CLINICAL PRACTICE

*Clinical Images***“If You Prick Us, Do We Not Bleed?”: An Uncommon Cause of Xanthochromia**

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A 50-year-old man presented with amoxicillin-induced cholestatic liver injury with resultant dialysis-dependent acute kidney injury. Total bilirubin ranged from 30 to 33 mg/dL, AST/ALT 200 to 400 U/L, and alkaline phosphatase from 3,000 to 4,000 U/L.

Because of fever and delirium, lumbar puncture (LP) was performed on hospital day 30 after normal head imaging. Moderate xanthochromia was immediately visualized after a

non-traumatic tap. Analysis revealed RBC 1/cu mm, WBC 1/cu mm, minimally elevated protein (50 mg/dL), and normal glucose (82 mg/dL).

Xanthochromia, “blond color” in Greek,¹ is caused by pigment in the cerebral spinal fluid (CSF). Xanthochromia is classically associated with subarachnoid hemorrhage (SAH): red blood cells lyse within hours and are metabolized from oxyhemoglobin (pink) to bilirubin (yellow). Xanthochromia is present in >90 % of patients with a SAH within 12 h of onset,^{1–3} but can also occur with increased CSF protein (≥ 150 mg/dL) due to bilirubin binding, traumatic LP with delayed analysis, and serum hyperbilirubinemia (>10 – 15 mg/dL), which was the most likely cause in this case.^{1,2,4} Literature suggests an association between CSF and serum bilirubin levels but not the duration of hyperbilirubinemia.⁵ Spectrophotometry can be used to identify bilirubin with or

Fig. 1 CSF vials after lumbar puncture, with the hallmark blond color of xanthochromia.

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without oxyhemoglobin.^{3,6,7} However, xanthochromia should continue to raise concern for SAH in appropriate clinical scenarios (Fig. 1).

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