Cerebral fat embolism in sickle cell disease crisis

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A 32-year-old man with Sickle Cell Disease (SCD) Hemoglobin SS (HbSS) presented with painful crisis, developed acute chest syndrome, became unresponsive, and required mechanical ventilation. He had anemia, thrombocytopenia, and multiple nucleated red blood cells. A CT head was unremarkable. An MRI brain showed a “starfield” appearance on diffusion-weighted imaging (DWI) [1]. DWI sequence showed numerous punctate bilateral hyperintensities throughout the brain, with matching hypointensities on apparent diffusion coefficient (Figure A), consistent with microinfarcts. Susceptibility-weighted imaging (Figure B) showed numerous punctate bilateral hypointensities throughout the brain consistent with microhemorrhages. These findings are consistent with cerebral Fat Embolism (FE); likely originating from necrotic bone marrow during SCD crisis. Fat emboli are thought to enter the veins in the marrow and can cross to the arterial system via a patent foramen ovale or transpulmonary shunts. With supportive care, he improved and three months later returned to his baseline neurological function [2]. This illustrates the usefulness of MRI in identifying the cause of unresponsiveness in this patient with HbSS and acute chest syndrome.

Clinical Image

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References
