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Giant middle cerebral aneurysm presenting as hemiathetosis in a child and its spontaneous thrombosis

A. Rashid Choudhury¹, Nizar H. Al Amiri¹, Khalaf R. Al Moutaery¹, Mohamed Aabed², and M. Keith Strelling³

¹ Department of Neurosurgery, ² Department of Neuroradiology, and ³ Department of Paediatrics, Riyadh Armed Forces Hospital, P.O. Box 7897, Riyadh 11159, Kingdom of Saudi Arabia

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Abstract. A 10-year-old girl presented with a 6-week history of gradually increasing, abnormal movements and weakness of the right upper and lower limbs. There were no features of raised intracranial pressure. Computed tomography scan and magnetic resonance imaging (MRI) of the brain showed the features of a partially thrombosed giant middle cerebral artery aneurysm, located deep in the left lentiform region and compressing the basal ganglia and the mesencephalon. The angiogram confirmed the aneurysm and its origin from the main trunk of the artery with occlusion of all the branches. A direct approach was unsuitable for the treatment of the aneurysm, so an embolization procedure to occlude the neck of the aneurysm was considered. During the waiting period, the patient improved and became asymptomatic. Follow-up MRI showed complete thrombosis of the aneurysm and eventually, reduction in its size and mass effect. The hemiathetosis may have been the result of direct pressure on the basal ganglia by the aneurysm. The spontaneous intra-aneurysmal thrombosis may have been due to the massive size of the aneurysm and its narrow neck.

Key words: Giant aneurysm – Middle cerebral artery – Spontaneous thrombosis – Hemiathetosis

Aneurysms larger than 2.5 cm in diameter are considered to be giant aneurysms and are rare, comprising about 5% of all intracranial aneurysms [5, 10]. Although aneurysms are rare in childhood, there seems to be a slightly higher proportion of giant aneurysms in this age group [8]. They usually present either with subarachnoid hemorrhage or progressive visual loss and headache due to their frequent proximity to the carotid and ophthalmic arteries [4, 5, 8, 9]. Rarely, they may present as a space-occupying lesion and produce a contralateral weakness or sensory disturbance [17]. We report here a child with a giant mid-

dle cerebral aneurysm who presented with hemiathetosis thrombosed spontaneously.

Case report

This 10-year-old girl was admitted to the Riyadh Armed Forces Hospital with a 6-week history of abnormal movement and weakness of the right limbs. This was associated with slowness of speech. These features were gradual in onset and slowly progressive. There was no history of headache, paresthesia, visual disturbance, or fits. She had increasing difficulty in writing and this led to her failing the school examination. Her past medical history had been uneventful and there was no family history of abnormal movement.

On 22 February 1989, examination revealed a healthy young girl of average build. She was fully conscious and cooperative. When she attempted a voluntary action, her right fingers went into a slow, purposeless, continuous writhing movement. This was followed successively by flexion of the wrist, inward movement of the forearm and arm toward her chest, and adduction with outward movement of the whole limb. This was associated with elevation of the inner border of the right foot with extension of the hallux, abduction at the knee and flexion at the hip. These movements disappeared at rest and during sleep. Neurological examination revealed a right-sided upper motor-neuron-type facial palsy and a mild spastic weakness (power: UK Medical Research Council) grade 4 of the right upper and lower limbs. Her reflexes were brisk on the right side with a right plantar extensor response. Her speech was slow but clear. The rest of the neurological examination, including the fundi, was normal. The general physical examination revealed no abnormalities.

The results of the routine hematological and radiological investigations were normal. A computed tomography (CT) scan of the brain showed a hyperdense mass lesion containing specks of peripheral calcification in the left temporal lentiform region measuring 4.5 × 3 cm and a mass effect with compression of the basal ganglia and mesencephalon and a shift of the midline. After injection of a contrast medium, the lesion showed well-marked homogeneous enhancement. Magnetic resonance imaging (MRI) showed a non-homogeneous mass consisting of a larger area void of signal located centrally and a smaller area peripherally with high signal intensity. This mass was deep in the left lentiform region (Fig. 1), indicating a giant aneurysm with peripheral thrombosis. It measured 4.5 × 3 cm, and produced a mass effect with compression of the basal ganglia and mesencephalon, and a shift of the midline. A left carotid angiogram (Fig. 2) confirmed a giant aneurysm arising from the main trunk of the middle cerebral artery, with occlusion of all its branches. It was partially thrombosed peripherally.

