

Incidental Finding of Multiple Enhancing Liver Lesions in the Setting of Mitochondrial Myopathy

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Case presentation:

A 28-year-old gastrostomy tube, tracheostomy, foley catheter-dependent female with a past medical history of mitochondrial myopathy and chronic neuromuscular respiratory failure presented with acute onset of shortness of breath and blood-tinged tracheal aspirate. On arrival, the patient was saturating at 60% on room air. Arterial blood gas analysis revealed acidosis and hypercapnia. The patient was stepped up to the ICU given increasing oxygen requirements. Her respiratory status improved after suctioning the tracheostomy tube and the patient was gradually transitioned to her home ventilation settings. Computed tomography of the chest with contrast ruled out a pulmonary embolism but did reveal bilateral lower lobe consolidations suggestive of pneumonia. Incidentally, multiple small enhancing lesions throughout the liver were also identified; the largest of which was 1.88 cm. Initially, liver function tests revealed an AST 63, ALT 48, and alkaline phosphatase 136, all of which normalized within a week of hospitalization. Abdominal ultrasound revealed a non-enlarged, homogeneous echotexture of the liver and a significantly dilated bladder. Urinalysis findings suggested a urinary tract infection (UTI) in the setting of urinary retention. Urine cultures grew *Providencia* UTI and blood cultures grew methicillin-resistant *Staphylococcus aureus* (MRSA). The patient was started on broad-spectrum empirical antimicrobial therapy with vancomycin, ceftriaxone, and azithromycin for ventilator-associated pneumonia (VAP), UTI, and MRSA bacteremia. Throughout this hospital stay, the patient also required vasopressor support in the ICU setting for worsening hypotension secondary to septic shock. The patient continued to improve clinically and steadily improved to her baseline prior to discharge with antibiotics.

Discussion:

Previous studies have estimated the abundance of mitochondria in the liver to be in the range of 13-20% of the liver volume. For this reason, there has been significant interest in elucidating the link between hereditary mitochondrial diseases and liver dysfunction. Some studies have estimated the prevalence of liver involvement in mitochondrial diseases in children to be as high as 20%. Neonatal liver failure is the most severe which has an acute onset and is characterized by lethargy, vomiting, and hypotonia. It is associated with significant neurological decline and results in death within weeks to months. On the other hand, Alpers's Disease starts in infancy and has a slow onset. It is characterized by "hepatomegaly, cholestasis, transaminitis, cirrhosis and progressive liver failure". However, incidentally found liver lesions in patients with hereditary mitochondrial myopathy have not been previously described. Small, enhancing liver lesions found incidentally on routine CT abdomen of patients without risk factors for liver disease can be a common finding and generally do not require follow up. The most common of these lesions include hepatic cysts and hemangiomas. Although it might be possible to distinguish between benign and malignant lesions via multiphase CT or MRI imaging, the routine, single venous portal phasic CT may not be as informative. This presents a challenge when attempting to differentiate between benign lesions, HCC and metastasis. Studies have shown that the size of the lesions is an important diagnostic clue and may guide further work-up. These studies highlight that enhancing lesions smaller than 1.5 cm are likely benign in low-risk patients and those larger than 1.5 cm may require further imaging such as MRI or multi-phase CT. Another important consideration is the presence or absence of cirrhosis. In the presence of cirrhosis, the clinical suspicion for HCC rises significantly. If the diagnosis remains uncertain, it is appropriate to proceed with fine needle biopsy, follow up imaging, or resection for lesions larger than 5 cm.

