Supratentorial intracerebral hemorrhage following posterior fossa surgery

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Five cases of supratentorial intracerebral hemorrhage following posterior fossa surgery are reported. Possible etiologies are discussed, but in only one case can a definite etiology (hypertension) be found. The differential diagnosis of declining level of consciousness after posterior fossa surgery must include supratentorial intracerebral hemorrhage, and computerized tomography seems to be the diagnostic method of choice.

Key Words • cerebral hemorrhage • cranial fossa, posterior • postoperative cerebral hemorrhage

Of the many complications that may follow neurosurgical procedures, intracerebral hemorrhage remote from the operative site is fortunately rare. Recent experience with an occipital lobe intracerebral hematoma following retromastoid craniectomy and microvascular decompression of the trigeminal nerve for trigeminal neuralgia led us to review our experience with this complication.

Summary of Cases

Tabulations of all surgical procedures and complications occurring on the neurosurgical service of Presbyterian-University Hospital (PUH) from January, 1972, through December, 1977, were reviewed. The records of all persons having surgical procedures for, or suffering morbidity from, intracerebral hemorrhage were examined to determine if the hemorrhage followed a surgical procedure. In addition, the files of the Division of Neuropathology were reviewed for this same period and all cases of intracerebral hemorrhage were examined for any relationship to surgical procedures. Hemorrhages occurring at the operative site were excluded.

No hemorrhages remote from the operative site were found following supratentorial or spinal procedures. During the time period of the review, 825 posterior fossa procedures were performed of which 687 were retromastoid craniectomy for various cranial nerve disorders. Of the remainder, 52 procedures were performed, for acoustic neurolipoma resection, 50 for other tumors, and 36 for miscellaneous disorders. Four supratentorial hematomas and one intraventricular hemorrhage occurred in the immediate postoperative period. Thus, the incidence of hemorrhage was 0.6% for all posterior fossa procedures. These five patients are reported in detail below and their clinical data summarized in Table 1.

Case Reports

Case 1

This 65-year-old woman developed typical trigeminal neuralgia in the second and third
### TABLE 1
Selected characteristics of patients

<table>
<thead>
<tr>
<th>Factors</th>
<th>Case 1</th>
<th>Case 2</th>
<th>Case 3</th>
<th>Case 4</th>
<th>Case 5</th>
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<tr>
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<td>41</td>
<td>64</td>
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<td>glossopharyngeal neuralgia</td>
<td>atypical trigeminal neuralgia</td>
<td>anesthesis dolorosa</td>
<td>acoustic neurilemmoma</td>
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<td>history</td>
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<td>hypothryoidism</td>
<td>uterine carcinoma</td>
<td>chronic serous otitis media; carpal tunnel syndrome; hysterec- tomy</td>
<td>breast carcinoma; leukemia</td>
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<td>negative</td>
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<td>negative</td>
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<tr>
<td>preoperative:</td>
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<td></td>
<td></td>
<td></td>
<td></td>
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<td>positive findings</td>
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<td></td>
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<td></td>
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<tr>
<td>first degree heart block: serum sodium</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td>medications</td>
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<td>thyroid, pentazocine</td>
<td>flurazepam, Fiorinal</td>
<td>propoxyphene, diazepam</td>
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<td>sitting</td>
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<td>160/120</td>
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<td>180/105</td>
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<td>200/?</td>
<td>170/96</td>
<td>168/?</td>
<td>190/90</td>
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<td>45 min postop</td>
<td>immediately postop</td>
<td>18 hrs postop</td>
<td>immediately postop</td>
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<td>rt basal ganglia</td>
<td>rt frontoparietal</td>
<td>lt frontal</td>
<td>intraventricular</td>
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<td>hemisphere drainage, death</td>
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<td>hemiparesis</td>
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<td>death</td>
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<td>no abnormality</td>
<td>no abnormality</td>
<td>not obtained</td>
<td>no abnormality</td>
<td>not obtained</td>
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</tbody>
</table>


**Text:**

divisions of the right trigeminal nerve ($V_2$ and $V_3$) in May, 1976. She was treated with percutaneous thermocoagulation of the Gasserian ganglion at another hospital. Her pain recurred in September, 1977. Carbamazepine afforded only partial relief. She was therefore admitted to PUH for consideration of microvascular decompression of the trigeminal nerve. Past medical history revealed chronic mild low-back pain and paroxysmal atrial tachycardia for which she was being treated with digoxin.

