Dynamics of subdural hygroma following decompressive craniectomy: a comparative study

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Object. This retrospective comparative cohort study was aimed at discovering the risk factors associated with subdural hygroma (SDG) following decompressive craniectomy (DC) to relieve intracranial hypertension in severe head injury.

Methods. Sixty-eight of 104 patients who had undergone DC during a 48-month period and survived > 30 days were eligible for this study. To assess the dynamics of subdural fluid collections, the authors compared CT scanning data from and the characteristics of 39 patients who had SDGs with the data in 29 patients who did not have hygromas. Variables significant in the appearance, evolution, and resolution of this complication were analyzed in a 36-week longitudinal study.

Results. The earliest imaging evidence of SDG was seen during the 1st week after DC. The SDG volume peaked between Weeks 3 and 4 post-DC and was gradually resolved by the 17th week. Among the mechanisms of injury, motor vehicle accidents were most often linked to the development of an SDG after DC ($p < 0.0007$), and falls were least often associated ($p < 0.005$). Moreover, patients with diffuse brain injury were more prone to this complication ($p < 0.0299$) than those with an evacuated mass ($p < 0.0001$). There were no statistically significant differences between patients with and without hygromas in terms of age, sex, Glasgow Coma Scale score, intraventricular and subarachnoid hemorrhage, levels of intracranial pressure and cerebral perfusion pressure, timing of decompression, and the need for CSF diversion. More than 90% of the SDGs were ipsilateral to the side of the craniectomy, and 3 (8%) of 39 SDGs showed evidence of internal bleeding at ~ 8 weeks postinjury. Surgical evacuation was needed in 4 patients with SDGs.

Conclusions. High dynamic accidents and patients with diffuse injury were more prone to SDGs. Close to 8% of SDGs converted themselves into subdural hematomas at ~ 2 months postinjury. Although SDGs developed in 39 (~ 60%) of 68 post-DC patients, surgical evacuation was needed in only 4. (DOI: 10.3171/2009.3.FOCUS0947)

KEY WORDS • decompressive craniectomy • subdural hygroma •
head injury • intracranial pressure

AUTOPSY verification of subdural fluid collections following severe head injury was first documented in 1733 by Professor Thomas Schwenneke.²⁰ In 1894 Mayo cared for a 12-year-old boy suffering from headache, dysphasia, and right hemiparesis following trauma.¹⁵ Trephination of the skull on the left side revealed a liquid similar to CSF, whose evacuation relieved the symptoms and led to functional recovery.^{9,15} In 1924 Naffziger⁴⁹ discovered a tear in the arachnoid membrane,

permitting egress of the CSF into the subdural space, which would then become a mass. In 1945 Walter Dandy^{16,17} coined the term “subdural hygroma” for a benign collection of CSF in the subdural space. In the 1980s, after the introduction of CT, SDGs became the subject of intense imaging investigations.^{4,6,15,22,28,33,64,65} Detailed electron microscopy studies of the arachnoid-dura interface by Haines et al.²⁵ and Schachenmayr and Friede⁶³ combined with documentation of CSF collections in the subdural space on contrast cisternography have helped us to better understand the pathogenesis of SDGs.^{27,46} The inflammatory response in the SDG cavity, as indicated by cytokine activation, results in neomembrane formation rich in porous and fragile capillaries. The rupture of these capillaries, together with stress-induced tearing of

Abbreviations used in this paper: ATP = adenosine-5'-triphosphate; CPP = cerebral perfusion pressure; DC = decompressive craniectomy; GCS = Glasgow Coma Scale; GOS = Glasgow Outcome Scale; ICP = intracranial pressure; MVA = motor vehicle accident; SDG = subdural hygroma; SDH = subdural hematoma.

