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Case report

Subarachnoid hemorrhage and extrapyramidal symptoms: a case report

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Summary. *Introduction:* Parkinsonism may occur after brain lesions such as subarachnoid hemorrhage (1), hydrocephalus (2,3), slit ventricle (4), or shunt revision (5).Until now, pathogenesis remains unclear. *Method:* Case Report. *Results:* We described the case of a 53 years Caucasian male with subarachnoid hemorrhage after anterior communicating artery aneurysm rupture, with subsequent complications and early development of bilateral extrapiramidal symptoms. A DatSCAN showed an impairment of the nigro-sytriatal dopaminergic way. Levodopa therapy induced complete symptoms remission. *Conclusions:* Patient developed Parkinson Disease responding to Levodopa. Subarachnoid hemorrhage itself, shunt placement and revision, hydrocephalus, slit ventricle: all of these complications occurred and could be possible causes of shear, torsion, and ischemia of the nigrostriatal projection fibres. (www.actabiomedica.it)

Key words: extrapyramidal symtoms, subarachnoid hemorrhage, Parkinson disease, hydrocephalus

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Acute extrapyramidal symptoms may develop after brain damage as subarachnoid hemorrhage and hydrocephalus (1,2). We report a patient showing hydrocephalus, shunt failure and slit ventricle after subarachnoid hemorrhage with subsequent developing of nigro-striatal involvement and Parkinson Disease (PD).

A previously healthy male, aged 53 was admitted to the Emergency Unit with headache and vomit. Cerebral Tomography Computer Scann (CT scan) showed subarachnoid haemorrhage due to an anterior communicating artery aneurysm treated with electrocoagulation and coiling insertion. After an immediate neurological recovery, patient became increasingly sleepy and drowsy in the following days. Patient's condition worsened until a vegetative state: on the 10th day an angiography showed vasospasm involving bilateral anterior, posterior and middle cerebral arteries which improved after Nimodipine infusion. Patient improved significantly but suddenly he became lethargic in the following 3 days. CT scan showed hydrocephalus and a ventriculoperitoneal shunt was inserted with subsequent reduction of ventricular enlargement at the control CT scan.

Three weeks after the cerebral haemorrhage the patient was admitted to the Rehabilitation Unit with a partial deficit of left eye medial rectus muscle; minimal strength deficit of the right limbs, minimal rigidity on the left side and marked ataxia. A few days after insomnia and anxiety a diffuse tremor appeared. EEG was within normal limits. Clinical conditions worsened with increasing inertia, bradykinesia, rigidity, urine incontinence and complete dysphagia. CT scan showed marked reduction of the right ventricle ("slit ventricle"), with left lateral ventricle only slightly diminished suggesting monolateral overdrainage. Patient was then moved to the Neurosurgery Unit where

MRI showed substantial re-expansion of the right lateral ventricle. Empirical therapy with Levodopa was started as the shunt valve was adapted to reduce drainage fluctuations. Patient gradually improved and after one week he returned to Rehabilitation Unit showing only moderate rigidity and minimal postural tremor. Complete neurological and functional recovery was reached in the following couple of weeks .

A DatSCAN (54 days after haemorrhage) showed bilateral moderate dopamine transporter radioligand hypocaptation in the caudate nuclei and bilateral severe putamen hypocaptation. Qualitative and quantitative analysis showed symmetrical impairment of the nigro-sytriatal dopaminergic way. These data confirmed the hypothesis of Parkinson's Disease. Levodopa was progressively replaced with Ropirinol. Two months after subarachnoid haemorrhage patient returned home without any extrapyramidal sign or anxiety.

Different conditions as hydrocephalus and shunt failure can cause extrapyramidal symptoms as a consequence of increasing pressure on the basal ganglia or midbrain. Akiyama et al. (2) reported symptoms after acute shunt obstruction. Etiology however remains unclear: increased intracranial pressure could interfere with the white matter fibers surrounding ventricles. Sudden reduction of excessive intracranial pressure could cause dopaminergic pathway malfunctioning as well, with secondary parkinsonism inducing "acute dopaminergic deficiency syndrome" by acute change in ventricular size (2).

Hydrocephalus could induce hypoperfusion of the striatum, resulting in reversible dopamine deficiency and suggesting possible reversible pre-synaptic nigrostriatal dysfunction by ischemic compression (3).

This mechanism has been implicated in a reduction of the blood flow in basal ganglia producing a reduction of the vascularization at the level of the pallidal branches of anterior choroidal artery to the globus pallidus, and branches of lenticulostriate artery to the head of caudate and putamen. Anterior choroidal artery compression against the rostral tentorium may occur in cerebral hemisphere swelling. Basal ganglia may be vulnerable to compression and hypoperfusion as they are supplied by non-anastomothing branches and arterioles. Striatum cells are also thought to be particularly susceptible to ischemia due to their high metabolic rate. Ling et al. documented midbrain and basal ganglia injury resulting in ischemic neurologic deficits after vasospasm. Thus, other mechanism could be involved in basal ganglia dysfunction as Wallerian degeneration associated with tracts originating in the basal forebrain. Authors suggest also a possible metabolic etiology: in case of poor response to levodopa, the damage could rely, instead of in the substantia nigra, in the basal ganglia rather than in substantia nigra to which it projects (4).

In our case, a sudden reduction of the right ventricular size occurred after hydrocephalus. "Slit ventricle syndrome" is a possible complication of ventricular shunt described in literature mostly when Cerebrospinal Fluid (CSF) overdrainage causes sudden intracranial hypotension and ventricular walls coaptation (2).

Our patient experienced marked anxiety while developing extrapyramidal symptoms as a possible dopamine depletion occurring in different regions of the striatum. In particular, anxiety in Parkinson may be mediated by destruction of dopaminergic caudate-frontal circuits. All of the aforementioned events could have caused or contributed to symptoms development. Ventricular pressure on upper midbrain and diencephalon may cause shear, torsion, and ischemia of the nigrostriatal projection fibres. Responsiveness to levodopa may be related to the reversible damage to the presynaptic nigrostriatal dopaminergic fibers caused by the pressure fluctuations induced by hydrocephalus or slit ventricle on the nearby midbrain rather than dysfunction of the basal ganglia and its frontal projection fibers (3).

Racette et al. (5) compared the DatSCAN of one patient with post-hydrocephalus parkinsonism with same exam applied to 12 patients with idiopathic PD. Author detected a similar dopamine cells loss in the caudate between the two groups while dopamine cells loss in putamen would be more charachteristic of Idiopathic PD, with posterior more damaged. In our case, DatSCAN quantitative analysis showed slight hypocaptation of the radioligand in the caudate nuclei bilaterally (typical of both idiopathic PD and post-hydrocephalus parkinsonism) and symmetrical severe hypocaptation in the putamen, more typical of idiopathic PD pattern of damage.

In conclusion patients with shunt for hydrocephalus, should be carefully monitored to guarantee an accurate valve pressure regulation and early levodopa administration should be considered, when extrapyramidal symptoms appear and parkinsonism is suspected.

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