Physical examination was normal with the exception of hypalgesia and hypesthesia in the distribution of the right $V_2$ and $V_3$. The electrocardiogram (EKG) showed first degree heart block. The serum sodium was 132 mEq/liter. Other studies including complete
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Case 1

This 55-year-old woman had severe pain in the right side of the throat, diagnosed in 1969 as glossopharyngeal neuralgia. Carbamazepine controlled her symptoms until 1973. She was then admitted for microvascular decompression of the right ninth cranial nerve. Past medical history revealed hypothyroidism treated with thyroid extract.

Physical examination was normal except for her blood pressure, which reached a maximum of 150/90 mm Hg on one occasion. Her only other medication was pentazocine. The preoperative tests were the same as outlined in Case 1, and were normal; CT scanning was not available at the time, but preoperative angiography showed only mild atherosclerosis. She underwent right retromastoid craniectomy and microvascular decompression of the ninth cranial nerve. She complained of severe headache 45 minutes postoperatively, and suddenly lapsed into coma. The wound was immediately re-explored and the lateral cerebellar hemisphere was excised because of suspected cerebellar edema.

Postoperatively she remained comatose, with fixed and dilated pupils. Right retrograde brachial cerebral angiography showed hydrocephalus and a slight right-to-left shift of midline structures. She died after several days of supportive care. Postmortem examination revealed a large intracerebral hematoma involving the right corpus striatum, thalamus, and temporal lobe. The hemorrhage had ruptured into the lateral ventricle. Multiple sections of the hematoma and surrounding cerebral tissue showed no evidence of vascular malformation or neoplasm.

Case 2

This 41-year-old woman suffered from atypical trigeminal neuralgia with hyperactive autonomic dysfunction that began in 1975. Because many evaluations failed to reveal a definite etiology for her pain and multiple drug trials failed to provide significant relief, she was admitted for microvascular decompression of the trigeminal nerve. Past medical history revealed uterine carcinoma treated by hysterectomy in 1967. There was no suggestion of tumor recurrence. Her medications were flurazepam and Fiorinal.
Physical examination was normal with the exception of mild hypalgesia and hypesthesia of the entire right side of her face. Preoperative studies showed only mild ventricular enlargement and cortical atrophy on CT scan. She underwent retromastoid craniectomy and microvascular decompression of the right trigeminal nerve. Intraoperative air embolism was treated by aspiration of 25 cc of air through an indwelling right atrial catheter.

Postoperatively she was alert but was noted to have "impaired eye movements." Five hours later she was obtunded and suddenly screamed, after which her eyes deviated to the right and horizontal nystagmus and left hemiparesis were evident. Her wound was immediately re-explored. No hematoma or edema was found. A CT scan then showed a right frontoparietal intracerebral hematoma. Her condition stabilized and she progressively improved without surgical intervention. She was discharged to a rehabilitation center with a moderate left hemiparesis which has since resolved.

Case 4

This 64-year-old woman suffered severe right-sided facial pain believed in 1962 to be secondary to a dental neuroma. Resection of the neuroma, multiple alcohol injections, and avulsion of the inferior alveolar nerve failed to relieve her pain, which spread to involve all the divisions of the right trigeminal nerve. Percutaneous thermocoagulation of the Gasserian ganglion at another hospital left her completely anesthetic over the right side of her face, but did not diminish her pain. She was admitted for further surgical therapy. Past medical history revealed chronic serous otitis media on the right, carpal tunnel syndrome, hysterectomy for benign disease, and lumbar laminectomy for herniated disc. Her medications were propoxyphene and diazepam. Her father had died of a cerebral hemorrhage.