TABLE 1: Summary of characteristics in 68 patients with or without SDG following DC for severe head injury*

Variable	No. (%)			p Value
	Total	w/ SDG	w/o SDG	
no. of patients	68	39	29	
male sex	50 (73.5)	27 (69.2)	23 (79)	NS
mean age (yrs)	27.8	24.9	29	NS
mechanism of injury				
MVA	45 (66)	31 (79.5)	14 (48.3)	0.0007
fall	13 (19)	3 (7.7)	10 (34.5)	0.005
assault	7 (10.3)	3 (7.7)	4 (13.8)	NS
other	3 (4.4)	2 (5.1)	1 (3.4)	NS
clinical characteristics				
mean admission GCS score	6.8	6.8	6.9	NS
mean admission GCS motor score	4.1	4.2	4	NS
patients w/APR	21	11	10	NS
mean injury severity score	30.5	31.4	29	NS
injury severity (no. of patients w/)				
>5-mm shift on CT	29 (42.6)	14 (35.9)	15 (51.7)	0.1918
compressed or obliterated BCs	53 (77.9)	33 (84.6)	20 (69)	0.1253
intraventricular hematoma	21 (30.9)	13 (33.3)	8 (27.6)	0.6108
subarachnoid hemorrhage	45 (66.2)	24 (61.5)	21 (72.4)	0.3458
diffuse injury	45 (66.2)	30 (76.9)	15 (51.7)	0.0299
evacuated mass	23 (33.8)	9 (23.1)	24 (82.8)	0.0001
treatment				
patients w/ early surgery†	31 (45.6)	16 (41)	15 (52)	0.3810
patients w/ delayed surgery‡	37 (54.4)	23 (59)	14 (48)	0.3810
ICP before DC (mm Hg)	22.6	22.2	23.1	NS
ICP after DC (mm Hg)	13.9	13.6	14.3	NS
CPP before DC (mm Hg)	69	68	69	NS
CPP after DC (mm Hg)	74.6	75	74	NS
patients needing CSF diversion	13	6 (46)§	7 (54)§	NS
GOS score				
1–3	30 (44)	15 (50)	15 (50)	NS
4–5	38 (55.9)	24 (63)	14 (37)	0.2760

* APR = abnormal pupillary response to light; BC = basal cistern; NS = not significant.

† Within 48 hours of injury.

‡ More than 48 hours after injury.

§ Percentages based on 13 patients total.

parasagittal veins, converts a benign-looking SDG into a potentially problematic chronic SDH.^{19,21,37,48,55,56} In the studies by Koizumi et al.³⁷ and St John and Dila,⁶⁴ 32 of 63 SDGs were bilateral, 12 were on the right side, and 19 were on the left side, indicating no predilection for any particular side of the brain.

In recent years, DC has been used more often as a means of reducing intracranial hypertension unresponsive to maximum medical treatment.^{1,2,10,11,23,29,47,58,68,70,74} Selecting DC as a second-tier option in the management of severe head injury mandates attention to a frequent complication of this operative procedure, namely the SDG.^{1,2,34,41,76}

In this investigation, we studied the clinical and imaging characteristics of 39 patients with SDGs and compared them with those of 29 patients without to better understand the dynamics of the initial appearance, increase

FIG. 1. Graph depicting volumetric CT evidence of an SDG after DC in 39 patients whose course of recovery was complicated by a hygroma. Fluid collection in the subdural space started from Week 1 post-DC, peaked at Week 4, and gradually declined and disappeared around Week 17. The volume of each SDG was calculated using the methods of Gebel et al. and Sucu et al. W = week.

FIG. 2. Noncontrast head CT scans obtained from a 24-year-old man who had been in an MVA for which he underwent DC in April 2000. His admission GCS score was 11 and the Injury Severity Score was 29. On Day 4 of his admission, he had to undergo urgent exploration because of neurological worsening. His postoperative clinical course was uneventful; however, on Day 8 after DC he underwent another CT study because of a decline in his mental status; the CT scan indicated evidence of an SDG with significant shift of the midline structures to the left. He underwent 2 attempts to evacuate the SDG by the insertion of a drainage system through a twist drill bur hole at bedside in the intensive care unit (arrow, Day 29). Eight months after the DC, he had an autologous cranioplasty. He remained neurologically intact 47 months after discharge from the institution.

in volume, and resolution of this seemingly benign-looking complication following DC.^{1-3,66,76,77}

Methods

The institutional review board of the University of Maryland School of Medicine approved this study.

