Meningeal infections generally originate in one of two ways: through the bloodstream as a consequence of other infections or by direct spread, such as might occur after a traumatic injury to the facial bones or secondary to invasive procedures.An acute fulminant infection occurs in about 10% of pa- tients with meningococcal meningitis, producing signs of over- whelming septicemia: an abrupt onset of high fever, extensive purpuric lesions (over the face and extremities), shock, and signs of disseminated intravascular coagulation (DIC).Bacter- ial or meningococcal meningitis also occurs as an oppor- tunistic infection in patients with acquired immunodefi- ciency syndrome (AIDS) and as a complication of Lyme disease (Chart 64-1).Acute fulminant presenta- tion may include adrenal damage, circulatory collapse, and widespread hemorrhages (Waterhouse-Friderichsen syn- drome).Once the causative organism enters the bloodstream, it crosses the blood-brain barrier and proliferates in the cere- brospinal fluid (CSF).The host immune response stimu- lates the release of cell wall fragments and lipopolysaccha- rides, facilitating inflammation of the subarachnoid and pia mater.CSF circulates through the subarach- noid space, where inflammatory cellular materials from the affected meningeal tissue enter and accumulate.