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Could Parkinsonism complicate craniotomy and excision of convexity meningioma? A case report.

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Summary

Parkinsonism is a neurodegenerative clinical disorder characterized by varying combinations and degrees of rest tremor, rigidity, slowing of voluntary movements and postural instability. Amongst the aetiologic factors, massive post-craniotomy haematoma has not been previously considered. This report is to arouse the suspicion that vascular displacement in the midbrain resulting from a massive unilateral intracranial haematoma could lead to the unusual complication of Parkinsonism. This is the report of a 55-year old retired male banker, without any previously identifiable risk factor except for his age, who developed Parkinsonism following a huge acute extradural haematoma that complicated a craniotomy for the excision of a haemorrhagic convexity meningioma. The patient was placed on medical treatment resulting in a slow but progressive neurological improvement. Reports of Parkinsonism following post-tumour excision are probably, very rare. We, hereby, report a case of Parkinsonism complicating a gross total excision of a convexity meningioma.

Keywords: *Convexity meningioma, craniotomy, extradural haematoma Parkinsonism*

Résumé

Le parkinsonisme est une syndrome clinique de la dégénération nerveuse avec plusieurs facteurs étiologiques rapportés, mais l'hématome extra axiale post opérative n'est pas inclut. Nous faisons la craniotomie et l'excision complète de la convexité du méningiome hémorragique chez un homme de 55 ans sans aucune idée de s facteurs du parkinsonisme à l'exception de l'age. Il développait des symptômes extensive d'hématome extradurale sur la couche de la tumeur 10 heures après l'opération, nécessitant une exploration de la blessure et l'évacuation du caillot. Il a eu une guérison apparemment simple mais lente après l'opération et a été déchargée pour la maison. Cependant dans la sixième semaine, il développait progressivement des caractéristiques de parkinsonisme ipsilaterale du coté de la craniotomie. Une tomographie informatisée répétée a cette période révélait une infraction lacunaire du coté droit du mid-encephale absent dans tous les autres films. Notre opinion est que le large hématome post-opératoire résultait probablement d'un déplacement rostro-caudale de l'encéphale et une interruption des artères centrale terminales avec une infraction ischémique de la partie affectée de la mid-encephale causant le parkinsonisme.

Introduction

Parkinsonism is characterized by rigidity (but not spasticity), bradykinesia and impaired postural reflexes [1, 2, 3].

Worldwide incidence is not certain but a recent study by Bower, *et al*, in Minnesota, USA found a local annual incidence ranging from 0.8/100,000 person-years (among 0 – 29 year age group) to 304.8/100,000 person-years (80 – 99 year age group), and an average annual incidence of 114.7/100,000 person-years (50 – 99 year age group) [4].

The most common type is Parkinson's disease (Idiopathic paralysis agitans) – the most widely investigated and reported – which occurs mostly in the >50-year olds, with a male:female ratio of 3:2 [5]. Other types include post-encephalitic, arteriosclerotic (vascular), drug-induced, post-infective, toxic, anoxic, post-traumatic and Alzheimer's.

The substantia nigra is the most commonly affected organ and reduction in the activity of the neurotransmitter, dopamine, in the basal ganglia is the fundamental disorder in all forms of the disease, leading to a relative over-activity of the excitatory receptor-dependent systems, the imbalance influencing other pathways in the brain via the thalamus and thalamic connections. This results in

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an increased inhibition of the regulatory gamma motor neuron, and increased activity of the stimulatory alpha motor neuron.

The mainstay of therapy is medical treatment with levo-dopa – considered as the gold standard – an attempt at replenishing the depleted nigro-striatal dopamine stock [6]. Combinations of multiple antioxidants and catechol-o-methyltransferase (COMT) inhibitors, which seem to have levo-dopa sparing effects, have variously been used as adjunct therapy [7, 8]. Due to the long term deficits and side effects associated with the use of levo-dopa, there has been a resurgence of alternative modalities viz: intracerebroventricular injections of glial-derived neurotropic factor, and functional neurosurgery (postero-ventral medial pallidotomy – introduced in 1992 by Laitinen). The foetal mesencephalic tissue transplantation is still under experimentation [9, 10, 11].

There are reports of traumatic parkinsonism resulting from closed head injury, including cases associated with chronic subdural haematomata [12], but the most popular of the traumatic type is among pugilists thought to be 'punch-drunk' from repeated trauma to the head, termed *dementia pugilistica*. This form of the disease, however, appears to respond poorly to levo-dopa. We are currently unaware of any previous report of Parkinsonism following craniotomy for convexity tumour excision, in our environment.

We hereby report, a case of Parkinsonism complicating gross total excision of a convexity meningioma in a 55-year old previously healthy man with no identifiable risk factors, except probably for his age.

Case Report

A 55-year old, right handed, retired male banker, was referred to our service in April 2005 by a physician, with complaints of progressive amnesia for recent events (8months), painless left upper extremity weakness (3months) and focal left hemi-corporeal extremity tonic-clonic seizures (1day). The earlier symptoms were of insidious onset and progressive, presenting finally with inability to move the left half of the body. There were associated throbbing headaches and slurring of speech, but no vomiting, irrational behaviour or sphincteric dysfunction. The history was otherwise unremarkable and he was not on any neurotropic medications.

