Short report

Brainstem ischaemia presenting as naloxone-reversible coma followed by downward gaze paralysis

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SUMMARY A 65-year-old man showed naloxone-reversible unconsciousness followed by downward gaze paralysis. CT scan suggested an ischaemic lesion in the mesodiencephalic region. This observation represents the first case of naloxone-reversible coma related to brainstem ischaemia.

Relief of unconsciousness by naloxone administration is considered a good therapeutic test for opiate intoxication. However, coma resulting from other causes such as encephalomyelitis1 and ethanol intoxication2 also has been reported to be relieved by naloxone. More recently the same therapeutic response has been observed in two cases of ischaemic lesions of cerebral hemispheres.3,4 In this paper, we report a case of brainstem ischaemia presenting as naloxone-reversible unconsciousness followed by down-gaze paralysis, a rare neuro-opthalmologic defect.

Case report

A 65-year-old Vietnamese was discovered in the early morning unconscious in bed. He had bilateral pin-point miosis, respiratory depression and no sign of obvious focal lesion. Narcotic overdose was suspected and naloxone 0.4 mg iv injection was followed within a few minutes by recovery of consciousness and adequate spontaneous ventilation. About 20 minutes later, the patient became unconscious again. A definitive recovery of consciousness was obtained through repeated 0.4 mg naloxone iv injection together with 0.4 mg im injection. Pupil size was then 2 mm each. The patient denied any drug intake and no opiate derivate could be detected in urine. Previous medical history was unremarkable except for a left otitis media with drum perforation.

Neurologic examination performed when the patient was conscious revealed him to be oriented in time and place with normal speech in Vietnamese and French.

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in this case by the clinical history, by the CT scan images and their evolution and by the arterial lesion. Improvement of ischaemic deficit by naloxone has been demonstrated in clinical and experimental studies previously.\(^1\)\(^\text{4-7}\) All the data concern hemispheric lesions in the territory of the internal carotid artery. In our case, the lesion is located in the territory of the vertebral basilar system. This is supported by the CT scan images and by the neuroophthalmologic deficit pointing to a lesion in the territory of the posterior thalamothalamic artery.\(^5\)

As to the effect of naloxone, perhaps local opioid release was induced by the ischaemic lesion. There is a particular abundance of opioid in the region involved.\(^8\)\(^-\)\(^11\) Further studies are necessary to understand the effect of naloxone on coma with an ischaemic cause and to determine if this effect is related to the opioid content of the region involved.

Discussion

Selective paralysis of downgaze has only been rarely described. Pierrot-Deseilligny \textit{et al}\(^7\) published the eighth and ninth clinicopathological cases recently. In our observations oculographic recordings demonstrated abolition of downward gaze for foveal pursuit, voluntary and visually guided saccades and preservation of the oculocephalic reflex. CT localised the lesion bilaterally and medially at the mesodiencphalic level. These findings are consistent with the hypothesis that isolated abolition of downward gaze is related to lesion of the efferent tracts of the rostral interstitial nuclei of the medial longitudinal fasciculus.\(^5\) Miosis, respiratory depression, lack of focal neurologic deficit, and unconsciousness improved by intravenous injection of naloxone are the main features of opiate intoxication. In this paper, we describe another cause for such a clinical presentation: ischaemia of the brainstem. The ischaemic nature of the lesion is suggested by the clinical presentation, by the CT scan images and their evolution and by the arterial lesion.

References


