

Short report

Severe chorea after acute carbon monoxide poisoning

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SUMMARY Ten days after an acute exposure to carbon monoxide, a 33-year-old woman exhibited severe chorea. CT scan revealed bilateral lucencies of the pallidum and anterior arm of the internal capsule. Chorea was successfully treated by chlorpromazine and did not relapse after treatment withdrawal. The mechanism of chorea in acute carbon monoxide poisoning is discussed.

Chorea is not a classical feature of carbon monoxide poisoning. In his 1968 review of carbon monoxide poisoning, Jellinger¹ was able to collect only eight references describing choreiform movements or choreoathetosis. Of 2360 acutely intoxicated patients reported by Choi,² 65 exhibited delayed neurologic sequelae but chorea was not observed. To our knowledge, the pathological lesion corresponding to these abnormal movements in carbon monoxide poisoning is not known.

Case report

A 33-year-old woman was referred to our neurological department for treatment of abnormal movements. She had been previously in good health, without psychiatric illness and she was not taking any medication. In August 1983, she was found unconscious after exposure to a combustion heater in a closed bathroom in Morocco. Her child was found dead. She was taken to a nearby hospital and awoke some hours after discovery. On the second hospital day, she complained of imbalance and dysarthria but was discharged. The fifth day after carbon monoxide exposure, she was referred to another hospital with intention tremor and ataxia but she was not admitted. Abnormal movements appeared ten days after poisoning.

On admission, 12 days after carbon monoxide exposure, temperature was 36.8°C, blood pressure 150/90 mm Hg, respiration 24 per minute. The patient was fully conscious. She was severely dysarthric and swallowing was impaired. General examination revealed no abnormalities. Neurological examination revealed a normal mental status. Abnormal involuntary movements were extremely violent. They were brisk, arrhythmic and of great amplitude. They involved the trunk, the neck, the four limbs and spared the

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face. They consisted of flexion-extension of the distal muscles of the legs and of the forearms. The proximal muscles of the legs were involved in a rotational movement. The trunk was arrhythmically pushed forward and the head was moving from side to side. Attitude fixing, stress and movement induced bursts of choreic movements. More distally, the hands were involved by choreoathetoid movements. The patient could not stand up or walk. There was diffuse hypotonia and normal strength of all muscles. Coordination testing was disturbed by abnormal movements. Deep tendon reflexes were mildly exaggerated on the left side but there was no Babinski sign. The sensory system was normal. Vision, eye movements, pupils reaction to light appeared normal.

The results of routine admission blood studies were normal. A polygraphic surface electromyogram recorded the activity of the right biceps and triceps, extensor and flexor muscles of the right wrist, right sternomastoid and left splenius, right tibialis anterior and soleus muscles. It showed a spontaneous arrhythmic activity (fig 1) made of short bursts 1 to 5 s duration and 100 to 600 mV amplitude. Occasionally, a simultaneous burst occurred in two antagonistic muscles. Tonic activity was observed in the wrist extensors and in the tibialis anterior. The frequency and amplitude of the arrhythmic movements were exaggerated by mental arithmetic or sensory stimulation.

CT scan without contrast material (fig 2) showed bilateral areas of lucency close to pallidum and anterior limb of the internal capsules. The patient was given chlorpromazine 150 mg daily and maintained two days on supportive care with nasogastric intubation. She improved dramatically over the next few days and was able to stand, walk and take fluids by mouth on the fifth day after admission. Abnormal movements were limited to rare bursts of the trunkal musculature and athetoid movements of the hands. The speech was very slow and dysarthric. Neurological examination demonstrated cerebellar dysmetria on the left side. Chlorpromazine was progressively reduced and stopped two months after admission.

The follow-up visit 3 months after initial exposure to carbon monoxide showed no personality change and neuro-

