ABSTRACT- The authors report the case of a 50 year-old hypertensive male patient with a pontine hematoma. The clinical presentation was characterized by pure pyramidal deficit signs (no other signs or symptoms were present). A pure hemiplegia syndrome, although common in supratentorial lesions, is considered to be a rare event in pontine vascular lesions. The pathophysiologic mechanisms of these neurological findings are unclear. The exclusive involvement of the pyramidal tract in this case is likely due to a variation in the vascular anatomy of the pons but, in some cases, a vascular malformation may be the cause.

KEY WORDS: pontine hemorrhage, pyramidal syndrome, pure hemiplegia.

Several clinical syndromes have been reported to exist in accordance to the extension and specific sites of involvement of brainstem hematoma. Different clinical manifestations can result either from destruction of structures or from the compressive effects exerted by the hematoma upon brainstem nuclei and fibers. Elevated blood pressure has been emphasized repeatedly as an etiologic factor in the development of pontine hemorrhage. It is also possible that vascular malformations could produce pontine hematoma and that the hemorrhage itself could obliterate the typical histologic organization. Teilmann, in his extensive review of the literature, referred to a few case reports in which the apoplectic onset of a hemipontine syndrome was associated with survival for a prolonged period of time, subsequent pathologic examination revealed findings consistent with vascular malformation in some of his cases. This could be expected to occur in only a small minority of cases.

Almost all primary pontine hemorrhages (PPH) occur in hypertensive persons, most of them having no prior symptoms of cerebral vascular disease although in some series nearly 50% of patients had a clinical history of previous hemiplectic strokes. Among various types of intracerebral bleeding, primary pontine hemorrhage is known to have the worst prognosis. Following a sudden onset, there

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is often coma with tetraplegia, decerebrate posturing, respiratory disturbance, hyperthermia, and pin-point pupils. Mortality rate due to pontine hemorrhage is reported to range from 30 to 68.7%\textsuperscript{12,16,23}. Tanaka et al\textsuperscript{22} classified pontine hemorrhage into [1] massive type, [2] tegmentum basis type, and [3] tegmentum type. It was noted that the prognosis for long-term survival and functional recovery for tegmentum type hemorrhage cases is good, the massive type being devastating. The prognosis for basis tegmentum type hemorrhage stands between the other two.

The authors report on a case of pontine hemorrhage in which a pure motor hemiplegia (PMH) was found as the only symptom. In 1965, a study published by Fisher and Curry\textsuperscript{7} outlined the clinical criteria of PMH: complete or incomplete paralysis of the face, arm, and leg without loss of sensation or disturbance of visual field, no dysphasia, apraxia or agnosia\textsuperscript{21}. Although considered a common finding in specific settings of hemispheric strokes, PMH is quite unusual as the clinical manifestation of pontine hemorrhage.

**CASE REPORT**

A 50 year-old male was admitted to the emergency room because of sudden weakness on his right leg and arm. An elevated arterial blood pressure (200/130 mmHg) was disclosed but his general physical exam was otherwise unremarkable. Neurological examination revealed a complete right-sided hemiparesis. Consciousness was not impaired and there were no cerebellar signs or cranial nerves dysfunction. There were no signs of ipsilateral or contralateral VIIth nerve palsy but a supranuclear facial palsy was present. A mild dysarthria was recorded and the patient referred no dizziness, tinnitus or dysphagia. Previous medical history indicated untreated elevated blood pressure for the last 5 years. A computed tomographic (CT) scan showed a hematoma located in the left pontine tegmentum (Fig 1).

He was discharged on the 7th in-hospital day with only a discrete dysarthria. No significant motor weakness was recorded at discharge. The patient was submitted to a magnetic resonance imaging of the head three months after discharge. Multiple hyper-signal images in T2 were recorded throughout the subcortical white matter as well as in the pons. A left midpons negative signal was recorded which was indicative of hemosiderin or calcification (Fig 2).

**DISCUSSION**

Brainstem hemorrhage has been extensively reported in the literature and focus specifically on the different pontine lesions and their clinical manifestations, etiologic factors, treatment and prognosis\textsuperscript{3,4,6,8,10}. The clinical presentation of a pontine hemorrhage varies considerably and depends on the site of the hematoma and its extension\textsuperscript{2,4,10,14}. Involvement of cranial nerve function in association with cerebellar and pyramidal tract dysfunction with tetraplegia is the rule in the clinical presentation of extensive bilateral pontine hematomas\textsuperscript{3,4,9,10,12,14,17,18}. Other clinical manifestations are coma, abnormal postures, punctiform pupils, changes in blood pressure and abnormal breathing patterns\textsuperscript{10}.

The etiologic PPH factors more commonly reported in the literature are hypertensive hemorrhages and arteriovenous malformations\textsuperscript{13,19}. It is generally accepted that about 10% of all cerebral hemorrhages occur in the pons\textsuperscript{10}.
The lack of intraventricular bleeding and the favorable outcome on follow-up associated with a diagnosis of hypertension led the authors to believe that an arteriovenous malformation was not the underlying etiology in this case although an arteriogram would conclusively exclude this possibility. Aleksic and Budzilovich, Caplan and Goodwin, and Gillilan discussed the anatomic correlation between brainstem blood vessels and different diagnostic possibilities. According to these authors, the distribution of the arterial branches in the pons, mainly under the intrinsic circulation viewpoint, composes distinct topographic zones that in turn may generate distinct clinical syndromes. The case reported herein displayed a head CT scan documenting hemorrhage located on the left paramedian pontine tegmentum. Involvement of the median perforating branches of the superficial arteries of the median line, that is, anterior spinal, vertebral and basilar arteries was likely in this patient. The pyramidal tract is irrigated mainly by median and paramedian branches along its course within the brain stem. The clinical involvement of the pyramidal tract may be produced either directly by an impairment of its arterial supply or indirectly by compression.

The importance of early CT in the diagnosis and management of such cases is clear. A patient suffering from brainstem stroke may be considered a candidate for anticoagulant therapy. Such an action would be clearly contraindicated if fresh hemorrhage is noted on CT scan. Our review indicate that persons suffering hemorrhage into the pons who have preserved consciousness, particularly with unilateral hematoma, have a more favorable prognosis. In these cases, vigorous supportive care is warranted.

Although PPH has been usually classified into three types as mentioned above, their clinical significance is still uncertain, since their prognosis are not significantly different except for the small unilateral tegmental type. Thus, for prognostic purposes, it may be best to classify PPH into the small unilateral tegmental type and others (massive, basal-tegmental, and bilateral tegmental). The survival rate was 94.1% for the small unilateral tegmental type as opposed to 18.2% for the others.

A lateral tegmental brainstem hemorrhage syndrome has been recognized and it manifests clinically with ipsilateral conjugated gaze palsy, ipsilateral internuclear ophthalmoplegia, a small reactive pupil, ipsilateral ataxia and contralateral sensorimotor impairment.
A hemorrhagic origin of PMH is considered classically to be a rare event and was reported only once in the literature. Fisher and Curry did not find any case of hemorrhage in their series of cases. However, they did not exclude the existence of hemorrhage as a cause of PMH.

The case reported herein had a lesion in the left pontine tegmentum without the other signs of cerebellar, ocular movement, Vth and VIIth cranial nerve dysfunction. There were clear signs of pyramidal tract involvement in the pons, a finding usually not reported in the literature as the only clinical manifestation. The absence of neurological signs other than pyramidal tract can only be explained by a variation in the vascular anatomy of the pons.

REFERENCES