Physical examination demonstrated analgesia and anesthesia of the right side of the face with absence of the corneal reflex. Preoperative studies were normal. She underwent retromastoid craniectomy and selective section of the right trigeminal nerve.

Postoperatively she had no new neurological deficit. Eighteen hours later she was disoriented, hypotonic, and hemiparetic on the right. A CT scan showed a large left frontotemporal intracerebral hematoma. Her condition worsened and the hematoma was evacuated. Histological examination of the hematoma and surrounding white matter showed no evidence of vascular malformation or neoplasm. Recovery was complicated by pneumonia, urinary tract infection, and slowly improving dysphasia. At the time of discharge she had a mild right hemiparesis and flat affect. Subsequently her affect has returned to normal, but the hemiparesis persists. Her anesthesia dolorosa is unimproved.

Case 5

This 62-year-old woman had left V2 and V3 trigeminal neuralgia since 1972. Carbamazepine relieved her pain, but the drug was stopped in 1975 because of an allergic reaction. Infraorbital neurectomy afforded pain relief for 4 months. She was referred for microvascular decompression of the trigeminal nerve. Past medical history revealed mastectomies for carcinoma in 1957 and 1961. There was no history of recurrence. Chronic myelocytic leukemia was diagnosed in 1975 and treated; at present evaluation she was in remission.

Physical examination showed trophic changes of the right upper extremity secondary to irradiation and hypesthesia and hypalgesia in the left V1 distribution. Preoperative laboratory studies were normal. Skull films suggested, and tomograms confirmed, widening of the left internal auditory canal. The audiogram revealed retrocochlear hearing loss on the left. A CT scan showed an enhancing mass in the left cerebellopontine angle. This was confirmed by angiography. The patient underwent left retromastoid craniectomy and resection of an acoustic neurommoma.

Postoperatively she was initially responsive to pain but became unresponsive in 30 minutes. A CT scan showed acute hydrocephalus and blood in the left lateral, third, and fourth ventricles. A ventriculostomy was performed, and the blood was removed by irrigation. She remained comatose and died on the fifth postoperative day. Permission for postmortem examination was denied.

Discussion

As can be seen in Table 1, the only consistent finding in these cases was that they were
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all women. The side and position of the procedure were the same in four of the five patients. None of these features are considered significant. The majority of our retromastoid procedures were done with the patient in the modified sitting position. Approximately 60% of the patients undergoing retromastoid craniectomy were female and the same proportion had right-sided procedures.

There are numerous causes of intracerebral hemorrhage, including trauma, hypertension, vascular malformation (including aneurysm and arteriovenous malformation), coagulation disorders (including therapeutic anticoagulation, thrombocytopenia, complications of hepatic and neoplastic disease), direct hemorrhage into a neoplasm, infectious and inflammatory disorders (including arteritis and mycotic aneurysm), hemorrhage into recent cerebral infarct, and multiple rare etiologies. In addition, it is unusual not to be able to find an underlying cause for such a hemorrhage.

Most of these potential etiologies can be dismissed in the cases under discussion. There was no significant head trauma in any patient. Coagulation disorders were eliminated with appropriate preoperative tests. In addition, all patients underwent surgical procedures without unusual bleeding at the operative site. Two patients had a history of neoplasm, but neither was known to have metastatic disease. In the other patients there was no indication of a neoplasm. Histological examination of the hematomas and surrounding cerebral tissue showed no evidence of tumor, but pathological specimens were not obtained in the patients with remote tumors. We cannot totally exclude the possibility of hemorrhage into neoplasm, but it seems unlikely. No patient had preoperative evidence of cerebral infarction. Sepsis was documented only as a postoperative complication.

The question of an underlying vascular malformation is more difficult. In one patient, preoperative angiography and postmortem examination eliminated this possibility. In the others, preoperative angiograms were not done. In two patients, no abnormal tissue was seen at the time of operation or by histological examination of biopsied specimens.

