Hemispheric Infarct Following a Cerebellar Hematoma: A Rare Coincidence

Abstract
Concomitant cerebral infarction developing soon after a parenchymal intracerebral hemorrhage is a rare occurrence. Usually, these remote site changes follow tumor decompression and are associated with hemorrhagic changes rather than infarcts. We report a case of a fatal malignant internal carotid territory infarct in a hospitalized patient being conservatively managed for a vermian hematoma and discuss the probable pathophysiology. Stroke physicians need to be aware that spontaneous intracerebral hematoma patients have a potential threat of developing large vessel occlusion with malignant cerebral infarcts, especially after surgical decompression. Although the exact pathogenesis is unknown, size of the clot, intraventricular hemorrhage, hydrocephalus, and aggressive reduction of blood pressure appear to be predictive factors.

Keywords: Hematoma, intracerebral, parenchymal, vermian

Introduction
Remote site bleeds are often encountered after tumor decompression and haematoma evacuation, in clinical practice. However the occurrence of a supratentorial infarct following an infratentorial cerebellar bleed is sparsely reported and often rarely encountered. Here we describe a case of a 73-year old patient who presented to us with a primary cerebellar haematoma which progressed to a supratentorial infarct following conservative management. We discuss the clinical dilemma, theories and management outcomes in such patients with a thorough review of literature.

Case Report
A 73-year-old diabetic, hypertensive, male presented with a history of sudden-onset altered sensorium. On admission, nearly 4 h after the ictus, he was opening eyes to call, obeying commands, and had a slurred speech. His blood pressure (BP) was high on admission (200/120 mmHg) and his blood sugars were elevated (236 gm%). Computed tomographic (CT) scan of the brain showed a posterior fossa hematoma with early obstructive hydrocephalus [Figure 1]. He was managed conservatively as his family members were not willing for surgery. His sensorium remained intact and he gradually improved with conservative measures. On the 5th day after admission, his sensorium deteriorated and he developed a left-sided weakness. Repeat CT scan showed a large right hemispheric infarct [Figure 2]. He was electively ventilated, and a decompressive surgery was offered to the family who decided against surgery. In spite of elective ventilation and full decongestants, he succumbed on day 5 of the second ictus.

Discussion
Cerebral infarction following spontaneous intracerebral hematomas (SICHs) is uncommon. Wang et al. observed an incidence of 8% infarcts in their cohort of 212 patients with SICH.[1] Prabhakaran et al. detected a 22.9% prevalence of associated infarcts as evidenced by diffusion-weighted image (DWI) abnormalities in a cohort of 118 SICH patients.[2] The majority of these infarcts, however, were small subcortical and subclinical. Large vessel occlusion causing malignant cerebral infarction has been rarely reported.[3,4] We add a case of vermian hematoma with infarct to our earlier reported two cases of malignant infarction following evacuation of a supratentorial SICH.

Several mechanisms have been postulated to explain the possible occurrence of an infarct following a spontaneous hematoma.
Table 1: The varied cases of remote site bleed following tumor excision and decompression

