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Severe Methimazole-Induced Hepatotoxicity Complicated by Renal Failure and Sepsis

Dr Julie Anne Gabat[1]
[1]*Philippine General Hospital, Manila, Philippines*

INTRODUCTION
Methimazole is associated with fewer incidences of adverse reactions compared to Propylthiouracil, therefore it is recommended as the drug of choice for hyperthyroidism. However, severe and fatal methimazole-induced liver injury does occur, particularly, in the presence of an underlying liver disease.

CASE REPORT
A 54-year old female with Graves' Disease was supposedly for radioactive iodine ablation when she developed panophthalmitis. Atrial fibrillation was noted on electrocardiogram with markedly elevated FT4 and suppressed TSH levels. Methimazole was resumed at 40mg per day. Orbital CT scan showed no evidence of thyroid ophthalmopathy. Echocardiography showed no valvular abnormalities. Panophthalmitis and thyrotoxicosis eventually resolved. Two weeks later, she developed fever, epigastric pain, vomiting, thrombocytopenia and leukopenia, hence, methimazole was stopped. Five days after stopping methimazole, she developed encephalopathy, jaundice, coagulopathy and renal failure for which she underwent hemodialysis. Holoabdominal ultrasound showed normal liver with normal bile ducts and gallbladder, normal renal sizes with signs of parenchymal disease and poor corticomedullary differentiation. She tested negative for hepatitis infection. She developed hospital acquired pneumonia from a multi-drug resistant organism (Acinetobacter baumanii) and eventually succumbed to septic shock. Post-mortem examination of the liver revealed canalicular cholestasis, microvesicular steatosis and portal triaditis consistent with drug-induced liver injury. No evidence of vasculitis or allergic interstitial nephritis was noted.

CONCLUSION
Methimazole can cause severe hepatotoxicity even in the absence of an underlying liver disease and it can occur even after stopping the medication. Factors other than direct liver injury may be responsible for this latency that needs further investigation.