Introduction: Liver dysfunction is associated with thyrotoxicosis itself and antithyroidal medications. Treating hyperthyroidism in these circumstances requires a deep consideration of the risks and benefits, which often proves challenging. Investigating for other underlying liver abnormalities is critical and may alter clinical course.

Clinical case: A 28-year-old female with a past medical history of hepatic steatosis and Graves’ disease with conflicting reports of methimazole use presented with progressively sharp abdominal pain and jaundice for one week. She had associated shortness of breath, heat intolerance, palpitations, lower extremity swelling, anorexia and weight gain for the preceding two months. On physical exam she was initially alert, oriented, afebrile, and tachycardic with irregular rhythm. She was markedly jaundice, had visible goiter, and +3 pitting edema in the lower extremities. Tests showed hemoglobin 7.3 g/dl (12-16 g/dL), hematocrit 23% (36-47%), cholestatic liver abnormalities AST 72 U/L (11-39 U/L), ALT 36 U/L (12-78 U/L), alkaline phosphatase 248 U/L (45-117 U/L), total bilirubin 25.4 mg/dl (0-1 mg/dl), direct bilirubin 22 mg/dl (0-0.3 mg/dl), albumin 2.3 g/dl (3.5-4.6 g/dl), and thyrotoxicosis with suppressed TSH <0.005 mIU/L (0.36-4.17 mIU/L), and elevated FT4 3.93 ng/dl (0.76-1.46 ng/dL). Serological antibodies were negative for viral causes of hepatitis. She quickly developed respiratory failure secondary to a large pleural effusion requiring mechanical ventilation complicated by acute renal failure. Plasmapheresis was performed normalizing her thyroid function tests. Subsequent testing showed elevated urinary copper 203.7 ug/dl (3-45 ug/d) and hepatic copper content of 331.8 ug/g (15-55 ug/g). Liver biopsy showed steatosis, cholestasis, stage 2 fibrosis. Her Leipzig score was 4 suggesting high probability of Wilson’s disease. She remained hemodynamically unstable requiring vasopressor support with poor right heart function. It was determined that she was not a candidate for liver transplant. She expired 3 weeks after hospitalization.

Conclusion: Abnormal liver function studies can be seen in thyrotoxicosis however, profound liver dysfunction is rare and complicates the decision to start antithyroid medications. Given this patient’s acute liver failure and unclear timing of the last dose of methimazole, plasmapheresis was a viable option and ultimately rendered correction of thyrotoxicosis, lowered Graves’ disease immunology markers, and improvement in liver function studies. Wilson’s disease is an autosomal recessive disorder which leads to copper accumulation in the liver and brain, liver transplantation is considered in the presence of acute liver failure. Due to this patient’s continued vasopressor dependence and multi-system organ failure, she was not a candidate for liver transplant and unfortunately died. This case underscores that plasmapheresis can serve as an effective treatment of thyrotoxicosis when first line medications cannot be used, or when rapid correction of thyrotoxicosis is essential.