Pneumocystis carinii

Pneumocystis carinii is an important cause of pneumonia in immunocompromised individuals. The classification and life cycle of Pneumocystis are unclear. Many aspects of its biochemistry indicate that it is yeast, but it also has several attributes of a protozoan. An analysis of rRNA sequences and mitochondrial DNA and of various enzymes supports the idea that it is a fungus. However, the findings that it does not grow on fungal media and that antifungal drugs are ineffective have delayed acceptance of its classification as a fungus. Each mammalian species is thought to have its own species of Pneumocystis.

Pathogenesis & Epidemiology

Transmission occurs by inhalation, and infection is predominantly in the lungs. The presence of cysts in the alveoli induces an inflammatory response consisting primarily of plasma cells, histiocytes, lymph resulting in a frothy exudate that blocks oxygen exchange. (The presence of plasma cells has led to the name "plasma cell pneumonia.") The organism does not invade the lung tissue.

*P. carinii* is distributed worldwide. It is estimated that 70% of people have been infected.

Life cycle:

Trophic forms, sporocytes and mature cysts are the three main morphological forms in the Pneumocystis life cycle. Trophic forms are the most abundant stage: these vegetative forms appear as mononuclear, 2-8 μm in diameter, with thin cell wall consisting of an electron-dense single layer. Ameboid in shape, trophic forms display cytoplasmic projections known as filopodia, which allow them to attach closely to type I pneumocytes.

Ultra-structural studies as well as the recent discovery suggest the existence of mating types and the probable conjugation of Pneumocystis trophic forms in. Following the fusion of mating types, a round, thin-walled, mononuclear and probably diploid early sporocyte is produced and shortly thereafter enters a meiotic division process. Meiosis is followed by an
additional mitotic replication resulting in eight nuclei in the late sporocyte stage.

An electron-lucent layer appears between the electron-dense layer and the plasma membrane of the intermediate sporocyte. It further thickens at the late sporocyte stage to finally produce a thick-walled mature cyst that measures 4-7 µm in diameter. Its surface is rather smooth, with few filopodia. In the mature cyst, eight individual spores are clearly delineated and are protected by a cell wall that appears as thick as the cell wall of the trophic form.

Clinical Findings

The sudden onset of fever, nonproductive cough, dyspnea, and tachypnea is typical of Pneumocystis pneumonia. In infants, the disease usually has a more gradual onset. The mortality rate of untreated Pneumocystis pneumonia approaches 100%.

Laboratory Diagnosis

Diagnosis is made by finding the typical cysts by microscopic examination of lung tissue or fluids obtained by bronchoscopy, bronchial lavage, or open lung
biopsy. Sputum is usually less suitable. The cysts can be visualized with methenamine-silver, Giemsa the cysts appear as a round masses of unstained cytoplasm containing 2-8 purple stained nuclei. Fluorescent-antibody staining is also commonly used for diagnosis. The organism stains poorly with Gram stain. There is no serologic test, and the organism has not been grown in culture. PCR-based tests are being developed.

**Treatment**

The treatment of choice is a combination of trimethoprim and sulfamethoxazole (Bactrim, Septra).
Sarcocystis

Causative agent of sarcocystosis

Sarcocystis hominis

Sarcocystis suihominis

Sarcocystis lindemani

Sarcocystis hominis and S. suihominis are known as human intestinal parasites. Infection results from ingestion of raw or insufficiently heated meat from cattle (Sarcocystis hominis) or pigs (Sarcocystis suihominis), which frequently contains muscle cysts of these species. The prevalence of intestinal sarcocystosis in humans is low and is only rarely associated with illness, except in volunteers who ingest large numbers of sarcocysts. Cases of infection of humans as intermediate hosts, with intramuscular cysts, number less than 100 and are of unknown origin.

Life cycle:

Man gets infection by eating raw or undercooked meat that contained sarcocyst, in the small intestine of definitive host (man) the brodyzoites are released and they migrate to sub epithelial lining and develop to male and female gametocyte. Fertilization occur to form thin-walled oocysts that sporulate in the intestinal wall. Once the frail oocyst wall has burst, free sporocysts containing four sporozoites each are excreted with stool in most cases. The sporozoites are infectious for intermediate hosts.

When the intermediate host get these sporocysts it infect the endothelial cells of blood vessels and schizogony cycle occurs. Those schizont rupture releases merozoites in the blood vessels and go to striated muscles and develop into tissue cyst (sarcocyst). In case of sarcocystis lindemani similar man can act as intermediate host and asexual cycle may occur in the striated muscle.
**Clinical manifestations:** Both species can cause short-lived (6 to 48 hours) symptoms within 24 hours of eating meat containing cysts, for example nausea, vomiting, and diarrhea as well as a mild fever. *S. suihominis* is more pathogenic than *S. hominis.*

**Diagnosis:** An intestinal infection with *Sarcocystis* can be diagnosed by detection of sporocysts (14 × 9 μm), or more rarely oocysts (approx. 20 × 13 μm) in stool using the SAFC or the flotation method. The two *Sarcocystis* species cannot be differentiated.

**Treatment:** An effective therapy has not yet been developed.

**Prevention:** consists in boiling or deep-freezing (−20 C for three days) of pork and beef.