| Author            | Age/sex | Primary diagnosis            | Site of bleed | Management                      | Outcome  |
|-------------------|---------|------------------------------|---------------|---------------------------------|----------|
| König et al.       | 56/male | Meningioma                   | Cerebellar bleed | Conservative therapy           | Dead     |
|                   | 42/female | Craniopharyngioma            | Cerebellar bleed | EVD                            | Dead     |
|                   | 59/female | Glioma                       | Cerebellar bleed | EVD                            | Good     |
| van Calenbergh    | 58/male | Metastasis of a keratinizing epithelioma | Cerebellar bleed | Conservative therapy | Good     |
| Kuroda et al.     | 63/male | Pituitary tumor              | Cerebellar bleed | EVD, VPS                       | Good     |
|                   | 72/male | Tuberculum sellae meningioma | Cerebellar bleed | VPS, decompressive surgery     | Good     |
| Brisman et al.    | 58/female | Sphenoid ridge meningioma   | Cerebellar bleed | Conservative therapy           | Good     |
| Papanastassiou et al. | 73/male | Tuberculum sellae meningioma | Cerebellar bleed | NA                            | Good     |
| Brisman et al.    | 54/female | Suprasellar meningioma      | Cerebellar bleed | EVD, decompressive surgery     | Disabled |
| Cloft et al.      | 47/male | Sphenoid ridge meningioma   | Cerebellar bleed | NA                            | Good     |
| Tomii et al.      | 37/male | Craniopharyngioma            | Cerebellar bleed | Conservative therapy           | Good     |
| Friedman et al.   | 64/male | Metastasis                   | Cerebellar bleed | Conservative therapy           | Good     |
|                   | 36/male | Glioma                       | Cerebellar bleed | EVD                            | Good     |
|                   | 53/male | Glioma                       | Cerebellar bleed | Conservative therapy           | Disabled |
|                   | 47/female | Schwannoma                  | Cerebellar bleed | Conservative therapy           | Good     |
|                   | 47/female | Tuberculum sellae meningioma | Cerebellar bleed | Conservative therapy           | Disabled |
|                   | 34/male | Craniopharyngioma            | Cerebellar bleed | Conservative therapy           | Good     |
|                   | 55/male | Metastasis                   | Cerebellar bleed | Conservative therapy           | Good     |
|                   | 36/male | Glioma                       | Cerebellar bleed | EVD                            | Good     |
|                   | 53/male | Glioma                       | Cerebellar bleed | Conservative therapy           | Disabled |
|                   | 47/female | Schwannoma                  | Cerebellar bleed | Conservative therapy           | Good     |
|                   | 47/female | Tuberculum sellae meningioma | Cerebellar bleed | Conservative therapy           | Disabled |
| Honegger et al.   | 54/male | Intraventricular meningioma  | Cerebellar bleed | Decompressive surgery          | Disabled |
|                   | 28/male | Ganglioglioma                | Cerebellar bleed | NA                             | Good     |
|                   | 33/male | Astrocytoma                  | Cerebellar bleed | NA                             | Good     |
| Marquardt et al.  | 31/male | Histiocytoma                 | Cerebellar bleed | EVD, decompressive surgery     | Disabled |
|                   | 42/male | Glioma                       | Cerebellar bleed | Conservative therapy           | Good     |
|                   | 73/male | Glioma                       | Cerebellar bleed | Conservative therapy           | Disabled |
|                   | 44/male | Glioma                       | Cerebellar bleed | EVD                            | Disabled |
| Siu et al.        | 64/male | Temporal tumor               | Cerebellar bleed | EVD                            | Dead     |
| Brockmann et al.  | 58/female | Temporal meningioma         | Cerebellar bleed | Conservative therapy           | Good     |
| Yang et al.       | 15/male | Pleomorphic xanthoastrocytoma | Cerebellar bleed | Conservative therapy           | Good     |
| Amini et al.      | 36/female | Oligodendroglioma            | Cerebellar bleed | Conservative therapy           | Good     |
|                   | 53/male | Glioblastoma                 | Cerebellar bleed | Conservative therapy           | Good     |
| Sasani et al.     | 14/male | Dysembryoplastic neuroepithelial tumor | Cerebellar bleed | Conservative therapy           | Good     |
| Mandonnet et al.  | 49/male | Meningioma                   | Cerebellar bleed | Decompressive surgery          | Good     |
| Rezazadeh et al.  | 60/male | Meningioma                   | Cerebellar bleed | Conservative therapy           | Good     |
| Paul et al.       | 23/male | Xanthoastrocytoma            | Cerebellar bleed | NA                             | NA       |
| Huang et al.      | 45/female | Sphenoid ridge meningioma   | Cerebellar bleed | Conservative therapy           | Good     |
|                   | 66/female | Sphenoid ridge meningioma   | Cerebellar bleed | Conservative therapy           | Good     |
|                   | 18/male | Suprasellar tumor            | Cerebellar bleed | Conservative therapy           | Good     |
|                   | 59/female | Oculomotor nerve tumor       | Cerebellar bleed | Conservative therapy           | Good     |
|                   | 65/male | Meningioma                   | Cerebellar bleed | Conservative therapy           | Good     |
| Dincer et al.     | 43/male | Astrocytoma                  | Cerebellar bleed | Conservative therapy           | Good     |
|                   | 49/female | Sphenoid ridge meningioma   | Cerebellar bleed | Conservative therapy           | Good     |
|                   | 44/male | Oligodendroglioma            | Cerebellar bleed | Conservative therapy           | Good     |
|                   | 37/female | Astrocytoma                  | Cerebellar bleed | Conservative therapy           | Good     |
| Hara et al.       | 44/male | Anaplastic oligoastrocytoma  | Cerebellar bleed | Conservative therapy           | Good     |