On clinical evaluation, the patient's general condition was satisfactory except for a mild systemic hypertension previously undiagnosed. He was fully conscious and alert with equal, briskly reactive (3mm) pupils bilaterally, but had a left supranuclear facioparesis and bilateral abducens paresis, and expressive aphasia. His memory, orientation, calculation and insight, were intact.

There was generalized muscle wasting, generalized hypertonia, left dense hemiparesis, absent plantar response bilaterally, absent abdominal reflex, left sided tendon hyperreflexia, and left hypoaesthesia.

There were no sphincteric or cerebellar deficits, and the meninges and spines were clinically normal. All the other organ systems were grossly normal. A provisional diagnosis of right hemispheric deficits due to a supratentorial mass lesion, was made.

Computerized tomography of the brain (Figure 1) showed a hyperdense right-sided parieto-temporal tumour with brilliant contrast enhancement, obstructive hydrocephalus and prominent tumour vasculature.

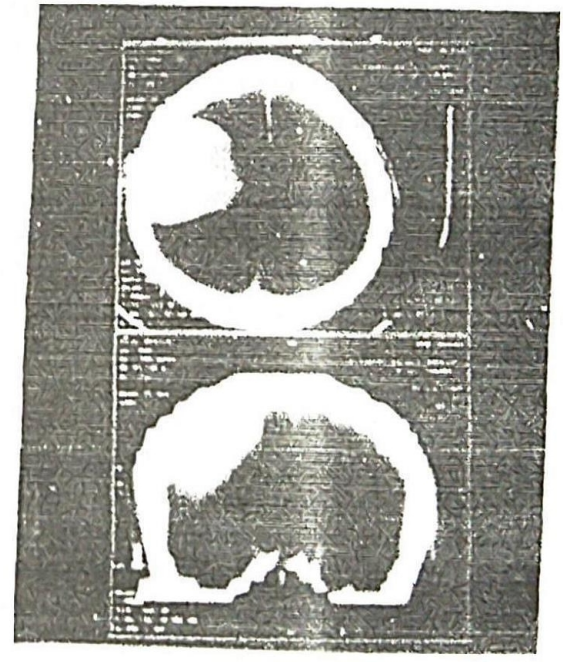

Fig. 1: Cranial CT (axial and coronal slices) of the patient showing the huge hyperdense contrast enhancing right fronto-patietal meningioma, pre-operatively

A definitive diagnosis of a parieto-temporal convexity meningioma was finally made.

The patient had a full pre-operative work-up, and subsequently, craniotomy and gross total tumour excision, under endotracheal general anaesthesia.

The operative findings included markedly dilated bridging and diploeic vessels, highly vascular mixed consistency tumour with extensive dural invasion and a well defined tumour – brain interface.

Ten hours post-operatively, the patient developed left focal seizures with secondary generalization, and associated progressive decline in sensorium from a Glasgow Coma Score of 14/15 to a score of 10/15.

An emergency cranial computerized tomography (Figure 2) revealed gross total tumour excision but with huge acute subgaleal and extradural haematomata over the tumour bed, and midline shift of 1cm to the left, with effacement of the ipsilateral lateral ventricle.

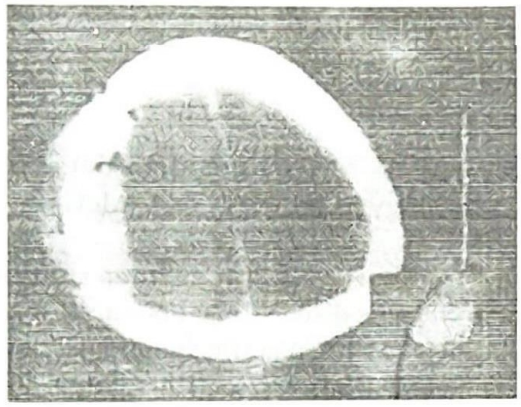

Fig. 2: Immediate post-operative cranial CT (native) showing a complicating acute extraduaral haematoma in the previous tumour bed, with significant midline shift to the left. The lesion thought to have displaced the arterial supply to the substantia nigra, causing the features of parkinsonism.

He was returned to the theatre and clot evacuation with wound review was urgently done under general anaesthesia, yielding 160ml of altered blood clots.

He made a slow post-operative neurological recovery, but had a lingering expressive aphasia, emotional lability and left hemiparesis, at the time of discharge from the hospital, one month after the craniotomy. The histology report confirmed a meningioma.

At the out-patient clinic, one month after discharge, superimposed on the resolving aphasia, emotional lability and left hemiparesis were additional deficits – festinant gait, right-sided cog wheel rigidity, right-sided tremors at rest with pill-rolling movements and bradykinesia, all of which were noticed at home in the sixth post-operative week.

