Mycobacterial lymphadenitis, especially involving the cervical lymph nodes, is known as scrofula or scrofuloderma when extension to the cutaneous surface occurs. Descriptions of scrofula date as far back as ancient Greece when Hippocrates commented on a tuberculosis patient with lymphadenopathy and overlying skin changes.¹

Scrofula in the immunocompetent patient is usually a disease of young children, most often caused by *Mycobacterium avium* complex (MAC) or *Mycobacterium scrofulaceum*, whereas the majority of cases in adults are due to tuberculosis.² However, in the adult HIV population, scrofula associated with atypical mycobacteria is becoming more common. For instance, in many areas of the United States, including Baltimore, coinfection with HIV and *Mycobacterium kansasii* is now more common than HIV and *Mycobacterium tuberculosis*.² Although most patients coinfected still develop pulmonary disease only, extrapulmonary disease including scrofula and widely disseminated infection accounts for close to 33% of cases.

The highest concentrations of *M. kansasii* cases are reported from the central and southern United States.¹² Although the natural ecologic niche for this organism is unknown, it has been cultured from tap water in endemic areas but not natural water or soil. Transmission is not thought to occur through person-to-person contact.

Pulmonary infection with *M. kansasii* causes a clinical picture similar to tuberculosis, including similar radiologic changes. Infection usually begins as CD4 counts fall below 50 cells/mm³. Scrofula most often involves the lymph nodes of the head and neck, as seen in our patient. Diagnosis rests on histologic examination of a lymph node showing caseating granulomas with or without acid-fast bacteria and a negative PPD (purified protein derivative). Cultures provide definitive diagnosis. Tissue can be obtained by fine needle aspiration or excisional biopsy if necessary. Incisional biopsies are not recommended because of the risk of fistula formation.

Recommended treatment for *M. kansasii* infection includes isoniazid (300 mg/d), rifampin (600 mg/d), and ethambutol (15 mg/kg/d) for 18 months.²⁴ Rifampin sensitivity testing is recommended for *M. kansasii* as resistance has been growing. Rifabutin or clarithromycin are substituted in patients on protease inhibitors due to interactions with rifampin. Surgical excision is the treatment of choice in immunocompetent children. When this is not feasible, the addition of clarithromycin to the previous medication regimen is recommended.

References

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