Although the relative frequency of the underlying disorders listed above has recently been questioned, hypertension is considered the leading cause of non-traumatic intracerebral hemorrhage. None of the reported patients was known to be hypertensive preoperatively by history or preoperative blood pressure recordings. Intraoperative blood pressure records for the retromastoid procedures were reviewed. One patient remained normotensive (less than 150/80 mm Hg) for the entire procedure. Three patients had transient elevations in the range of 160/95 to 180/120 mm Hg for less than 5 minutes. In one patient (Case 2) the pressure was consistently in the range of 170/90 to 180/110 mm Hg.

Maximum blood pressure in the recovery room was approximately 162 to 170 mm Hg systolic and 90 to 96 mm Hg diastolic, with the exception of one patient (Case 2) who had a systolic pressure of 200 mm Hg 45 minutes before the precipitous decline in her level of consciousness.

No surviving patient developed persistent postoperative hypertension. Both patients who died were hypertensive postoperatively until their deaths. Only in Case 2 can hypertension be implicated in the development of the hemorrhage. In addition to persistent intraoperative hypertension and a very high recovery room blood pressure, she was found at postmortem examination to have mild cardiomegaly. Furthermore, her hematoma was located in the corpus striatum, which is a frequent location for hypertensive intracerebral hemorrhage.

The remaining patients had only modest elevations of blood pressure and the lobar locations of the hematomas are less typical of hypertensive hemorrhage. It is important to note, however, that the operative records may not reflect the blood pressure at certain critical times, such as intubation, extubation, and transportation to the recovery room, when stimulation is maximal and pressures much higher than those recorded may be reached.

The literature on postoperative intracerebral hemorrhage remote from the operative site is sparse. We are unable to locate any reports of cases similar to ours. Intracerebral hemorrhage following cardiac surgery is well documented, but these patients are anticoagulated intraoperatively and most have had episodes of significant hypertension. Hemorrhages into recently infarcted cerebral tissue following carotid endarterectomy are likewise well documented but the typical
pattern includes preoperative evidence of cerebral ischemia and the hemorrhage usually occurs several days postoperatively.\textsuperscript{1,4,10}

Pontine hemorrhage following supratentorial surgery for tumor was reported by Madow in 1960,\textsuperscript{6} but a review of his cases indicates that these were brain-stem hemorrhages that occurred secondary to transtentorial herniation.

Fisher\textsuperscript{3} has stated that hemorrhages may be seen following operative procedures in which vasopressors are used. These hemorrhages are "atypically located compared with the hypertensive hemorrhages" and only a "modest rise in blood pressure seems to precipitate hemorrhage." His cases are not reported in detail. Vasopressors were not used in any of our cases.

The relationship of the modified sitting position of the patient at operation to the etiology of these hemorrhages is unclear. Hypotension was not noted in any of our patients. Changes in intracranial dynamics in this position are more likely to produce extracerebral rather than intracerebral hemorrhage. The possibility of occlusion of carotid or vertebral vessels in the neck by improper positioning of the head leading to intraoperative infarction should be mentioned. Restoration of blood flow postoperatively might then lead to hemorrhage within the infarcted brain. The good initial postoperative status of most of these patients argues against this possibility. In addition, as noted above, the time course of hemorrhage is more rapid than expected for hemorrhage into an area of infarction.

Although the etiology of these hemorrhages is not clear, it is important to recognize that bleeding may occur at sites remote from the operation. The differential diagnosis of a declining level of consciousness following posterior fossa surgery must be considered in the differential diagnosis of decreased level of consciousness following posterior fossa surgery. We hope to stimulate further reports of such cases with careful consideration of possible etiologic factors so that this unusual but serious complication may be avoided in the future.

References


Summary

We have presented five cases of supratentorial intracerebral hemorrhage following retromastoid craniectomy. In four, the etiology is unknown although modest blood pressure elevations were noted intraopera-

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