

Case Study Analysis



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Sickle cell disease represents a complex hereditary hemoglobinopathy that affects millions worldwide, with particularly high prevalence among individuals of African descent. This genetic disorder fundamentally alters red blood cell structure and function, leading to a cascade of pathophysiological processes that manifest as varied clinical symptoms. Marcus W., a 16-year-old African American male, presents with classic manifestations of sickle cell disease, including severe pain crises, chronic anemia, and organ involvement. Understanding his presentation's cellular mechanisms is crucial for Advanced Practice Registered Nurses (APRNs) to provide comprehensive care. This case analysis examines the pathophysiological processes, genetic foundations, immunological impacts, treatment mechanisms, and culturally sensitive educational approaches necessary for optimal patient outcomes in sickle cell disease management.

Cellular Pathophysiology and Symptom Correlation

The fundamental pathophysiological process in Marcus's condition involves deoxygenated sickle hemoglobin polymerizing, leading to rigid, crescent-shaped red blood cells that occlude microcirculation and cause tissue hypoxia (Sundd et al., 2019). This vaso-occlusive process directly correlates with Marcus's severe joint and chest pain, representing classic pain crises that occur when sickled cells block blood flow to bones and organs. The chronic hemolysis of fragile sickled cells explains his low hemoglobin level of 7.1 g/dL and elevated reticulocyte count, as his bone marrow attempts to compensate for ongoing red cell destruction (Darbari et al., 2020). Additionally, the breakdown of red blood cells releases hemoglobin, which is metabolized to bilirubin, accounting for his scleral icterus and elevated total bilirubin levels. In contrast, releasing intracellular contents explains his elevated lactate dehydrogenase levels.

Genetic Mutation and Inheritance Pattern