Patient Population

During a 48-month period between March 2000 and March 2004, 104 of 967 patients with severe head injury

underwent DC. The clinical characteristics as well as the prehospital, initial medical, maximum medical, and surgical treatments in these patients have been reported elsewhere.^{2,3} Included in the present analysis were 39 patients in whom an SDG developed postoperatively and 29 patients without an SDG. Excluded were those patients who died shortly after DC (36 patients). Demographics, clinical and imaging profiles, and outcomes in these 2 groups of patients are listed in Table 1.

Timeline of SDG Evolution

We monitored the postoperative head CTs of the

TABLE 2: Composition of subdural fluid in 5 patients with SDGs

Case No.	Red Blood Cells (/mm ³)	White Blood Cells (/mm ³)	Protein (mg/dl)	Glucose (mg/dl)
1	40,000	726	408	48
2	22,000	17	556	78
3	13,000	55	246	88
4	8,950	50	118	61
5	1,990	17	60	100

group with SDGs for 36 weeks and measured the volume of hygroma fluid using the methods of Gebel et al.²⁴ and Sucu et al.⁶⁷ The mean volume of SDG fluid was ~ 10 ml during the 1st week, rose to 60 ml during the 4th week, and then gradually decreased and ultimately disappeared around the 17th week post-DC (Fig. 1).

Lateralization of SDGs

Thirty-six (92%) of 39 SDGs were ipsilateral to the site of DC and 3 were contralateral. Significant shifts of the midline structures were noticed in 1 (2.8%) of the 36 patients with an ipsilateral SDG and in 2 (67%) of 3 patients with contralateral hygromas (Fig. 2).

Hydrocephalus With or Without SDG

Six (15%) of 39 patients with SDGs had evidence of hydrocephalus during Weeks 5–8, 17, and 22 post-DC and underwent placement of a shunting device (Fig. 3). In 4 of these patients autologous cranioplasty was performed 1, 2, 5, and 26 days after CSF diversion, and in 2 patients a shunting device was inserted 35 and 155 days after cranioplasty for late-onset hydrocephalus. Among the patients without SDGs, hydrocephalus developed in 7 (24%). Criteria for the diagnosis of hydrocephalus were CT evidence of dilation of the temporal horn tips, globu-

FIG. 3. Computed tomography scans obtained in a 19-year-old man following an MVA, who underwent DC for sustained high ICP (22.5 mm Hg) and a right frontal blossomed contusion. Three axial CT scans obtained on Days 9, 15, and 21 showed the gradual appearance of an SDG. The hygroma was absent on a CT scan obtained 24 days later (Day 45), although there was evidence of hydrocephalus. A shunting device was inserted, and autologous cranioplasty was performed 2 days later. Three months after his accident the patient remained severely disabled (GOS Score 3).

FIG. 4. Three days after her admission, this 16-year-old girl experienced sustained ICP of 31.8 mm Hg for which she underwent DC. A follow-up CT scan indicated the first evidence of an SDG on Day 2 (D2) after DC. Although her condition was clinically stable, a head CT obtained on Day 56 (D56) showed evidence of internal bleeding. The SDH gradually disappeared, and she had cranioplasty on Day 228. Thirty-one months after the injury she was asymptomatic with a GOS score of 5.

lar deformation of the third ventricle, enlargement of the lateral ventricles, or evidence of transependymal absorption of CSF. Clinical evidence of a changed mental status or the arrest or slow progression of rehabilitation milestones, as judged by rehabilitation providers, also triggered new CT studies to confirm or disprove the presence of hydrocephalus.

Composition of SDG Fluid

We checked the composition and cellular content of the SDGs in 5 patients. This fluid was usually bloody and its protein content was high (Table 2). The fluid was collected either in the operating room or at the bedside under aseptic conditions. The volume of the collected fluid was anywhere from 10 to 50 ml. One sample cultured enterococci twice after bur hole evacuation of a contralateral SDG on Days 8 and 19 following DC (Fig. 2). The

patient in this case was treated with antibiotics. Tapping the SDG collection never resulted in complete resolution of the hygroma.

