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Case Report

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*

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Introduction

Remote site bleeds are often encountered after tumor decompression and haematoma evacuation, in clinical practice. However the occurence of a supratentorial infarct following a 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.

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Table 1: The varied cases of remote site bleed following tumor excision and decompression

Author	Age/sex	Primary diagnosis	Site of bleed	Management	Outcome
Konig *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
	58/female	Sphenoid ridge meningioma	Cerebellar bleed	Conservative therapy	Good
Brisman *et al*.	73/male	Tuberculum sellae meningioma	Cerebellar bleed	NA	Good
Papanastassiou *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...

Table 1: Contd...

Author	Age/sex	Primary diagnosis	Site of bleed	Management	Outcome
Landeiro *et al*.	45/male	Ruptured Acom	Cerebellar bleed	Conservative therapy	Good
	58/female	Left frontal convexity meningioma	Cerebellar bleed	Right FTOZ and 3rd nerve decompression	Good
	42/female	Left cavernous sinus meningioma	Cerebellar bleed	Conservative therapy	Good
Nam *et al*.	61/male	L3-L4, L4-L5 canal stenosis	Bilateral cerebellar	SOC	Good
Chadduck	59/male	Cervical canal stenosis	Left cerebellar	SOC + EVD	Good
Mikawa *et al*.	75/male	Cervical fusion	Bilateral cerebellar	SOC + EVD	Good
Andrews and Koci	36/male	Lumbar spinal scoliosis	Bilateral cerebellar	EVD	Good
Gabel *et al*.	40/female	Lumbar disc	Right cerebellar	SOC + EVD	Good
	57/female	Lumbar spondylolisthesis	Bilateral cerebellar	EVD	Good
Satake *et al*.	62/male	Cervical intramedullary tumor	Cerebellar	SOC	Good
Morandi *et al*.	34/male	Cervical schwannoma	Right cerebellar+left temporal	Conservative therapy	Good
Friedman *et al*.	43/male	Thoracic herniated disc	Right cerebellar	Conservative therapy	Good
	56/male	Lumbar spinal stenosis	Bilateral cerebellar	Conservative therapy	Good
Thomas *et al*.	38/female	Thoracic tumor	Left cerebellar+right temporal	Conservative therapy	Good
Karaeminogullari *et al*.	73/female	Lumbar spinal stenosis	Left cerebellar	SOC	Good
Brackmann *et al*.	52/male	Lumbar spondylolisthesis	Bilateral cerebellar	EVD	Good
Kanya *et al*.	48/female	Lumbar herniated disc	Bilateral cerebellar	Conservative therapy	Good
Calisaneller *et al*.	67/female	Lumbar spondylolisthesis	Bilateral cerebellar	Conservative therapy	Good
Agrawal *et al*.	47/female	Posterior fossa meningioma	Diffuse SAH	Conservative therapy	Good
Garg *et al*.	28/female	Right sylvian fissure arachnoid cyst	Bilateral frontal EDH	Craniotomy and evacuation of EDH	Good
	50/female	Left vestibular schwannoma	Diffuse SAH	VPS	Good
	20/female	Left subdural hygroma	Supratentorial diffuse hematoma	Conservative therapy	Good
	38/female	Right vestibular schwannoma	Left parieto-temporal EDH	Craniotomy and evacuation of hematoma	Good
	64/male	Cerebellar hematoma	Left fronto-parieto-temporal SDH	Craniotomy and evacuation of hematoma	Good
	13/female	Left lateral ventricular neurocytoma	Right frontal EDH	Craniotomy and evacuation of hematoma	Good

SAH – Subarachnoid hemorrhage; EDH – Extradural hemorrhage; VPS – Ventriculoperitoneal shunt; EVD – External ventricular drainage; NA – Not available; FTOZ – Fronto-temporo-orbito-zygomatic; SOC – Suboccipital craniectomy; Acom – Anterior communicating artery

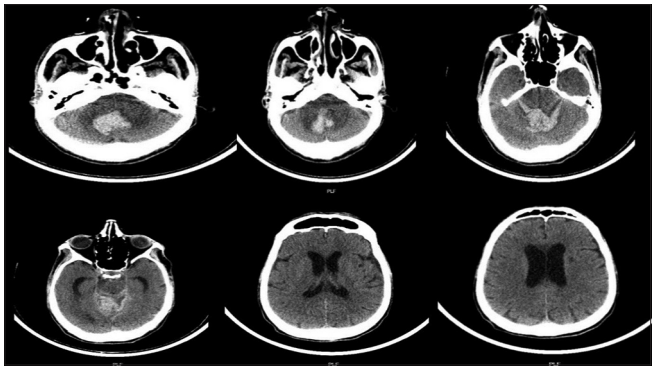

Figure 1: Computed tomography brain showing vermian bleed

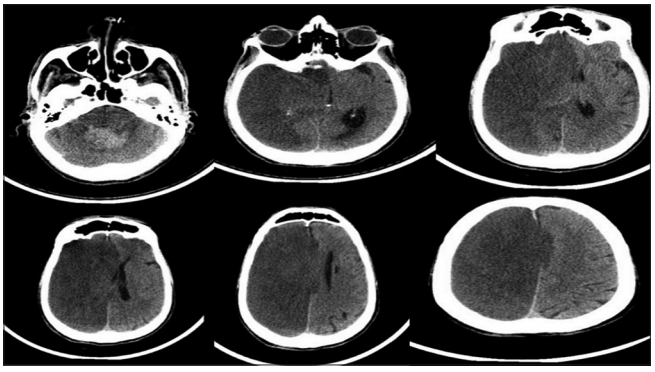

Figure 2: Computed tomography brain showing massive right internal carotid artery territory infarct

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.[1] 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.[2] 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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