

## METHANOL POISONING DIAGNOSED BY COMPUTERISED TOMOGRAPHY SCAN

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### METHANOL POISONING DIAGNOSED BY CT SCAN

The death rate is high in untreated methanol intoxication and early diagnosis is of fundamental importance for effective treatment. The characteristic computerised tomography (CT) scan findings may be helpful in the diagnosis of methanol poisoning. Bilateral necrosis of the putamen is the most well known sequela of methanol poisoning that can be identified by CT.<sup>1</sup> We report a case of a patient, with methanol intoxication, who initially presented diagnostic difficulties and in whom the use of CT scan was important in making the diagnosis.

### CASE REPORT

A 42-year-old hypertensive male patient was admitted to hospital with a history of headache, dimness of vision and gradual deterioration of consciousness. There was one episode of generalised seizure but there was no history of fever, drug overdose or head injury. His wife denied any alcohol abuse and there was no other significant history. Personal and family history was non-contributory.

On examination, he was comatose (Glasgow Coma Scale 6/15), with mild icterus and a blood pressure of 210/140 mm Hg, the pupils were constricted and reacting poorly to light; fundoscopy revealed slight blurring of the optic discs. There was no neck rigidity and plantar responses were bilaterally flexor. Abdominal examination revealed firm hepatomegaly (span 14 cms). There was no splenomegaly or ascites, and respiratory, cardiovascular and genital system examination was unremarkable.

Investigations revealed normal haematology and renal biochemistry. Blood glucose was normal. Liver biochemistry revealed a bilirubin of 55  $\mu\text{mol/L}$  with AST of 54 U/L and ALT of 42 U/L. Serological markers for hepatitis A, B and E were absent. An arterial ammonia level was normal. There was a severe metabolic acidosis with pH of 7.0 and increased anion gap. A chest X-ray showed mild cardiomegaly and an electrocardiogram revealed lateral wall ischaemia.

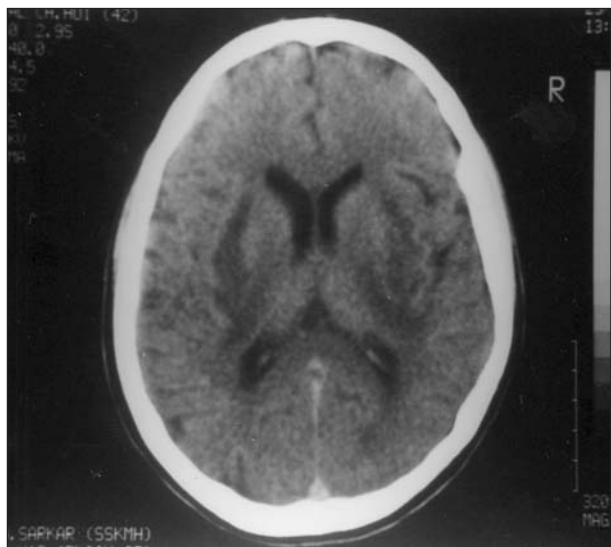
The initial diagnoses of hepatic encephalopathy and/or hypertensive encephalopathy were made and the patient was being managed accordingly. At this point a CT scan did reveal areas of hypodensity symmetrically localised especially in the region of the putamens (Figures 1 and 2). In light of the CT scan findings, a blood methanol

level was carried out and it was found to be 26 mg/dl. The patient was put on haemodialysis.

Three days later the patient regained consciousness. Clinical examination showed impaired vision, with dilated and fixed pupils; the optic discs were pale and oedematous, suggesting necrosis of the papillary part of the optic nerves. The patient improved gradually, and four weeks later was alert, oriented and without gross memory defects. He had, however, a marked visual deficit (visual acuity of 3/60 in right eye and only hand movements could be perceived by the left eye) and minimal weakness in the limbs. He now admitted to drinking denatured spirit the day before he had been taken ill.

### DISCUSSION

Methanol intoxication, a rare and potentially lethal form of poisoning, usually results from ingestion, and occasionally inhalation, of methanol. The clinical presentation of methanol poisoning varies greatly from patient to patient. Onset is variable and may be delayed. Early manifestations are caused by methanol itself, and late features are due to the metabolite of this alcohol, formic acid. Initially, nausea, vomiting, abdominal pain, headache, vertigo and an ethanol-like intoxication occur. Late manifestations include stupor, coma and seizures. Ophthalmologic manifestations occur 15–19 hours after ingestion and include clouding and diminished vision, dancing and flashing spots, dilated or fixed pupils, hyperaemia of the disc, retinal oedema and blindness.<sup>2</sup> Initial symptoms of blurred vision, an increased anion gap and metabolic acidosis are typically delayed and may not at first be recognised as a methanol-related complaint.<sup>3</sup> Once diagnosed, treatment must be prompt and definitive. The principal therapeutic procedures include titrated correction of acidosis with sodium bicarbonate, administration of ethanol, folic acid, 4-methylpyrazole, and secondary detoxification with haemodialysis. The therapeutic measures must be started quickly to improve the prognosis of methanol intoxication and to decrease the frequency of serious late complications such as ophthalmologic and neurological lesions. The most characteristic pathological findings after methanol intoxication are discrete regions of necrosis involving the putamen, in which varying degrees of haemorrhage have occurred. These characteristic changes can be seen if the patient survives for longer than 24 hours. Discrete regions of necrosis have also been described in the cerebral white matter of patients surviving longer



**FIGURE 1**  
Unenhanced head CT scan.



**FIGURE 2**  
Contrast enhanced head CT scan.

than several days. These changes are due to the metabolites of methanol, hypoxemia, severe acidosis, and coexistent circulatory depression. The bilateral putaminal lesions as well as separate lesions within the cerebral white matter can be identified with CT or magnetic resonance imaging.

The case presented is unusual in terms of the early onset of cerebral lesions demonstrated by CT scan. Other reports have described findings on a CT scan obtained within the first three days. The present case also underscores the fact that the clinical outcome does not correlate with the severity and extent of necrosis of the lentiform nucleus.<sup>4</sup> The findings in the cerebral white matter and putaminal lesions can also occur in Wilson's disease<sup>5</sup> or in Leigh disease.<sup>6</sup>

In the appropriate clinical setting, however, methanol poisoning should be suspected when these findings are seen on a CT scan. Thus in certain situations, as in the current case, the characteristic CT findings may be helpful in the diagnosis of methanol poisoning.

## REFERENCES

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