Conversion to SDHs

The unexpected appearance of bleeding in an SDG cavity was noted in 3 patients: in 1 on Day 55 of the post-DC period and in 2 on Day 57. One SDG with internal bleeding had to be drained (Fig. 4).

Surgical Intervention

Four patients (10%) underwent surgical drainage of an SDG—by twist drill in 1 patient, bur hole in 1, and percutaneous insertion of a draining system in 2.

Statistical Analysis

Statistical analysis was performed using JMP soft-

Fig. 5. Schematic representation of dural border cells as observed by electron microscope.²⁵

ware (versions 7 and 8, SAS Institute). Two-sided probability values were calculated using the likelihood ratio chi-square for the categorical variables in the 2 groups of patients with or without hygroma. Unpaired t-tests were performed for continuous variables to test for differences in the means between patients with and without hygroma.

Results

Patient and Injury Factors

Patients with SDGs were close to 4 years younger than those without, but this difference was not statistically significant. Motor vehicle accidents, which were responsible for 66% of the injuries, were significantly associated with SDG following DC. Among the 45 patients who had been in an MVA, an SDG developed in 31 (69%) and did not in 14 (31%); this difference was significant ($p = 0.0007$). In contrast, a fall was the injury mechanism in 10 of 29

patients without a hygroma versus 3 of 39 patients with an SDG, which represented a statistically significant difference between the 2 patient groups ($p = 0.005$).

Clinical Injury Severity

There were no significant differences between the 2 patient groups with respect to GCS score, GCS motor score, abnormal pupillary response to light, or injury severity score.

Imaging Profile

Diffuse injury⁴⁴ occurred in 30 of 39 patients with an SDG compared with 15 of 29 patients without hygromas; this difference was significant ($p = 0.0299$). A compendium of CT characteristics was considered as evidence of diffuse injury: the presence of a mass < 25 ml, effacement of gyral patterns, disappearance of the third ventricle, and variable compression or obliteration of the basal cisterns.

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Fig. 6. This 19-year-old woman underwent DC 5 days after her admission for ICP control and CPP management. Axial (*left*) and coronal (*right*) T2-weighted MR images obtained 14 days after a traumatic brain injury, indicating an SDG collection over and under the expansive duraplasty (*arrows*).

Furthermore, 82.7% of patients with no hygroma—compared with 23.1% of those with an SDG—had an evacuated mass ($p = 0.0001$).⁴⁴ A shift of the midline structures > 5 mm on preoperative CT, compressed or obliterated basal cisterns, and the presence or absence of an intraventricular or subarachnoid hemorrhage were not significant variables in the development of an SDG.

Medical and Surgical Management

Although Lang et al.³⁸ have reported a relationship between increased CPP and the appearance of an SDG, we could not verify that finding in our study. In addition, the timing of a DC did not play a role in the development of an SDG. Hydrocephalus occurred in 13 patients and required CSF diversion; 6 of these patients had a hygroma whereas 7 did not (Table 1).

Patient Outcome

Although an unfavorable outcome (GOS Score 1–3) was split between the 2 patient groups, there seemed to be an indication that patients with an SDG had a better outcome (GOS Score 4 or 5) than those without. Almost 67% of the patients with an SDG fared well compared with $< 40\%$ of patients without an SDG who fared well; however, this difference was not a statistically significant finding.

Discussion

Dynamics of CSF Physiology

The CSF located in the ventricular system and subarachnoid space constitutes ~ 150 ml of the 1600 ml de-

fined as the volume contained in the intracranial vault. It is a continually produced, absorbed, and circulated fluid providing both mechanical and chemical protection for the brain through the regulation of the CNS chemical environment, the removal of metabolic waste in a role analogous to the peripheral lymphatics, the support/buoyancy of the brain, and the maintenance of ICP in accordance with the Monro-Kellie doctrine.^{26,59,69} The CSF is formed in a diurnal fashion at a rate of ~ 35 ml/hour or 500 ml/day with maximal production occurring at around 2:00 a.m. and minimal production at around 6:00 p.m.⁵² Located in the lateral, third, and fourth ventricles, the choroid plexus is responsible for ~ 60 – 70% of the daily CSF production, with the remaining fluid generated through a number of mechanisms including the movement of water across parenchymal capillaries, across the ependyma, and via the generation of water from the oxidative metabolism of glucose.^{18,26,59,72}

