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ISOLATED MEDULLARY HEMORRAGE: A SERIES OF THREE ETIOLOGICALLY DISTINCT CASES.

Neurology

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ABSTRACT

BACKGROUND

Isolated haemorrhages rarely occur in the medulla. With regards to brainstem they are more common in pons, secondary to hypertension. Analysis of reported literature for causes of medullary haemorrhage include vascular malformations, haemorrhagic transformation of an infarct, hypertension and in a large subsect, causes remain unknown. We report, three etiologically variable, isolated acute medullary haemorrhages, encountered in a tertiary care hospital in India

KEYWORDS

Brainstem haemorrhage; Medullary haemorrhage

INTRODUCTION

Isolated haemorrhages rarely occur in the medulla. With regards to brainstem they are more common in pons, secondary to hypertension. Analysis of reported literature for causes of medullary haemorrhage include vascular malformations, haemorrhagic transformation of an infarct, hypertension and in a large subsect, causes remain unknown. We report, three etiologically variable, isolated acute medullary haemorrhages, encountered in a tertiary care hospital in India.

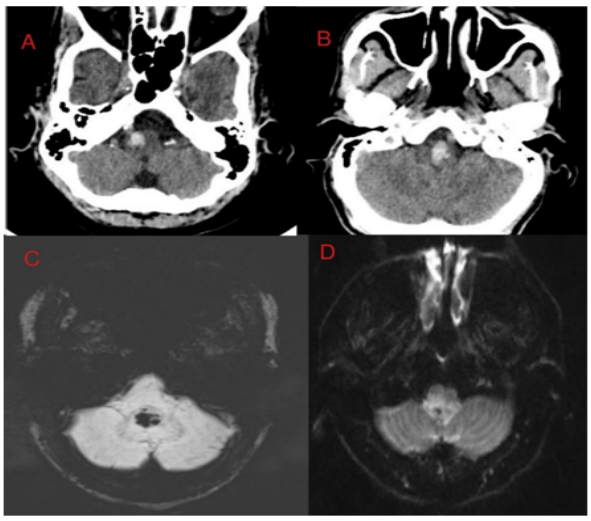

Figure Legend

Figure A: CT brain axial sections showing right medullary haemorrhage. Figure B: CT brain axial cuts showing right medullary haemorrhage. Fig C and D: Well defined heterogeneously enhancing lesions in the lower half of the fourth ventricle with oedema in the right side of the medulla with multiple foci of hemorrhages seen in GRE (fig C) with hypointense signals in T2 Flair cuts (fig D) as well.

Case vignette:

Case 1:

A 73-year female, hypertensive on irregular medication presented with one day duration of left sided hemiparesis, vomiting and vertigo with blood pressure of 180/100 mmHg. Examination added deviation of the tongue to the left on protrusion, with reduced right palatal movement. CT brain showed the presence of right hemi medullary haemorrhage (Figure A). The patient was initiated on antihypertensives, anti-oedema measures, physiotherapy and palatal stimulation. She was further evaluated with imaging (MRI and CT angiogram) and coagulation panel to rule out vascular malformations and bleeding diathesis respectively. The aetiology of intra parenchymal haemorrhage was subsequently attributed to hypertension. After a few days in intensive care the patient was discharged, with moderate improvement in power and palatal weakness.

Case 2:

A 50-year normotensive male, presented to the emergency room with one day history of reduced responsiveness and right hemiparesis. He had undergone thrombolysis the previous day, following a diagnosis of ST segment Elevation Myocardial Infarction in an outside hospital, after which he had developed weakness. Examination confirmed right hemiparesis with GCS- E2 V2 M3 with CT brain revealing the presence of an acute haemorrhage in the left medullary parenchyma (Figure B). The patient required mechanical ventilation initially. In his subsequent course, the patient continued to improve, was weaned off ventilator and initiated on physiotherapy. But three weeks after admission the patient had a sudden cardiac arrest, and despite best resuscitative efforts, was declared dead.

Case 3:

A 35-year male, presented with complaints of three episodes of loss of consciousness lasting for 30 seconds each, over a period of 2 months, with history of intermittent twitching of his eyelids. Initial radiological evaluation revealed multiple foci of haemorrhages in the right medulla. On contrast administration there was heterogeneous enhancement of the lesion, with additional involvement of lower half of the fourth ventricle. The possibilities of ependymoma, sub-ependymoma or meningioma were considered. The patient was advised neurosurgical consult but was subsequently lost to follow-up. (Fig C and D)

DISCUSSION

Conventionally, with regards to isolated brainstem haemorrhages, pons has been referred to, as the most common site, with prevalence of midbrain and medullary haemorrhages though reported, rare[1]. An aetiological review of reported literature for brainstem parenchymal haemorrhages showed hypertension as the most common cause of pontine hemorrage in contrast to medullary haemorrhages. Shen et al[2] did a etiological analysis of all the reported 32 cases of isolated medullary hemorrage between 1963 to 2017 including their own case which was published in 2018, out of which 13 (40.6%) patients had

vascular malformations, 2 were haemorrhagic transformation of infarcts, four due to hypertension(12.5 %), one due to anticoagulation, one case secondary to coagulopathy and 11 (32.3%) cases with undetermined causes.

The difference in the incidence and aetiology of haemorrhages in pons and medulla, which are anatomically adjacent structures with almost similar blood supply is intriguing. Firstly, the number of long penetrating arteries into pons is much higher than into medulla[3] and since hypertensive bleeds occur predominantly into penetrating arteries, this could be a significant reason. Furthermore, microaneurysms which are liable to rupture are postulated to occur at an increased rate in these penetrating arteries[4]. Hypertensive bleeds tend to occur either during the onset of high blood pressures or much later in old age, following lipohyalinosis of these penetrating arteries. Secondly, the mean blood flow and flow velocities are higher in basilar than in vertebral arteries contributing to increased shear pressure into basilar penetrating branches when compared to vertebral penetrating branches[5]. This possibly may cause increased incidence of rupture of penetrating arteries in pons than in medulla. Additionally, Mutlu et al[6] hypothesised that most medullary arteries run horizontally in comparison to the vessels of pons, making them less liable to rupture. But despite the quoted explanations, the correlation between hypertension and medullary hemorrage continues to be controversial.

The prognosis of patients with medullary hemorrage is variable, as are its clinical presentation, considering the site and size of the lesion, progression and time of presentation. When compared with pontine haemorrhages where mortality ranges from 30 % to 80 %[7], insufficient data is available to generate a definitive prognostic conclusion in medullary haemorrhages. In Sun-Uk Lee et al's[8] case series of 11 consecutive patients, after three months, eight of the patients had MRS ≤ 2. In a case series by Barinagarrementeria et al[9], all 4 cases recovered with minor residual deficits at three months, with one patient's sudden demise after 4 months.

CONCLUSION

We here report three etiologically variable acute medullary haemorrhages, and possibly to the best of our knowledge, the first case of thrombolytic therapy induced medullary haemorrhage. Isolated medullary haemorrhage though rare, have a diverse etiological spectrum, which should be investigated especially vascular malformation. Prognosis, from the limited literature analysis seems favourable.

DECLARATIONS

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Authors' contributions – enclosed in title page

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