PP100 UNUSUAL STROKE MIMIC: SUSPECTED METHANOL POISONING IN A COVID-19 PATIENT

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Introduction: Methanol poisoning from fraudulent adulterated alcoholic beverages is an important differential in a patient with stroke-like symptoms. This presentation is overlooked frequently by physicians leading to inadequate or delayed therapeutic interventions. Neurological sequale are inevitable usually mortality documented to suprass 40%.

description: Case Α 64-year old. with gentleman underlying diabetes mellitus, hypertension, and chronic alcoholism presented with slurring of speech, blurring of vision, unilateral limb weakness, altered mentation, and cough. History revealed, he had consumed three bottles of whiskey that was locally brewed prior to presentation. Upon arrival, his vitals were blood pressure 121/81 mmHg, heart rate 140 beats/min, and temperature of 37 C. His GCS was E4,V3,M5 with significant limb weakness over the left side of his body. A computed tomography (CT) of the brain showed hypodense lesions over both lentiform nuclei, and a well-defined hypodensity at the right cerebellar peduncle. A diagnosis of stroke was made. His blood however, revealed significant tests. metabolic acidosis with a high anion gap (HAGMA). His observed osmolality was 386 mmol/kg, but his calculated serum osmolality was 294 mmol/kg which

indicated a high osmolal gap. COVID-19 RT-PCR was also positive. The diagnosis was revised as methanol toxicity. The patient was isolated and mechanically ventilated. Haemodialysis commenced, and 20% oral ethanol was administered via a naso-gastric tube. His acidosis improved and responded well to treatment. The neurological sequele is yet to be determined.

Discussion: Altered mental state, blurred vision, HAGMA, and a high osmolal gap are all classic symptoms of methanol poisoning. Stroke-like presentations are reported infrequently. The CT and MR findings of bilateral putaminal necrosis with or without haemorrhage, as well as subcortical white matter lesions, are unique, and a high index of suspicion for methanol should be considered. Several mechanisms have been proposed including vascular drainage pattern of the lenticular nucleus, direct toxic effects, and relative ischemia caused by the high metabolic rates of basal ganglia and optic nerves.

Conclusion: Even before the HAGMA emerges, a poor correlation of clinical and CT findings, notably infarct or haemorrhage of bilateral lentiform nuclei, should raise suspicion of additional differentials in stroke mimics such as methanol poisoning.

Keywords: methanol poisoning, ethanol, stroke, alcohol