



Welcome to Neuroradiology

CASE # 6348701259

Titulo: Fat embolism

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Sections: [Brain](#)

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Patient: F, 18 year(s)

CLINICAL HISTORY

18 years old male with multiple bone fractures including head trauma

IMAGING FINDINGS

Examination demonstrates multiple punctate form diffusion signal abnormality lesions in bilateral cerebellar hemispheres, left worse than right bilateral cerebellar peduncles, midbrain, upper vermis as well as bilateral thalami, basal ganglia and hemispheric subcortical and cortical distributions in keeping with multiple embolic lesions. Susceptibility weighted images demonstrate punctate form scattered too numerous to count susceptibility signal loss lesions without blooming effects matching the distribution of the diffusion signal abnormality lesions. FLAIR and T2-weighted images demonstrate hyperintense behavior in the same distributions. T1-weighted images demonstrate few of the lesions demonstrating hyperintense behavior. Imaging findings of concern for diffuse fat embolism, which may correlate with tubular bone fractures with associated bone marrow fat embolism.

DISCUSSION

Since its first emergence in Zenker in 1862, fat embolism syndrome has been known to be associated with displaced

long bone fracture of the lower extremities and is characterized by respiratory disability, petechial skin rash, and neurologic symptoms, typically seen between 12 and 72 hours after the injury. The incidence of cerebral fat embolism (CFE) has been reported to be 0.9%–2.2%.

Neurological symptoms usually follow pulmonary manifestations and may present as altered levels of consciousness, seizures, focal neurologic deficits, and coma. Neurologic dysfunction in FES has been theorized to occur by several similar mechanisms including: (1) cerebral blood vessel occlusion by fat emboli, (2) disruption of the blood brain barrier due to toxic FFAs, and (3) obstruction due to alteration in the solubility of fat in blood secondary to fractures.

Depending on size, emboli may reach the brain by traversing the pulmonary microvasculature to reach the systemic circulation or through a right-to-left cardiac shunt such as a patent foramen ovale.

Typically, in FES, CT reveals no abnormalities even in patients with neurologic symptoms. Cerebral T1W images in FES may demonstrate either hypointense lesions or may be normal. Gadolinium enhancement on T1W images may also be useful to detect disruptions of the blood–brain barrier in the hyperacute state of cerebral FES. FLAIR and conventional T2W sequences typically reveal multiple diffuse foci of hyperintensity in the white matter of the subcortical, periventricular, and centrum semiovale regions. The changes seen on T2W images may require several days to develop and are associated with vasogenic edema. A characteristic starfield pattern of restricted diffusion may be seen in the centrum semiovale, with multiple hyperintense foci visible against a darker white matter background.

However, patchy and confluent-restricted diffusion may also be rarely seen with DWI.

FINAL DIAGNOSIS

Fat embolism

DIFFERENTIAL DIAGNOSIS LIST

diffuse axonal injury
cardiogenic cerebral emboli or septic cerebral emboli

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CITACION

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