinum are incidental findings from a prior coronary artery bypass operation. Which of the following diseases does the patient most likely have?

A. Bronchogenic carcinoma
B. Actinomycosis
C. Staphylococcal infection
D. Tuberculosis
E. Sweet’s syndrome
Answer: B. Actinomycosis.

The patient under discussion has disseminated actinomycosis. It initially began as a thoracic infection that spread to the skin. Clues to the diagnosis are as follows: 1) the presence of risk factors for aspiration of an excessive amount of anaerobic oropharyngeal secretions (e.g., bleeding gums and at least 2 glasses of wine each night); 2) indolent nature of the illness; 3) the classic description of the patient seeing and feeling sulfur granules in the draining skin lesion; and 4) the disrupted interlobar pleura (see arrow in Figure 3 which points to the disruption) where the infection spread from one lobe to another across the minor fissure. The diagnosis was confirmed by crushing, staining and examining granules from the drainage of the patient’s face lesion, and anaerobically culturing the same material. The smear revealed a sulfur granule (Figure 4); the culture grew *Actinomyces israelii*.

Actinomycosis is a term used to describe an anaerobic or microaerophilic infection caused by bacteria in the genus Actinomyces. While nine species have been implicated in human disease, *A. israelii* accounts for the majority of human cases. These bacteria are Gram-positive, pleomorphic organisms that range in appearance from bacillary to long, branching filamentous forms that may simulate fungal hyphae. While these organisms are not acid-fast (in contrast to Nocardia species) when stained by the Ziehl-Neelsen method, they can be when stained with the method of Putt.

The organisms that cause actinomycosis are part of the indigenous, microbiological flora of the upper respiratory and lower gastrointestinal tracts. The pathogenesis of thoracic actinomycosis includes aspiration of oropharyngeal secretions, extension from cervicofacial infection through the mediastinum to the pleura or lungs, or extension from abdominal infection through the diaphragm.

Because infection with these organisms typically ignores anatomical boundaries, pneumonia is almost always complicated by pleural involvement. Delayed diagnosis and treatment of pleural infection can lead to spread of infection to bone and soft tissue with cutaneous draining sinus tracts, and damage to oral mucosa can lead to cervicofacial infection that presents as a lumpy jaw (Figure 5). Lumpy jaw refers to the lumpy appearance of the subcutaneous tissue around the mandible created by subcutaneous induration and edema due to the spread of this infection. In Figure 5, the infection extends from the middle of the figure, corresponding to the most proximal aspect of the mandible, to the right hand margin.

Sulfur granules (so-named because of their yellow color in tissue) are comprised of masses of matted, interconnected bacterial filaments. They form because the organisms secrete a polysaccharide-containing protein that cements filaments together. Although characteristic of actinomycosis and extremely useful in narrowing the diagnostic possibilities, the presence of sulfur granules is not
specifically diagnostic of this disease. They have been found in botryomycosis of the skin due to staphylococci, mycetomas of the skin due to Nocardia species, and visceral botryomycosis due to Pseudomonas and Proteus species and *Staphylococcus aureus*. There is no convincing evidence that visceral infections due to Nocardia species are associated with sulfur granules.

The other listed diseases were not likely to cause our patient’s constellation of findings for the following reasons: bronchogenic carcinoma, tuberculosis, and Sweet’s syndrome (acute febrile neutrophilic dermatosis that can involve the lung) have not been associated with sulfur granules; and staphylococcal lung infection has been reported to disrupt pleural surfaces only when it causes a lung abscess, and this was not present in our patient. In addition to actinomycosis and lung abscess, other diseases that have been reported to disrupt pleural surfaces are nocardiosis, bronchogenic carcinoma, tuberculosis, and a variety of fungal infections.

**Selected Readings**


Varkey B. Sulfur granules. JAMA 1982; 248:3025