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1-MINUTE CONSULT

Q: Are antibiotics indicated for the treatment of aspiration pneumonia?

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Antibiotics are indicated for primary bacterial aspiration pneumonia and secondary bacterial infection of aspiration (chemical) pneumonitis, but not for uncomplicated chemical pneumonitis.

THREE TYPES OF ASPIRATION PNEUMONIA*

Aspiration pneumonia is a broad and vague term usually used to refer to the pulmonary consequences of abnormal entry of oropharyngeal or endogastric contents into the lower airways. It can be classified as:

- Primary bacterial aspiration pneumonia
- Secondary bacterial infection of chemical pneumonitis.

These three are sometimes difficult to differentiate, as their signs and symptoms can overlap.

CHEMICAL PNEUMONITIS

Aspiration of stomach contents is relatively common, even in healthy preterm and usually has no clinical consequences.¹ However, it has also been clearly related to community-acquired and nosocomial pneumonia in some studies.²

Chemical pneumonitis is usually a consequence of the aspiration of a large volume (> 25 mL) of sterile acidic (pH < 2.5) gastric contents into the lower airways (Mendelson syndrome).³ The clinical picture varies from asymptomatic to signs of severe distress, hypoxia, cough, and low-grade fever; these signs and symptoms may develop rapidly, within minutes to hours after a witnessed or suspected episode of aspiration.^{4,5} However, they represent an inflammatory reaction to the gastric acid rather than a reaction to bacterial infection.^{6,7}

Chemical pneumonitis can be hard to differentiate from bacterial aspiration pneumonia

Chemical pneumonitis affects the most dependent regions of the lungs

Chest radiographic abnormalities in the most dependent regions of the lung, if aspiration occurs while the patient is supine, the posterior segments of the upper lobes and the apical segments of the lower lobes are most affected. The basal segments of the lower lobes are usually affected if aspiration occurs while the patient is standing or upright.^{8,9,10,11}

Clinical course varies

The clinical course varies. In almost 60% of cases, the patient's condition improves and the lung infiltrates resolve rapidly, within 2 to 4 days. On the other hand, in about 15% of cases, the patient's condition deteriorates quickly, within 24 to 36 hours, and progresses to hypoxic respiratory failure and acute respiratory distress syndrome.

In the other 25% of cases, the patient's condition may improve initially but then worsen as a secondary bacterial infection sets in. The death rate in these patients is almost three times higher than the rate in patients with uncomplicated chemical pneumonitis.^{12,13}

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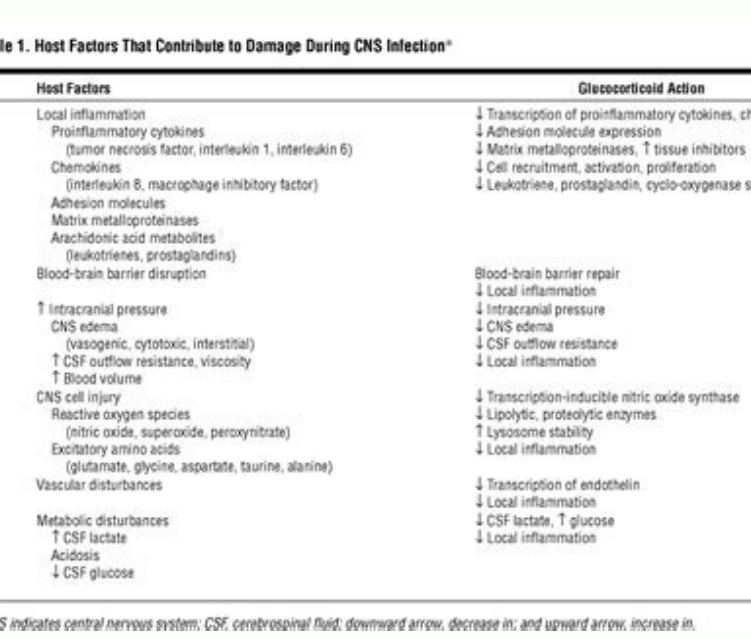
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Table 2. Clinical Features of Acute Mesenteric Ischemia

Cause	Incidence, %	Presentation	Risk Factors	Treatment
Arterial embolism	40-50	Acute catastrophe	Arrhythmia, myocardial infarction, rheumatic valve disease, endocarditis, cardiomyopathies, ventricular aneurysms, history of embolic events, recent angiography	Embolectomy, papaverine, excise infarction
Arterial thrombosis	25	Insidious onset with progression to constant pain	Atherosclerosis, prolonged hypotension, estrogen, hypercoagulability	Papaverine, thrombectomy, excise infarction, revascularization
Nonocclusive	20	Acute or subacute	Hypovolemia, hypotension, low cardiac output status, α-adrenergic agonists, digoxin, β-receptor blocking agents	Treat cause first, papaverine, excise dead bowel
Venous thrombosis	10	Subacute	Right-sided heart failure, previous deep vein thrombosis, hepatosplenomegaly, primary clotting disorder, malignancy, hepatitis, pancreatitis, recent abdominal surgery or infection, estrogen, polycythemia, sickle cell disease	Thrombectomy, excise dead bowel, heparinize, long-term complication

Table 1. Host Factors That Contribute to Damage During CNS Infection*

Host Factors	Disseminated Action
Local inflammation	↓ Transcription of proinflammatory cytokines, chemokines
Proinflammatory cytokines (tumor necrosis factor [interleukin 1, interleukin 6])	↓ Adhesion molecule expression
Chemokines (interleukin 8, macrophage inhibitory factor)	↓ Matrix metalloproteinases, ↑ Tissue inhibitors
Adhesion molecules	↓ Cell recruitment, activation, proliferation
Matrix metalloproteinases	↓ Leukostasis, prostaglandin, cyclo-oxygenase synthesis
Ascholic acid metabolites (folic acids, prostaglandins)	
Blood-brain barrier disruption	Blood-brain barrier repair
↑ Intracranial pressure	↓ Local inflammation
CNS edema (vasogenic, cytotoxic, interstitial)	↓ Intracranial pressure
↑ CSF outflow resistance, viscosity	↓ CSF outflow
Blood volume	↓ Local inflammation
CNS cell injury	↓ Transcription-inducible nitric oxide synthase
Reactive oxygen species (nitric oxide, superoxide, peroxynitrite)	↓ Lipolytic, proteolytic enzymes
Excitatory amino acids (glutamate, glycine, aspartate, taurine, acinate)	↑ Topoisomerase stability
Vascular disturbances	↓ Local inflammation
Metabolic disturbances	↓ Transcription of endothelin
↓ CSF outflow	↓ Local inflammation
↓ CSF outflow	↓ CSF outflow, ↑ glucose
↓ CSF outflow	↓ Local inflammation



Antibiotics pdf with references.

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