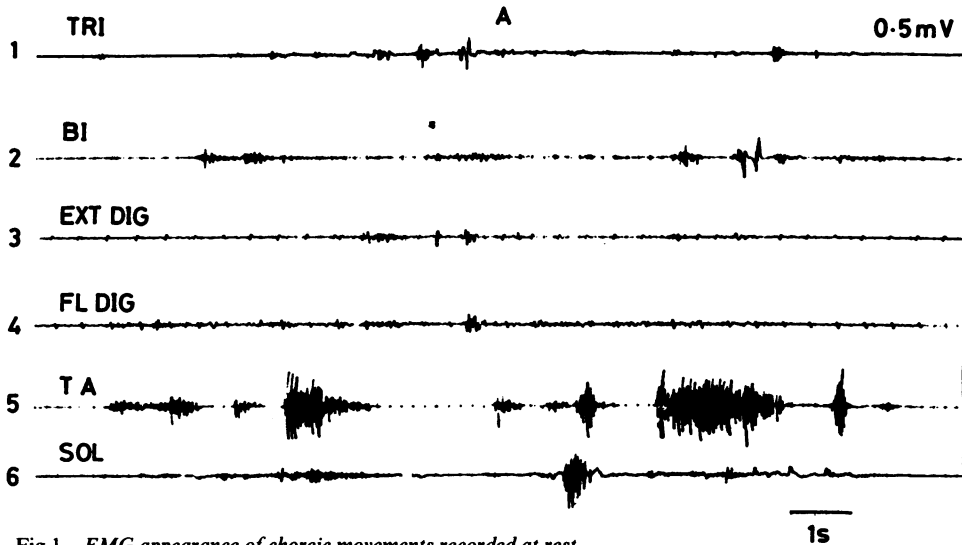


Fig 1 EMG appearance of choreic movements recorded at rest

psychologic assessment of cognitive functions was within normal limits. The patient had a slow dysarthric speech but she could sing with melody. Choreoathetotic movements were very rare and only initiated by emotions or attitude. Except for mild incoordination and hypotonia on the left side, the neurological examination was normal.

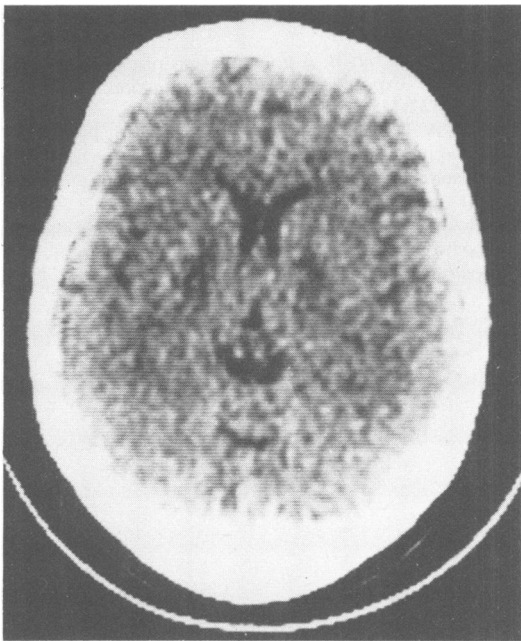


Fig 2 Unenhanced computed tomographic scan two weeks after acute carbon monoxide intoxication demonstrating lucencies in both basal ganglia.

Discussion

The anatomical basis for abnormal movements in carbon monoxide poisoning is not known. In a recent paper, Schwartz *et al*³ reported a case of choreoathetosis with infarction of the neostriatum on CT scan, suggesting a relationship between this structure and the abnormal movements. Nevertheless, a similar lesion was observed on the right side by Delouvrier *et al*⁴ without chorea. Low density areas seen on CT scan of our patient did not involve the caudate or the putamen nuclei. To our knowledge, the only report of chorea following carbon monoxide poisoning which was anatomically verified was by Merguet.⁵ There was no remarkable involvement of the basal ganglia but some glial proliferation and granular cells in these structures. In addition, patients affected by chorea of vascular origin were rarely demonstrated to have significant lesions in their basal ganglia.^{6,7} Conversely, the most frequent lesion of the basal ganglia observed after carbon monoxide poisoning, the necrosis of both pallida, is not followed by chorea but by hypertonic features, Parkinsonian state or dystonia.^{1,8-14}

Thus, it is likely that chorea observed in our case was not related to the pallidal lucencies observed on CT scan but was attributable to some lesion in the vicinity of the internal pallidum, either the subthalamic region or the adjacent internal capsule. Of the rare cases of chorea following carbon monoxide poisoning, some of them were transient,^{3,8,15,16} others had a chronic course.^{5,17} In our case, the fact that chorea did not relapse after neuroleptic arrest could suggest a functional rather than an anatomical impairment. The pathophysiological pattern of the

choreic syndrome was probably excessive dopaminergic output on the receptors of the striatum since chorea was alleviated by neuroleptics. A tentative explanation of the abnormal movements could be related to neuronal plasticity, as suggested by hyperactivity and delayed spine development of caudate neurons observed after carbon monoxide exposure in the rat.^{18,19} The chorea observed in our case could be the result of deafferentation of the striatum and increase output until compensatory inhibition of abnormal activity. Such an interpretation could explain the latency of such sequellae and spontaneous improvement. Whether this could be related to the delay in appearance of CT changes and to their disappearance, as suggested previously,^{20,21} is still debatable.

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