Role of dural fenestrations in acute subdural hematoma

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Object. Patients with acute subdural hematomas (ASDHs) have higher mortality and lower functional recovery rates compared with those of other head-injured patients. Early surgical decompression and active intensive care treatment represent, so far, the best way to assist these patients. Paradoxically, one of the factors contributing to poor outcomes in cases of ASDHs could be rapid surgical decompression, owing to the severe extrusion of the brain through the craniotomy defect in response to acute brain swelling. To avoid the deleterious consequences of abrupt decompression of the subdural space with disruption of brain tissue, the authors have adopted a new surgical technique for evacuation of ASDHs. This procedure consists of creating multiple fenestrations of the dura (MFD) in a meshlike fashion and removing clots through the small dural openings that are left open, avoiding the creation of a wide dural opening and the disruption of and additional damage to brain tissue.

Methods. Thirty-one patients (26 male and five female patients with a mean age of 32.5 years) harboring ASDHs were treated using this method. On admission there were 16 patients (51.5%) with Glasgow Coma Scale (GCS) scores of 3 to 5, 11 patients (35.5%) with GCS scores of 6 to 8, and four patients (12.9%) with GCS scores of 9 to 12. Postoperative computerized tomography scans of the brain revealed evacuation of more than 80% of the hematoma in 29 of 31 patients. The overall mortality rate in this group was 51.6%.

Conclusions. This preliminary report of a new surgical approach for patients who have sustained ASDHs should be considered to avoid abrupt disruption of the brain and to allow the gradual and gentle release of subdural clots. This is especially important in cases in which there are severe midline shifts and a tight brain. Further clinical studies should be conducted in a more selected series to estimate the impact of this new procedure on morbidity and mortality rates.

KEY WORDS • acute subdural hematoma • dural fenestration • cerebral tissue disruption • decompression surgery

The management and treatment of ASDHs over the years have been disappointing despite the advancement and development of intensive care units. Patients suffering from ASDH continue to have higher mortality and lower functional recovery rates compared with those of other head-injured patients. Surgically treated patients have been found to bear the poorest prognoses, an estimate based on the fact that this group always includes moribund patients who have little chance of survival in any case. Therefore, the advantages of surgical treatment remain controversial, particularly in patients in whom there is a large midline shift, compared with those in whom there is a relatively small SDH. In these cases, the primary structural and ischemic damages to brain tissue play a more important role in determining outcome. Surgical evacuation of the hematoma by creating a large decompression craniotomy and a wide opening of the dura mater is still considered the treatment of choice; however, abrupt decompression of the hematoma can lead to brain disruption and secondary ischemia in the brain surrounding the craniectomy site. In our opinion, this could be another factor leading to poor results in the management of ASDHs. Furthermore, in many cases, acute brain swelling and brain extrusion, which take place shortly after decompression, can lead to a catastrophic situation during the intervention owing to the impossibility of dural closing, bone flap relocation, and, sometimes, even appropriate closure of the scalp.

On this basis, we adopted a new surgical approach to keep intact, at least partially, the natural covering of the brain and to avoid the development of severe mushrooming and disruption of tissue caused by acute brain swelling.

Clinical Material and Methods

Surgical Procedure

After elevation of a wide flap of scalp, the temporal muscle is thoroughly dissected from the skull and a large osteoplastic frontotemporoparietal craniotomy is performed. The bluish bulging dura mater is opened through a 5-mm incision at the level of the sylvian cistern. This usually constitutes the first step of brain decompression by releasing an entrapped bloody cistern. With the aid of a delicate skin hook, the dura mater is successively picked out and small cuts ranging from 4 to 5 mm are made over the entire exposed area (Fig. 1).
A suction tube is gently applied over the dura mater and the clots are delicately aspirated through the dural fenestrations. In case of active bleeding through one of the small holes, the dural opening is slightly enlarged to permit adequate hemostasis. Subdural washing is performed using a No. 8 feeding tube to allow removal of more adherent clots.

The bone flap is replaced after multiple small holes are created, and the flap is partially fixed using No. 0-0 silk with a loose knot. The edges of the fascia are attached to the muscle at the opposite side to achieve maximal decompression.

**Patient Population**

The clinical characteristics of the 31 patients surgically treated using the MFD procedure are presented in Table 1. Twenty-six patients (83.9%) were male and five (16.1%) were female. The mean age (± SD) of the patients was 32.5 years (± 16.5 years) and their ages ranged from 2 to 83 years. Twenty-eight patients presented with a closed head injury, two with penetrating injuries, and one patient with a spontaneously occurring SDH. Thirteen patients had been involved in a motor vehicle accident, 10 patients had been injured due to falling from a height, and five had sustained an assault injury. Eight of 31 patients suffered from additional extracranial injuries. At admission 27 patients were comatose (GCS Scores 3–5 in 16 patients, GCS Scores 6–8 in 11, and GSC Scores 9–12 in four). Abnormal pupillary responses were found in 17 patients on admission; bilateral fixed dilated pupils were found in 10 patients and unilateral dilated nonreactive pupils in seven patients.