Choroid Plexus Anatomy

The choroid plexus, responsible for the majority of CSF production, consists of a superficial layer made of a single sheet of epithelial cells, supported by a core of connective tissue interspersed with blood vessels of various diameters from small arteries to capillaries to small veins. The epithelial lining of the choroid plexus is a single sheet of polarized cuboidal to columnar cells with a smooth basal side, a more structurally complex luminal side containing numerous microvilli intended to increase the luminal surface area, and tight junctions composed of occludin and claudin found laterally between adjacent cells, all contributing to the formation of the blood-CSF

barrier.⁵⁹ The blood supply originates from the choroidal arteries, travels through a fenestrated network of capillaries, and ultimately drains into the vein of Galen via the internal cerebral veins at a rate of ~ 3 ml/g/min, 10 times that of the brain parenchyma.

Choroidal Production of CSF

In the simplest terms, the production of CSF at the choroid plexus is the result of the active secretion of Na, Cl, and bicarbonate ions in exchange for K ions across the luminal surface of the choroidal epithelium to create an osmotic gradient that allows movement of water from the vasculature into the subarachnoid spaces. The primary driving force of CSF secretion is an Na⁺/K⁺-ATPase transporter located on the luminal surface, which actively secretes 3 molecules of Na⁺ in exchange for 2 molecules of K⁺ at the expense of a molecule of ATP hydrolyzed.^{45,60,78} Additional transporters including the ion channel NKCC1, the K⁺/Cl⁻ cotransporter (KCC), and several K⁺-specific channels (Kir7.1, Kv1.1, and Kv1.3) are involved in generating an ionic gradient across the luminal surface.

In contrast to luminal transport mechanisms, basolateral ion movement is not as well understood. Several channels have been proposed to have a role in the movement of Na⁺ and Cl⁻ into cells from the basolateral surface, including Na⁺/H⁺ exchangers and an electroneutral Cl⁻/HCO₃⁻ exchanger, but the work to clarify these processes is ongoing.^{42,59} Intracellularly, the H⁺ and HCO₃⁻ needed for these channels are generated by carbonic anhydrase.

The movement of water along the osmotic gradient generated across the CSF barrier, a relatively water-impermeable structure, is facilitated by AQP1, a member of the aquaporin family of water channels that is expressed at both the basolateral and luminal cell surfaces.^{51,52,54}

Cerebrospinal Fluid Circulation

The flow of CSF originates at its sites of production within the ventricles, with the majority thought to be within the lateral ventricles. Cerebrospinal fluid continues through the foramina of Monro into the third ventricle, through the sylvian aqueduct into the fourth ventricle, and subsequently into the cisterna magna via the foramina of Luschka and Magendie. From the cisterna magna, CSF flows caudally into the spinal subarachnoid spaces, rostrally into the basal cisternal spaces (preptontine, interpeduncular, ambient, and suprasellar cisterns), and dorsally into the subarachnoid spaces over the cortical convexities and cerebellum. Movement of the CSF via these pathways is thought to occur primarily through bulk flow with multiple contributory factors generating a current including respiratory variation, pulsatility imparted by the cardiac pressures transduced through the circle of Willis within the basal cisterns, ciliary movement at the surface of the choroid, and production of new CSF itself.^{12,13,30}