Contd...
The majority of such infarcts are small and subcortical, conforming to border-zone or single perforator territories. Few recent studies have observed subclinical DWI lesions in 15% of patients with acute intracerebral hematoma (ICH) attributable to cerebral amyloid angiopathy. Since both ischemic and hemorrhagic stroke share common risk factors and certain common pathogenic mechanisms, it is possible that an ischemic stroke may simply be a co-occurrence in the presence of common risk factors. Hypotension
due to attempted aggressive reduction of BP following a hypertensive bleed may precipitate an ischemic event. Surgery for evacuation of the hematoma may be associated with intraoperative hemodynamic instability, which can result in an ischemic event. Though unlikely, infarcts following craniotomy may also be attributable to iatrogenic compression of vascular structures during craniotomy or through the durotomy defects. Large hematomas with surrounding edema can theoretically directly compress the adjacent cerebral vessels causing ischemia. Infection with persistent fever, dehydration, and electrolytic imbalance can all result in a hypercoagulable state following a bleed and can result in ischemia. One another postulated mechanism for ischemia is the massive release of blood and blood breakdown products into the CSF and subsequent inflammatory changes in the smooth muscle of the large cerebral arteries. Cerebral vasospasm, due to the presence of concomitant intraventricular hemorrhage or after indirect vessel manipulation at the time of craniotomy, is another suspected mechanism for ischemia. After acute brain injury, autoregulation may be abolished such that cerebral blood flow is linearly related to cerebral perfusion pressure. Aggressive BP lowering beyond the lower limits of cerebral autoregulation might induce cerebral ischemia in chronic hypertensive ICH patients. Our patient did not undergo surgery and the infarct and hematoma were located in different compartments. He did develop features of early sepsis while under treatment for vermian hematoma. Sepsis with an associated hypercoagulable state may be the precipitating factor resulting in the infarct.

Attempts have been made to predict the risk of developing concomitant infarcts in patients with SICH. Wang et al. observed that the presence of intraventricular hemorrhage (IVH), hydrocephalus, the volume of intracranial hematoma, and neurosurgical intervention were important predictors of infarction of which IVH had the most statistical significance. Similarly, Prabhakaran et al. observed that the factors independently associated with DWI abnormality were a prior ischemic stroke, lowering of mean arterial pressure by over 40%, and craniotomy for ICH evacuation. Our patient did have the evidence of fourth ventricular blood but did not satisfy most of the above criteria.

The rarity of such complications makes it less likely to appear in our clinical diagnosis and we feel one should consider this scenario as well. The remote site hemorrhages which are often encountered after tumor or hematoma decompressive surgery are shown in Table 1.

The exact etiopathogenesis of these large infarcts remains uncertain and the management of such large infarcts needs to be on similar lines as for any ischemic infarct. The outcome, however, is usually grave.

**Conclusion**

Stroke physicians need to be aware that SICH patients have a potential threat of developing large vessel occlusion with malignant cerebral infarcts, especially after surgical decompression. Although the exact pathogenesis is unknown, size of the clot, IVH, hydrocephalus, and aggressive reduction of BP appear to be predictive factors.

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**Conflicts of interest**

There are no conflicts of interest.

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