**Case Management Protocol**

All patients were admitted to the emergency department and received primary care as usually recommended by advanced trauma life support guidelines. After surgery, all patients were admitted to the neurosurgical intensive care unit where they underwent continuous monitoring of blood pressure, ICP, and central venous pressure. The treatment protocol included slight hyperventilation (PCO2 values 30–32 mm Hg) and administration of mannitol 20% (0.5 g/kg/4 hrs), Pentothal (3 mg/kg/hr), or propofol 3-5 mg/kg/hr). Phenytoin was the first choice for anticonvulsive treatment, but valproic acid was given perioperatively in case the patient demonstrated an allergic reaction to phenytoin. Prophylactic antibiotic therapy included first-generation cephalosporins and gentamicin for patients with closed head injuries, or disodium ceftriaxone administered for 3 days in patients with penetrating injuries.

**Results**

Clinical results are presented in Table 2.
Surgical Outcomes

All 31 patients underwent the same surgical procedure that was described earlier. Ten patients, seven of whom had sustained concomitant extracranial injuries, received blood transfusions. No wound infection, CSF fistula, or meningitis was seen in this series. Only one patient required a repeated operation; this was due to the development of a delayed contralateral EDH.

Neuroimaging Findings

The preoperative CT findings in these patients are shown in Table 1. Computerized tomography scanning of the brain was performed in all patients harboring an ASDH within a maximum of 3 hours postinjury; scans demonstrated that ASDHs averaged 10.66 ± 3.8 SD in width, with a midline shift measuring an average of 10.3 mm ± 4.1 mm SD (Fig. 2 left). Concomitant intracranial injuries included the following: diffuse axonal injury in 12 patients, brain contusion in 14 (Fig. 3 left), subarachnoid hemorrhage in 18, EDH in five, and intraventricular hemorrhage in six.

Postoperative CT findings are presented in Table 2 (Fig. 2 right). Computerized tomography scanning of the brain was performed in all the patients within 6 hours to 5 days postoperatively. Residual SDH (more than 20% of the original hematoma) was found in only two patients who otherwise required no additional operation because their ICP was controlled and they had suffered a minimal mass effect. In two patients, CT scans demonstrated the presence of an EDH; in one case the EDH was located on the side opposite the SDH that was surgically treated and in the other case an ipsilateral EDH was located at the posterior edge of the craniotomy site and was treated conservatively. In none of the patients did fungus cerebri develop in the craniotomy site. Enlargement of brain contusions was found in 10 patients (Fig. 3 right) who otherwise required no reintervention, and none of the patients experienced delayed intracerebral hemorrhage leading to surgical implications. Brain infarction was noticed in nine patients.

Control of ICP

Continuous postoperative monitoring of ICP was performed in all patients for at least 3 days or until death. In 27 patients ICP was measured using a subdural feeding tube catheter and in four patients by using an intraventricular catheter. In nine patients the peak ICP was less than 15 mm Hg. In 10 patients ICP was controlled (15–25 mm Hg) by medical treatment, whereas in 12 patients the ICP remained persistently elevated over 25 mm Hg (Table 2).

Incidence of Morbidity and Mortality

The distribution of Glasgow Outcome Scale scores 1 year after injury are shown in Table 2. Sixteen patients (51.6%) died; in 12 the cause was brain herniation. Three patients died as a result of septic complications and one patient died of an acute cardiorespiratory distress syndrome that most probably was pulmonary embolism. None of the patients experienced wound infection or postoperative CSF leak. Extracranial complications included pneumonia in 21 patients, sepsis in 13, and pulmonary embolism in one patient.