Absorption of CSF

Under normal conditions, CSF is maintained at a relatively stable volume that is balanced by the difference between fluid production, as described above, and the return of fluid to the venous circulation. The traditional

view of CSF circulation and cycling has been that the major, perhaps sole, site of CSF absorption and return to the venous system is the arachnoid granulation. In actuality, CSF outflow involves both the arachnoid granulations and lymphatic drainage in what has been referred to as the “dual system of CSF drainage.”³² Arachnoid granulations or villi, also called “pacchionian bodies,” are small protrusions of the arachnoid membrane through the dura into the venous sinuses. These structures have also been demonstrated in the arachnoid angles of spinal nerves. A hydrostatic pressure gradient in which subarachnoid pressure exceeds venous sinus pressure forces the return of CSF to the venous system.¹⁴ Interestingly, in the reverse situation, when venous pressures exceed ICP (for example, venous sinus thrombosis), CSF return is inhibited via the arachnoid villi, but reverse flow is not seen.³² Electron microscopy studies have shown that the arachnoid cells of these granulations form large vacuoles that appear to simultaneously communicate with the luminal and subarachnoid portions, and thus allowing the movement of water.⁶⁹ As mentioned above, CSF resorption is believed to be facilitated through a lymphatic pathway. It is true that no lymphatic system exists within the CNS, but experimental data from both animals and humans have shown that tracer molecules injected directly into the brain can be found in the perivascular areas of cranial nerves and ultimately within cervical and spinal lymph nodes. Additionally, experimental occlusion of lymphatics draining the head in several animal models has led to increased ICP, suggesting an important role for lymphatic drainage.^{7,8,30,31,35,50}

Incidence of SDGs

In patients with a closed-head injury who have not undergone a DC, the reported incidence of SDG varies from 5 to 21%.^{5,22,33,39,40,53} Ohno and colleagues³³ have reported 43 SDGs (6%) in 715 patients, and in the Kaufman et al. study³³ the incidence of an SDG was 38 (4%) in 881 patients with a closed-head injury. Born and associates⁵ have described 16 patients (15%) with SDGs among 109 with blunt traumatic brain injury. The incidence of an SDG following DC for severe head injury is not well documented in the literature.^{1-3,34,66,76,77} In a 2009 study Yang et al.⁷⁵ demonstrated 11 cases (6.5%) of contralateral SDGs among 169 patients who had undergone DC for severe head injury. In an earlier study⁷⁶ these same authors documented 23 cases (21.3%) of SDGs in 108 patients who had undergone DC for severe head injury. In that study 11 patients had SDG collections ipsilateral and 9 had hygromas contralateral to the side of the DC; 3 of the 23 SDG collections were interhemispheric. Contralateral hygromas have also been documented in case reports by Kilincer et al.³⁴ and Su et al.⁶⁶ In the present study we saw that SDGs developed in 39 (57.4%) of 68 patients who had survived > 1 month after traumatic brain injury and DC.¹⁻³

Pathogenesis and Evolution of SDG

Traumatic Shearing Stress. Detailed electron microscopy studies by Haines et al.²⁵ and Schachenmayr

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and Friede⁶³ have indicated that there is no dead space between the dura and arachnoid layers. The interface between the dura mater and subarachnoid space is made of an interface layer composed of an arachnoid barrier layer and a dural border cell layer (Fig. 5 upper). Whereas a significant number of collagen fibers bond the cellular structure of periosteal and meningeal dura, the dural border cell layer contains very little collagen, and instead, cellular elements are loosely held together by an amorphous material prone to disruption by physical stress.^{25,63} Protecting the subarachnoid space and its trabeculae is the arachnoid barrier cell layer in which the cellular elements are tightly packed together by desmosomes, tight junctions, and gap junctions.^{25,63} Shearing stress generated by kinetic energy can tear arachnoid barrier cells and damage the integrity of the arachnoid-dura interface layer.²⁵ In our study patients who had been in MVAs were significantly more prone to SDGs ($p < 0.0007$) than were those who had fallen. It is quite likely that the former patients had a significantly more damaged arachnoid-dura interface layer during sudden exposure to a highly dynamic kinetic energy. Supporting this notion are the reported cases of SDGs contralateral to the side of the DC.^{66,75,76} In our series of 39 cases with SDGs, 3 hygromas were contralateral to the side of DC (Fig. 2).

