To the Editor: Acute-on-chronic liver failure (ACLF) is a syndrome in patients with chronic liver disease/cirrhosis, characterized by acute deterioration of liver function with one or more extra hepatic organ failures.\(^1\) ACLF may lead to an increased 28-day mortality rate, ranging from 23% to 74%.\(^2\) The common precipitant injuries that influence the course of ACLF include viruses, drugs, alcohol, infection/sepsis, gastrointestinal hemorrhage, ischemia, surgery, and idiopathic insults.\(^1\) There are few case reports on thyrotoxicosis triggered by iodinated contrast media (ICM) exposure that promotes ACLF. Here we report one such case.

A 30-year-old woman with a 2-year history of abnormal liver function but without symptom was admitted to our hospital due to persistent jaundice, abdominal distension, and intermittent mild fever for 1 month. Her serologic antibodies were negative, including hepatitis virus A, B, C, and E, Epstein-Barr virus, cytomegalovirus, and rubella virus. Antibodies of autoimmune diseases and alpha fetoprotein (AFP) were also negative. There was no alcohol abuse, hepatotoxic prescriptions, gastrointestinal hemorrhage, and surgery in the last few months. One week before admission, her abdominal contrast-enhanced computed tomography (CT) showed liver cirrhosis with moderate ascites. There were no space occupying lesions in the liver.

Physical examination at admission showed temperature 38.1°C, heart rate 98 beats per minute (bpm), and normal breathing and blood pressure. She was fully conscious. Her thyroid was non-tender and there was no thyromegaly. Breathing and blood pressure. She was fully conscious. Her thyroid was non-tender and there was no thyromegaly.

Due to apparent abnormal liver function, anti-thyroid drugs were contraindicated, and radioactive iodine therapy was considered. However, her iodine level in the 24-h thyroid uptake was only 4.1% (much lower than the 30% requirement of iodine level in the thyroid). Anti-copper treatment was also not effective. The only solution could be liver transplantation. Several adjuvant medications, including polynye phosphatidyl choline, ademetionine 1, 4-butanedisulfonate, diuretics, and infusion of albumin and plasma were used as a bridge therapy to liver transplantation. However, the patient had persistent mild fever and her liver function deteriorated [Figure 1]. There was no suitable liver donation for her during the short time. On day 16 after admission, she developed hepatic encephalopathy and progressed to coma in 2 days. She underwent plasma exchange, mechanical ventilation, and other supportive treatments in the intensive care unit. She was still very sick with persistently comatose condition, increasing serum bilirubin level and bleeding in gastrointestinal, respiratory and urinary tract consecutively. Then after admission, her liver function persistently worsened with increasing bilirubin and coagulopathy [Figure 1]. Low serum level of ceruloplasmin 0.108 g/L (normal range [NR]: 0.210–0.530 g/L) was found. Higher urinary copper 5.5 μmol/L (NR: 0–1.6 μmol/L) and normal blood copper 9.7 μmol/L (NR: 0–20.0 μmol/L) were reported. Kayser Fleischer ring on her cornea was detected by slit lamp. Positive gene detection of ATP-7B suggested Wilson disease (WD). New Wilson index score was 13, which meant high probability of death without liver transplantation.\(^3\) Moreover, thyroid test showed thyroid-stimulating hormone <0.005 mU/L (NR: 0.270–4.200 mU/L), free thyroxine 50.67 pmol/L (NR: 12.00–22.00 pmol/L), and thyrotrphin receptor antibody 4.52 IU/L (NR: <3.00 IU/L). Doppler ultrasound of the thyroid revealed a non-uniform density of thyroid with rich blood flow.

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her family members stopped her treatment. She died at home.

The Jod-Basedow phenomenon in patients with underlying thyroid diseases following the exposure of exogenous iodide has been described for a long time. Abdominal contrast-enhanced CT is one of the most common examinations for the patients with chronic liver disease. The development of thyrotoxicosis and ACLF in this case with WD and hyperthyroidism after the contrast-enhanced CT examination is a rare occurrence. Thyrotoxicosis might be induced by iodide exposure and aggravated liver damage. It was reported that thyrotoxicosis may cause mild liver function abnormality, even severe acute liver failure.\(^4\) The hepatic manifestations of WD are very variable, ranging from asymptomatic abnormal liver tests, simple acute self-limited hepatitis-like illness to autoimmune-like hepatitis, or even cirrhosis and rare acute liver failure.\(^5\) Managing the patients with both thyrotoxicosis and liver failure is challenging, because it is very difficult to administer anti-thyroid medicines, I-131 radioiodine ablation, or thyroidectomy. This case imparts a lesson that assessing thyroid function before contrast-enhanced CT examination for the patients with chronic liver disease may be necessary to avoid thyrotoxicosis, severe liver failure, and poor prognosis.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms in which the family members of this patient provided their consent for the clinical information to be reported in the journal. They understand that the patient’s name and initial will not be published and efforts will be made to conceal her identity, but anonymity cannot be guaranteed.

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**Conflicts of interest**

None.

**References**

1. Jalan R, Yurdaydin C, Bajaj JS, Acharya SK, Arroyo V, Lin HC, et al. Toward an improved definition of acute-on-chronic liver failure. Gastroenterology 2014;147:4–10. doi: 10.1053/j.gastro.2014.05.005.

2. Gustot T, Fernandez J, Garcia E, Morando F, Caraceni P, Alessandria C, et al. Clinical course of acute-on-chronic liver failure syndrome and effects on prognosis. Hepatology 2015;62:243–252. doi: 10.1002/hep.27849.

3. Dhawan A, Taylor RM, Cheeseman P, De Silva P, Katsiyiannakis L, Mieli-Vergani G. Wilson's disease in children: 37-year experience and revised King's score for liver transplantation. Liver Transpl 2005;11:441–448. doi: 10.1002/lt.20352.

4. Sousa Dominguez A. Severe acute liver failure and thyrotoxicosis: an unusual association. Rev Esp Enferm Dg 2015;107. doi: 10.17235/reed.2015.3607/2014.

5. Czlonkowska A, Latwin T, Dusek P, Ferenc P, Lutsenko S, Medici V, et al. Wilson disease. Nat Rev Dis Primers 2018;4:21. doi: 10.1038/s41572-018-0018-3.

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