Hemichorea—a late sequel of an extradural haematoma

R. SANDYK
M.D.

Department of Medicine, Johannesburg Hospital, Johannesburg 2000, South Africa

Summary

Hemichorea developed several weeks after evacuation of a traumatic extradural haematoma. The abnormal movements were confined to arm and leg on the side opposite the surgery. No other causes of chorea were found. The connection between the extradural haematoma and the late onset hemichorea is not well understood; mechanical pressure on basal ganglia structures, vascular disturbances of the anterior choroidal artery as well as biochemical alterations are all possible mechanisms.

KEY WORDS: extradural haematoma, basal ganglia, hemichorea.

Introduction

Chorea has been reported to occur in association with a variety of metabolic as well as structural brain lesions (Greenhouse, 1966; Bean and Ladisch, 1977; Martin, 1968). Extradural haematoma, however, has not been previously reported to cause chorea. I report an adult patient with hemichorea which developed several weeks after removal of an extradural haematoma.

Case report

A 28-year-old man suffered right-sided head trauma following a motor vehicle accident. In the emergency room, he was found to be unconscious; the right pupil was 6mm and the left 4mm in diameter. Both pupils reacted to light. There was no papilloedema. There was hemiparesis involving the entire left side with an extensor plantar response. The blood pressure was 90/60 mmHg.

Carotid angiography revealed posterior and upward displacement of the proximal portion of the anterior cerebral artery. The anterior-posterior view showed medial displacement of the sylvian point and shift of the anterior cerebral artery to the contralateral side. Computed tomographic (CT) scan of the brain confirmed the presence of a right subfrontal-extradural haematoma, after removal of which the patient made an uneventful recovery.

Approximately 6 weeks later, the patient was readmitted for assessment of left-sided abnormal movements. There was no history of drug intake and no family history of note. Examination revealed distinct choreoathetoid movements of the left arm and foot associated with occasional twitching of the left face. The movements were less apparent during sleep and increased in severity during emotional stress. There was no evidence of hemiballistic movements or focal neurological deficit.

Laboratory analysis, including blood count, serum biochemical estimations, glucose, thyroid studies, antinuclear antibody tests, lupus erythematosus preparation, serum manganese levels, caeruloplasmin level, syphilis serology and cholesterol were normal or negative. CT scan revealed no structural abnormalities and there was no evidence for a previous vascular accident. The electroencephalogram (EEG) was normal except for occasional bursts of theta activity over the right frontotemporal region. Lumbar puncture cerebrospinal fluid was normal.

The patient was treated with haloperidol and the choreoathetoid movements decreased considerably in both frequency and intensity.

Discussion

Martin (1968) reported a leukaemic child who developed severe choreic movements of the right arm and leg secondary to a left-sided subdural haematoma. The movements ceased promptly after evacuation of the haematoma. In the present case, a cause-and-effect relationship was not as clearly established because removal of the extradural haematoma preceded the chorea by several weeks.

Chorea is poorly understood. Attempts to localize the area of neurological involvement have been based on pathology, animal experiments and surgical procedures to alleviate the movement. Experimentally, only lesions in the subthalamic nucleus consistently produce chorea (Martin, 1968). In humans, lesions have been found in diverse areas of the basal ganglia, and many investigators believe that chorea

does not occur with a single lesion, but only with a combination of lesions (McDowell and Lee, 1977; Klintworth, 1965).

The exact aetiology of the hemichorea in the present patient is not understood. Mechanical pressure on basal ganglia nuclei could be caused directly by the extradural haematoma or indirectly by torsion or displacement of brain structures and herniation through the tentorial notch. Transient circulatory disturbances of basal ganglia structures could be caused by displacement and compression of the anterior choroidal artery (Siegfried, 1968). Finally, mechanical pressure could have decreased the number of dopaminergic and gabaergic receptors in the striatum and subthalamic nucleus (Yebeles *et al.*, 1982).

Whatever the precise mechanism may be, this report reveals that hemichorea may result as a late sequel of a removed extradural haematoma.

References

- BEAN, S. & LADISCH, S. (1977) Chorea associated with a subdural hematoma. *Journal of Pediatrics*, **90**, 255.
- GREENHOUSE, A. (1966) On chorea, lupus erythematosus and cerebral arteritis. *Archives of Internal Medicine*, **117**, 389.
- KLINTWORTH, G.K. (1965) The pathogenesis of secondary brain-stem hemorrhages as studied in an experimental model. *American Journal of Pathology*, **47**, 525.
- MARTIN, J.P. (1968) Choreatic syndrome. In: *Handbook of Neurology Vol. 6* (Eds. P.J. Vinken and G.W. Bruyn), p. 435. North Holland Publishing Company, Amsterdam.
- MCDOWELL, F.H. & LEE, J.E. (1977) Extrapyrarnidal diseases. In: *Clinical Neurology Vol. 2* (Eds. A.B. Baker and L.H. Baker), p. 10. Harper & Row Publishers, Inc., Hagerstown, MD.
- SIEGFRIED, L. (1968) *Die Parkinsonsche Krankheit und ihre Behandlung*. Springer, Vienna.
- YEBENES, J.G., GERVAS, J.J., MENA, M.A., RIO & R.M., SOMOZA, E. (1982) Biochemical findings in a case of parkinsonism secondary to brain tumor. *Annals of Neurology*, **11**, 313.

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