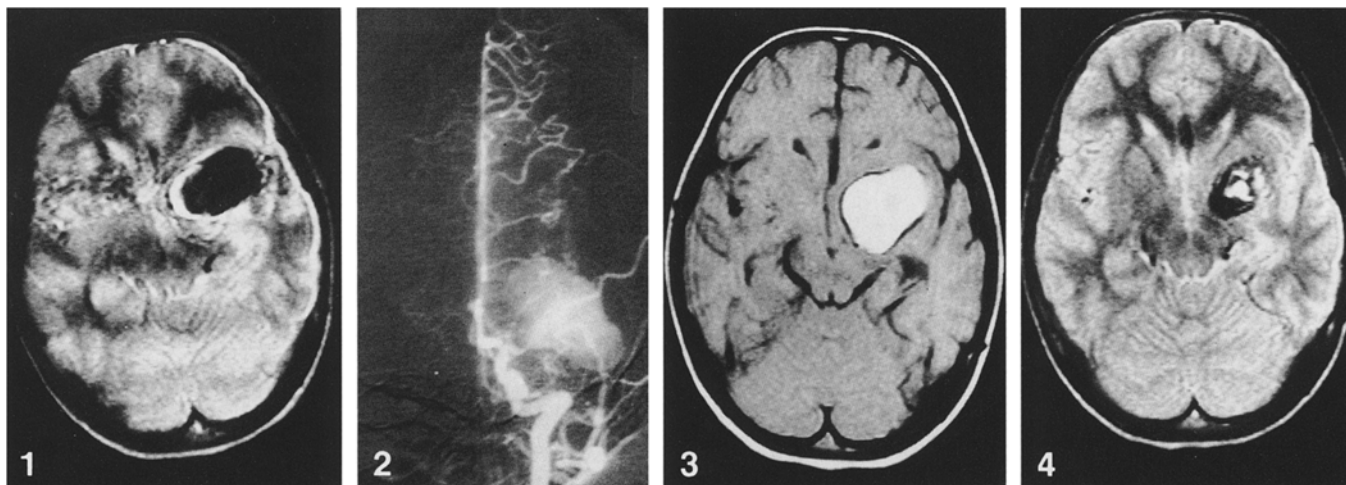


Fig. 1. Initial axial magnetic resonance image (MRI) (spin echo-T2-2100/100) showing a mass lesion in the left lentiform area with void signal centrally and hyperintense signal peripherally, and having a mass effect

Fig. 2. Left carotid angiogram. A-P view showing a partially thrombosed giant aneurysm arising from trunk of the middle cerebral artery with absence of its branches

Fig. 3. Axial MRI (SE-T1-800/30) after 2 months, showing the whole mass lesion with hyperintense signal, indicating complete thrombosis of the aneurysm

Fig. 4. Axial MRI (SE-T2-2100/100) after 7 months, showing organization of the thrombus and reduction in size of both the thrombosed aneurysm and the mass effect

A direct approach was unsuitable for the treatment of the aneurysm. This was due to the nonfeasibility of external carotid-internal carotid bypass because of occlusion of all the branches of the middle cerebral artery. An embolization procedure was considered in order to occlude the neck of the aneurysm. During a 2-month waiting period, the condition of the patient improved. The weakness resolved, abnormal movements subsided and her speech returned to normal. At this stage, follow-up MRI (Fig. 3) showed complete thrombosis of the aneurysm; however, the mass effect remained unchanged. MRI (Fig. 4) repeated 7 months later showed reduction of the size of both the thrombosed aneurysm and the mass effect. The patient remained well and asymptomatic.

Discussion

An extrapyramidal movement disorder has been reported as a rare complication of intracranial space-occupying lesions [1, 2, 6, 7, 14, 15]. In each case, the symptoms resolved after removal of the lesion. Thus a causal relationship between the two has been thought to exist [12]. In our patient, the assumption of a causal relationship between the hemiathetosis and the middle cerebral aneurysm is based on gradual resolution of the symptoms after spontaneous thrombosis of the aneurysm.

Giant aneurysms have a pleomorphic pattern of clinical presentation [3]. Only 1 of 22 patients in Bull's series [3] presented with a tremor in one arm with associated hemiparesis but no other extrapyramidal features. The presentation was either headaches or visual disturbances in 21 patients. In other series [4, 5, 8–10, 17], no extrapyramidal syndrome has been described. Recently, Gross [7] described a patient with a giant middle cerebral artery aneurysm presenting as hemiparkinsonism. Following surgery, the patient made an excellent recovery with complete resolution of the extrapyramidal features. In our patient, the aneurysm led to hemiathetosis and mild pyra-

midal signs on the right side. These might be the result of capsular involvement, together with the involvement of the basal ganglia on that side, leading to a combined pyramidal and extrapyramidal syndrome. The fact that resolution of the symptoms followed aneurysmal thrombosis suggests the release of capsular and basal ganglionic compression. This was proved by follow-up MRI.

Spontaneous intra-aneurysmal thrombosis is a well-documented phenomenon that has been noted in about 50% of giant intracranial aneurysms [3, 5, 9, 13]. This high incidence of thrombus formation within giant intracranial aneurysms is related to a critical ratio between the size of the aneurysm and that of its neck, below which intra-aneurysmal thrombosis occurs [18]. This may be the result of eddies forming within the aneurysm. In our patient, the spontaneous thrombosis reflects the natural course of a giant aneurysm that has not been complicated by subarachnoid hemorrhage. When such a lesion regresses, release of mass effect and resolution of symptoms occur. It is, however, advisable to observe the patient and to do a follow-up at regular intervals with MRI. Such a patient may be treated without surgery [16].

MRI has greatly facilitated the diagnosis of giant intracranial aneurysms [11]. It can clearly demonstrate the presence or absence of intra-aneurysmal thrombosis and the progression or regression of the aneurysm. In our patient, follow-up MRI showed complete thrombosis of the aneurysm, and subsequent imaging showed the organization of the thrombus with a reduction in the size of the aneurysm and a reduction of the mass effect. MRI is therefore the most helpful tool in the nonsurgical management of such thrombosed giant aneurysms.

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