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Remote Primary Ventricular Hemorrhage in Aneurysmal Subarachnoid Rupture

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Abstract

Background: Intra-ventricular hemorrhage (IVH) post-aneurysmal subarachnoid hemorrhage (aSAH) is common, where peri-ventricular intra-cerebral clot has ruptured the ependymal lining. However primary intra-ventricular hemorrhage (PIVH) without associated parenchymal clot is rare. Migration of blood via outlet foramina of the fourth ventricle through CSF pulsation and flow is thought to be the mechanism for this occurrence and would account for small volumes in the sub-acute setting. *Case Report*: Here, we present a case of a 52-year-old woman with CT demonstrating spontaneous middle cerebral artery (MCA) bifurcation aneurysmal subarachnoid hemorrhage and a large acute third ventricular clot spanning the foramen Monroe. *Conclusion*: Reflexive hypertensive hemorrhages may occur in aSAH and that these hemorrhages may occur in varied locations.

Keywords: Cerebral Hemorrhage, Hypertension, Intracranial Aneurysm, Middle Cerebral Artery, Subarachnoid Hemorrhage.

Introduction

Intra-ventricular hemorrhage is associated with significant mortality and morbidity. The incidence of primary intra-ventricular hemorrhage (PIVH) without parenchymal clot is reported as 3% of all intra-cranial hemorrhage presentations [1]. Most cases of IVH result from the extension of parenchymal hemorrhage or rupture of an aneurysm [2]. Although intra-ventricular hemorrhage and its association with aneurysmal rupture is well described in the literature, remote hemorrhages are poorly differentiated and understood. The simultaneous occurrence of remote hypertensive intra-cerebral hemorrhage (ICH) following aneurysmal rupture has been published three previous times in the literature [3]. However, to our knowledge this is the first documented case of remote PIVH associated with aneurysmal subarachnoid hemorrhage (aSAH). We performed a literature review of both primary intra-ventricular bleeds and other rare hemorrhages associated with aSAH. This case serves to provide an opportunity to examine a CT demonstrated hemorrhage that led to a delayed diagnosis of an aneurysmal rupture.

Case Report

A 52-year-old woman presented with an episode of generalized tonic-clonic seizure witnessed by her husband. The seizure lasted one minute and was associated fecal and urinary incontinence. Following a five-minute post-ictal period of unresponsiveness, she regained consciousness and remained confused. She had no significant past medical or surgical history, took no regular medications and was a non-smoker with no significant family history.

Initially, the patient was transported by ambulance to a non-neurosurgical hospital. Examination by an ED consultant revealed mild hypertension (BP 157/88) and otherwise normal

vital signs (GCS 15; pupils equal and reactive, heart rate 66 beats per minute; blood pressure 167/88; respiratory rate 16/minute; temperature 36.8ºC). Her central nervous system and review of systems examinations were unremarkable.

The initial CT performed at the peripheral hospital showed acute sub-arachnoid hemorrhage with fresh blood tracking along the course of the left middle cerebral artery into the Sylvian fissure. A hyper-dense circumscribed lesion anteriorly in the third ventricle measuring 16×13×12 mm was reported. This lesion appeared to be continuous with a further component present in the anterior horn of the left lateral ventricle 1.91 cm in diameter. The lesion was hyper-dense posteriorly but iso-dense to the adjacent brain. No layering of blood was identified on the lateral or third ventricles.

On arrival to the neurosurgical department, the patient was GCS 13, E3V4M6; equal and reactive pupils. She was drowsy orientated to person, denied a headache and reported feeling sleepy. Her power was globally reduced to 4/5. She was started on treatment for aSAH despite the negative CT angiogram. A repeat non-contrast CT brain showed an increase in the sub-arachnoid hemorrhage and new intra-ventricular extension into the posterior horns of both lateral ventricles. The apparent mass in the third ventricle seen on the afternoon CT was noted to have increased in size and density indicating that it could be a clot. Formal angiogram found a multi-lobulated aneurysm projecting infero-laterally from the left MCA bifurcation, which approximated 6.5×4 mm. Two daughter sacs were evident. No other aneurysm or arterio-venous malformation (AVM) was identified.

The patient received craniotomy and aneurysmal clipping on day two of her presentation and localized to the tube post-clipping. Post-operative CTA brain, day three, demonstrated the aneurysm was excluded from circulation with no other occult aneurysms identified. The patient

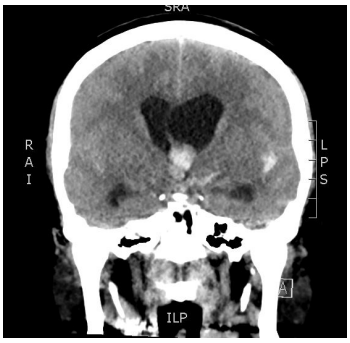

Fig.1: *Coronal section demonstrating the third ventricular hemorrhage, with small amount of blood at the site of aneurysm.*