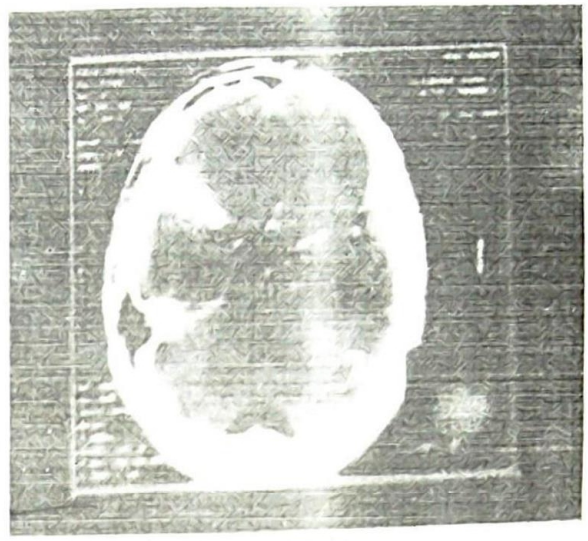

Fig. 3: Repeat post-operative cranial CT, one month after, showing a prominent right mesencephalic lacuna which was absent in a similar slice in the immediate post-operative period (Fig. 4).

A repeat cranial CT (Figure 3) showed a right mesencephalic lacuna in the tegmentum involving the dorsal margin of the substantia nigra, ipsilateral to the craniotomy site. The immediate post-operative images (Figure 4) did not show a similar lesion.

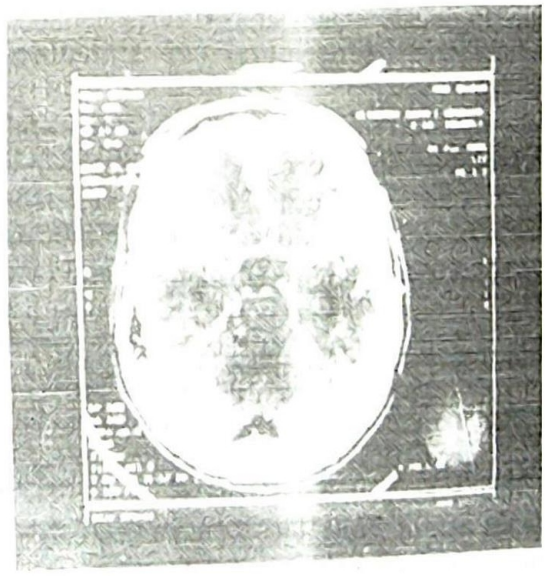

Fig. 4: Cranial CT slice at the same level as Fig. 3, but in the immediate post-operative period, which showed an intact midbrain without a lacuna.

A diagnosis of Parkinsonism was made, and the patient was referred to our neurologists. He was placed on levo-dopa, and made some clinical improvement with improved affect, reduction of tremors and rigidity, and lessened aphasia, in the first six months of therapy.

Discussion

Previously unreported in our environment, parkinsonism following a complicated craniotomy, with a lacunar infarct in the mid-brain could be pathophysiologically related to the post-head injury variety, and to some extent, anoxic or vascular parkinsonism, since the events leading up to the disease entity very readily compares in both conditions – ischaemia. It has been projected that resultant infarction leading to a loss of about 80% of the dopaminergic cells in the nigrostriatal pathway (connecting substantia nigra and corpus striatum), would be sufficient to cause parkinsonism [13], irrespective of the pathogenesis. Our patient's clinical features were classic, and the post-operative emotional lability should have been the first warning sign since this is a typical symptom of vascular parkinsonism, usually associated with gait disturbance and pseudobulbar deficits [3].

The prominent right-sided mesencephalic lacuna in Figure 3, one-month post-operative image, on the same side as the parkinsonian features and ipsilateral to the craniotomy massive clot site, and which was not present in the same location in both the pre-operative (Figure 1) and immediate post-operative (Figure 4) images supports the pathogenesis of a peri-operative vascular event leading to nigrostriatal deficits.

The blood supply to the basal ganglia takes origin from the middle cerebral (medial and lateral branches), recurrent branch of the anterior cerebral, anterior choroidal and posterior communicating arteries [14]. The mid-brain is supplied by the posterior cerebral and superior cerebellar arteries with the medial and lateral central branches entering the crura to supply the substantia nigra and red nucleus [13]. Pontine haemorrhage following displacement of central vessels during rostro-caudal herniation is well known. Our view is that the massive post-operative acute clot could have led to a similar, though uncommon, vascular displacement with necrosis in the territory of such an end vessel. This could explain the lacunar finding only in the last radiological study. This severe problem underscores the importance of painstaking intra-operative haemostasis and the increased operative risks in giant tumours. This possibility seems also to suggest the need for a prospective study of brain stem lesions in patients who die from massive acute unilateral clots.

We, therefore, wish to pose the question – whether parkinsonism could result from a craniotomy for the excision of a giant convexity meningioma complicated by post-operative intracranial haematoma.

Conclusion

We report this unusual case of post-craniotomy parkinsonism with corresponding new mesencephalic lesion, to document an unusual possible complication, seek for possible explanation, and highlight the need for further studies of the brain stem even in patients who die from various forms of acute unilateral clots, to evaluate any possibilities of such unsuspected lesions as we found in our patient.

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