Discussion

The morbidity and mortality rates of patients with ASDH are still very high despite modern intensive care and surgical treatment. Associated parenchymal injuries or alterations in cerebral autoregulation have been considered responsible for these poor results. Many factors, such as the preoperative neurological condition of the patient, advanced age, extent of concomitant brain injury, presence of severe extracranial injuries, timing of...
which was proposed by Britt and Hamilton, could lower used, allows decompression of the brain, but cannot avoid formed using the classic approach even when a graft is late complications, and to take a shorter time than the clas-
ra mater. This is clearly achieved by creating MFD in a the entire hematoma without complete opening of the du-
omy. We then found that it is possible to remove almost brain swelling and extrusion of tissue through the craniot-
results in the loss of the natural protective layer covering surgery, and uncontrollable postoperative increased intra-
cranial hypertension, have been advocated as being re-
ponsible for the disastrous results in the management of patients with ASDH. Most authors agree that, in se-
vere cases of ASDH, significant structural damage is sustained by the underlying brain. This is probably the main reason why neurosurgeons continue to learn and teach that the great killer of all head injuries is ASDH; de-
spite all efforts, the mortality rate remains almost un-
changed and the low functional recovery rate is also the same. On the other hand, the diversity of medical and surgical options is limited. Based on the significant-
disappointing experience we have encountered using the surgical procedure commonly used for evacuation of ASDH—wide decompressive craniotomy and wide open-
ing of the dura mater—we propose a new surgical ap-
proach to treat this devastating injury. We started with the hypothesis that wide opening of the dura mater with prompt removal of clots could be deleterious because it results in the loss of the natural protective layer covering the brain and, in many cases, the development of acute brain swelling and extrusion of tissue through the craniot-
omy. We then found that it is possible to remove almost the entire hematoma without complete opening of the du-
ra mater. This is clearly achieved by creating MFD in a meshlike fashion without the hazard of brain extrusion ings. This was avoided by meticulous fenestration of the dura mater and suctioning through the dural open-
ments. This was avoided by applying the suction tube gently, and not directly, over the fenestrations while removing the small suction tip. 2) In some cases hemostasis can be difficult to control through small dural openings. In these cases we enlarged the dural openings as much as necessary to achieve satisfactory hemostasis. It is important to note that almost all subdural clots were removed in the majority of patients, and in only two of our patients did postoperative CT scans of the brain display a residual SDH measuring more than 20% of the original hematoma. Otherwise, these patients required no reoperation. On the contrary, postoperative brain hypoten-
sion seems to be the main factor in the development of EDHs in two of our patients, as well as the enlarging of cerebral contusions, as was shown during the postopera-
tive period in an additional 10 patients.

We were surprised that, although the dura mater re-
mained open, none of the patients presented with a CSF fistula or an important local bulging of the operation wound by CSF accumulation under or over the bone flap.

Fig. 3. Left: Preoperative CT scan obtained in a 20-year-old man revealing an ASDH on the left side with a severe hemorr-
ic contusion on the posterior part of the left temporal lobe. Right: Postoperative CT scan obtained 2 days after evacuation of the subdural clots through MFD. Note the enlargement of the brain contu-

sion although there are almost no signs of increased ICP (enlarging of the perimesencephalic cistern).

As indicated by Aruga and colleagues, during surgical treatment of ASDH bone decompression achieved using dural grafting seems successful in some patients, but surely enhances cerebral swelling and exacerbates edema in others. We believe that the MFD procedure allows further decompression of blood and CSF for a few days, at least until the effects of delayed brain edema and ischemic changes disappear.

Despite the findings of Seelig, et al., regarding the importance of prompt evacuation of subdural hematoma, Wilberger and associates found no significant statistical difference in the morbidity and mortality rates of patients who underwent surgery early. Information provided by the national databank (Mar-
marou, et al.) reveals the same results. Obviously, the majority of patients who underwent surgery early were in a deep comatose state owing to brain herniation, but the effect of the abrupt opening of the dura mater and the rapid evacuation of clots in patients with underlying brain injury until now has been underestimated. It is those cases in which the decision of whether to operate is controver-
sial because of the disproportion between midline shifts and the width of the hematoma appearing on CT scans of the brain. Regarding our series, we note that we have deliberately operated even on moribund patients, 10 of whom (32%) had bilateral fixed pupils. Nevertheless, the mortality rate in our series seems to be lower than those of other reported series (Haselsberger and coworkers, 57%; Stone, et al., 59%; Seelig, et al., 57%; and Wilberger and associates, 66%). Wide decompressive craniotomy accomplished using the MFD method could play a main role in the treatment and outcomes of these patients.

The MFD procedure is associated with some pitfalls. 1) There is some hazard of further injury to the brain during incision of the dura and suctioning through the dural openings. This was avoided by meticulous fenestration of the dura achieved using an appropriate brain spatula and by applying the suction tube gently, and not directly, over the fenestrations while removing the small suction tip. 2) In some cases hemostasis can be difficult to control through small dural openings. In these cases we enlarged the dural openings as much as necessary to achieve satisfactory hemostasis. It is important to note that almost all subdural clots were removed in the majority of patients, and in only two of our patients did postoperative CT scans of the brain display a residual SDH measuring more than 20% of the original hematoma. Otherwise, these patients required no reoperation. On the contrary, postoperative brain hypoten-
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Only one patient required CSF diversions, but this was due to delayed hydrocephalus. Moreover, the MFD procedure will permit additional decompression in case of additional ischemic damage. The decompressive effect seems to be more important than simple trephination and irrigation of the subdural space, as was observed by Shigemori and colleagues in his series of patients with ASDH and low GCS scores.

Conclusions

We believe that the MFD procedure allows for the safe removal of subdural clots in patients with ASDH, while the protective property of the dura mater is not altered. The MFD method is mainly indicated for patients with tight brain injury and severe midline shifts, compared with those harboring relatively small SDHs. Further comparative clinical studies should be performed to evaluate the real value of this procedure and to assess its impact on mortality and morbidity rates.

References

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