Yellow Dialyzer: A Rare Manifestation of Profound Hyperbilirubinemia

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KEY WORDS: dialyzer, Dubin-Johnson syndrome (DJS), end-stage kidney disease (ESKD), hyperbilirubinemia, jaundice

IMAJ 2026; 28: 185–186

PATIENT DESCRIPTION

A 55-year-old male with a history of Dubin-Johnson syndrome (DJS), obesity, and smoking presented to the emergency department with generalized weakness and jaundice. On admission, he was hypotensive (blood pressure 87/56 mmHg), and profound jaundice was noted. Laboratory investigations revealed severe acute kidney injury with a creatinine level of 5.53 mg/dl and blood urea nitrogen of 92 mg/dl. Liver function tests were mildly elevated, and his lipid profile was within normal limits. Total bilirubin was markedly elevated at 52.5 mg/dl, predominantly direct (40.9 mg/dl). The patient was anuric at the time of catheter insertion.

A non-contrast abdominal computed tomography scan showed normal kidney size and appearance without hydronephrosis. The liver was normal size with sharp borders. The patient was treated with intravenous fluids, inotropic support, and intravenous antibiotics. Despite these interventions, he remained anuric with worsening hyperkalemia, necessitating urgent hemodialysis.

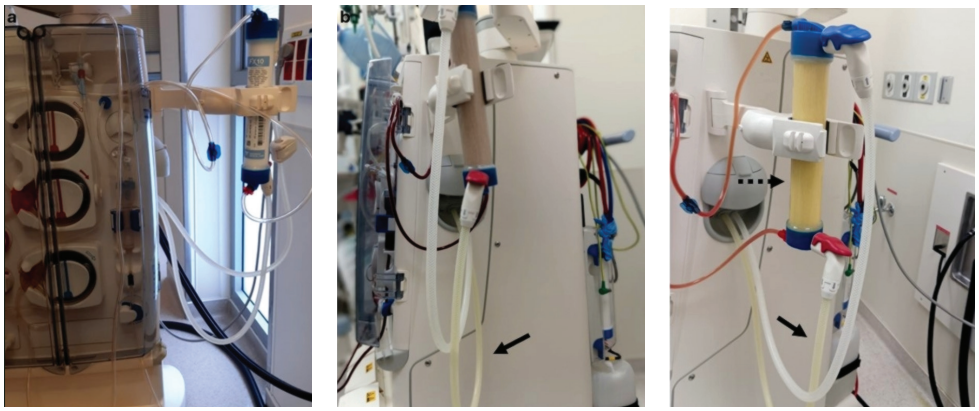
Within 10 minutes of initiating hemodialysis, a yellowish discoloration appeared in the effluent tubing of the dialysate. Simultaneously, the dialyzer fibers, which are typically pinkish in color, began to develop a yellowish tint. By the end of the session, the dialyzer appeared distinctly yellow, likely due to bilirubin deposition [Figure 1A–1C].

Figure 1. Yellow dialyzer

[A] Dialyzer fibers prior to connection, appearing white

[B] Yellowish discoloration of the effluent dialysate fluid is observed (black arrow) 10 minutes after initiating hemodialysis

[C] The dialyzer fibers are distinctly yellow (dotted arrow), and the effluent dialysate in the tubing appears yellow (black arrow) at the end of the hemodialysis session



COMMENT

Dialyzer discoloration is a rare phenomenon. Sporadic cases of yellowish discoloration of a dialyzer due to hyperbilirubinemia, particularly among patients on chronic hemodialysis, have been reported [1]. In these cases, hyperbilirubinemia was significantly milder, and the yellow dialyzer was the first clue that raised suspicion of hyperbilirubinemia. This finding is particularly relevant in patients with end-stage kidney disease, where jaundice may be more difficult to recognize due to uremic skin changes.

Another potential cause of dialyzer discoloration, such as pigments from diagnostic agents (fluorescein), has also been reported [2,3]. How-

ever, in our case, the discoloration was uniquely attributed to extreme hyperbilirubinemia, with a total bilirubin level of 52.5 mg/dl, one of the highest levels reported in the literature. In DJS, mutations in adenosine triphosphate binding cassette subfamily C member (ABCC2) abolish the canalicular export pump multidrug resistance protein 2 (MRP2), resulting in lifelong retention of conjugated bilirubin with typical serum levels of 2–5 mg/dl; values > 25 mg/dl are exceptional [4].

We hypothesize that the superimposed acute kidney injury, sepsis-related cholestasis, and systemic hypotension synergistically impaired hepatocellular handling of bilirubin, pushing levels to the unprecedented range observed. A comprehensive literature review revealed no previous reports of dialyzer or tubing discoloration in DJS, underscoring the novelty of this observation. Although the exact mechanism of dialyzer/tubing staining has not been fully clarified, one hypothesis is that unfiltered conjugated bilirubin/bilirubin-albumin complexes become entrapped within the polysulfone membrane. This presumption requires confirmation in future experimental studies.

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Capsule

Protecting the nervous system

Patients with cancer undergoing chemotherapy can present with chemotherapy-induced peripheral neuropathy (CIPN); however, predicting the risk of CIPN is understudied. **Chen** et al. integrated clinical data, whole-genome sequencing, and mouse studies to identify predisposing genetic alterations for CIPN susceptibility and therapeutic interventions. They first identified a polymorphism that confers vulnerability to CIPN. Next, they found two compounds that alleviate CIPN in mouse models. These findings offer a potential biomarker for patients who are susceptible to CIPN, providing a potential neuroprotective intervention that warrants further consideration.

Sci Transl Med 2026; 18 (837): eadx1436
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Capsule

A minimally invasive dried blood spot biomarker test for the detection of Alzheimer's disease pathology

The DROP-AD project investigated the potential of dried plasma spot (DPS) and dried blood spot (DBS) analysis, derived from capillary blood, for detecting AD biomarkers, including phosphorylated tau at amino acid 217 (p-tau217), glial fibrillary acidic protein, and neurofilament light. In total, 337 participants from seven centers were included, with 304 participants providing paired capillary DPS or DBS and venous plasma samples. **Huber** and colleagues observed strong correlations between DPS p-tau217 and venous plasma p-tau217 (r_S = 0.74, P < 0.001). DPS p-tau217 progressively increased with increasing disease severity and showed good accuracy in predicting CSF biomarker positivity (area under the curve = 0.864). Similarly, the authors demonstrated the successful detection of glial fibrillary acidic protein and neurofilament light with strong correlations between DBS and DPS, respectively, using paired venous plasma samples. Notably, the method was also effective in individuals with Down syndrome, a population at high genetic risk for AD but in whom standard blood sampling by venipuncture may be more complicated, revealing elevated biomarkers in those with dementia compared with asymptomatic individuals. The study also explored unsupervised blood collection, finding high concordance between supervised and self-collected samples. These findings underscore the potential of dried blood collection and capillary blood as a minimally invasive, scalable approach for AD biomarker testing in research settings.

Nature Medicine 2026; 32: 599
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