Fig.2: *Axial CT brain of remote third ventricular hemorrhage.*

Fig.3: *Sagittal CT brain of remote third ventricular hemorrhage.*

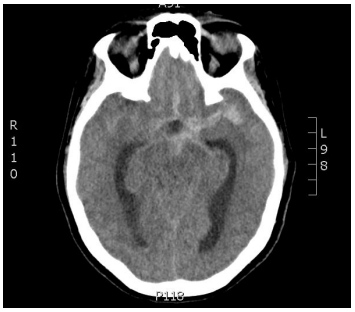

Fig.4: *Axial CT brain demonstrating the volume of blood at the inter-hemispheric fissure.*

remained in intensive care unit for six days. She remained a GCS 14 and MSQ 2-3 post-extubation. She remained shunt-dependent and received a ventriculo-peritoneal shunt day twenty-one. She was discharged on day twenty-five with further out-patient rehabilitation. Two years on she has made full recovery return to driving with no major neurological deficits.

Discussion

Primary intra-ventricular hemorrhage (PIVH) was first described by Sanders in 1881 as the flooding of the ventricle by blood without the presence of any rupture or laceration in the ventricular wall [4]. Venous angioma has been suggested to be the most likely cause when a parenchymal injury is not present [5]. Other studies have reported hypertension followed by vascular malformation as the most common risk factors for PIVH [1].

The incidence of IVH associated with aSAH varies from 10% [6] to 70% [7] depending on whether the parenchymal clot is included. This suggests that the etiology of a ventricular clot in the setting of aSAH may also be quite varied. In the presented CT images there is no continuity between the patient's sub-arachnoid hemorrhage (SAH) and the intra-ventricular clot. It is reasonable to assume that the bleeds are independent in origin. Despite an extensive literature review this appears to be the first case reported of a remote PIVH with aSAH.

Middle cerebral artery aneurysms have been more commonly associated with ICH while anterior communicating artery aneurysms and posterior circulation aneurysms with IVH [8]. Rarely aneurysmal rupture presents without SAH [8,9]. There are three case reports of remote ICH following aSAH. Hypertension is postulated to be the most reasonable cause as a reflex of the initial aneurysmal bleed or independent of the bleed [3]. The location of IVH without parenchymal clot has previously been reported not to correlate with either the clinical status of the patient or the nature, site or size of the bleeding lesion [6].

Fig.5: *Left carotid angiogram demonstrating the MCA bifurcation aneurysm.*

However, recent studies report 64-65% mortality following SAH with IVH associated with poor clinical status [10,11]. Patient age, location, and size of an aneurysm are described as predictors for development of IVH post aSAH [12]. Other studies reported a smaller size of aneurysm to be predictive of occurrence and severity of ICH [7]. This reflects the progression in our understanding of predictors for development of IVH/ICH in aSAH.

The presence of IVH in the setting of aSAH is a risk factor for the development of hydrocephalus [12]. Location of an aneurysm may also predict shunt dependency when IVH is not present [13]. Placement of an EVD is the procedure of choice for the treatment of acute hydrocephalus in patients with SAH [14]. This was performed bilaterally as a trapped ventricle was suspected. This patient had a Graeb score of 6 and while clot itself is a predictor of shunt dependency severity of or size of clot is not [12]. Jabbarli *et al*. found no impact of IVH severity on shunt dependency, while other recent studies report a Graeb score of 6 or greater to be significantly associated with the development of hydrocephalus. The development of predictive scores may help with early assessment of shunt dependency [15,16].

A seizure occurs in approximately 26% of patients following aSAH [17]. Early onset seizures are thought to negatively affect the grading of

the aSAH. This subset of patients may have significantly improved recovery given the early seizure distorting the grading [17]. Current opinion is that post-resuscitation neurological status is the best predictor of outcome [18]. However, patients that are WFNS grade I initially tend to do better than those patients that improve to a grade I [18].

Rupture of the lamina terminalis may be one cause of IVH in aSAH. However, this is more often associated with an ACA/ACOM aneurysm where the membranous layer offers little resistance against bleeding forces [5]. The ascent of blood products through CSF outlet forminae via elastic recoil of brain tissue following cardiac systole has also been suggested as a reason for IVH [6]. Neither theory adequately serves to explain the combination of the radiographic findings presented here. Certainly, the delay in observing the mass as a clot is reasonable given in the hyper-acute setting (first 4–6 h), which during this time, CT imaging does not reveal hemorrhage well, because it appears iso-dense to the brain [8]. In this case, the delay to radiological diagnosis did not halt pre-emptive aneurysmal treatment or the need for a further radiological investigation.

Conclusion

This report illustrates a case where the location of hemorrhage and CT findings do not match leading to delayed radiological diagnosis. Whether IVH represents an isolated clot or passage of blood via CSF spaces is difficult to delineate in the literature and may represent different etiologies. The link between aSAH and IVH, is described in the literature, however, it is difficult to find examples of studies that reflect the presentation or course of this patient. There are no previous reports of a link between PIVH and aSAH. The presentation of this case aims to show that while IVH in aSAH is known that the etiology behind the IVH may be varied. It is our hypothesis that this case is representative of how reflexive hypertensive hemorrhages may occur in aSAH and that these hemorrhages may occur in varied locations, whether this itself impacts patient presentation and outcome will require further investigation.

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