Iatrogenic Surgical Disruption of the Arachnoid-Dura Interface. Another mechanism contributing to the egress of CSF into the subdural space and the formation of an SDG in the context of a DC is iatrogenic damage to the arachnoid-dura interface layer by the neurosurgeon during expansive duraplasty (Fig. 5 lower). Up to 90% of hygromas in our study were ipsilateral to the side of craniectomy. This finding is contrary to the laterality of SDGs in closed-head injury, which is almost equally distributed on the right and left sides. Koizumi et al.³⁶ and St John and Dila⁶⁴ have documented 63 cases of SDGs: 32 bilateral, 12 on the right side, and 19 on the left side.

Evolution of an SDG. The accumulation of CSF in the subdural space in closed-head injury, as reported by Koizumi et al.,³⁷ peaks at 20–30 days. The evolution of an SDG following DC has never been systematically evaluated. In our analysis an SDG could be seen even on the 2nd postoperative day (Fig. 4). When studied in the aggregate, the fluid collections increased in size for up to 4 weeks post-DC and gradually tapered, but lingered for up to 4 months after surgery (Fig. 1). Once in the subdural space, an SDG may exit the suture line of an expansive duraplasty and collect underneath the skin (Fig. 6).

Hydrocephalus. An added complexity to the pathogenesis of these hygromas includes the appearance of hydrocephalus after the complete disappearance of SDGs in 3 patients, pointing to possible difficulties with the dynamics of CSF circulation in patients with DC (Fig. 3). In our study hydrocephalus and the need for surgical CSF diversion was noted at Weeks 5–8, 17, and 22 after DC in 6 patients.

Hemorrhagic Conversion. Subdural hygroma fluid noted in severe head injury and DC is usually xanthochromic, contains a varied number of red blood cells,

and is high in protein.^{15,39,40,61,62,65,71,73} Hygroma fluid aspirated in 5 patients at the Shock Trauma Center contained high numbers of red blood cells and high protein levels (Table 2). Evidence indicates that this high-protein, highly xanthochromic fluid induces an inflammatory response in the subdural space, predisposing to neomembrane formation with fragile and leaky capillaries.^{21,61,62} The maturation of a hygroma neomembrane, containing fragile and leaky capillaries superimposed on stretched parasagittal veins,⁵⁵ may make hygromas susceptible to internal bleeding and foster the conversion of a benign SDG into a chronic SDH.^{19,21,37,39,43,48,53,56,57} Between 6 and 32% of SDGs convert into chronic SDHs.²¹ In the Liu et al. series⁴³ conversion happened from Days 22–100 after traumatic brain injury. In the Ohno et al. study⁵³ 20 of 43 patients with hygromas eventually showed evidence of bleeding inside the collection. In the Koizumi et al. series³⁷ hygromas in 4 patients converted to SDHs on Days 50, 70, 71, and 72 after their trauma. The hygromas in 3 patients in our study converted into chronic SDHs on Days 55, 57, and 57 post-DC (Fig. 4).

Management and Outcome

The visualization of SDGs on imaging studies does not suggest immediate intervention. Contralateral SDGs are more likely to induce a global decline in cognition and consciousness. Yang et al.⁷⁵ have reported on 11 patients with contralateral SDGs among 169 who had undergone DC; the hygroma spontaneously resolved in 7 patients and had to be drained in 4. Kilincer et al.³⁴ have described a 56-year-old man who had undergone a DC for malignant stroke and in whom a contralateral SDG later developed. The SDG in this patient was evacuated first by drainage and subsequently by subdural-peritoneal shunting. Similarly, Su et al.⁶⁶ have described a 63-year-old man with a posttraumatic evacuated mass who had undergone a DC and whose level of consciousness globally declined due to a contralateral SDG and significant shift of the midline structures. This patient responded well to simple drainage of the hygroma and was discharged symptom free 1 week later. There seems to be a tendency for contralateral SDGs to cause more symptoms.^{34,66,75} In our study 4 patients underwent drainage of the SDG. Three (8%) of 39 patients with hygromas had evidence of mass effect, that is, a shift in the midline structures > 5 mm: 1 of 36 patients with an ipsilateral and 2 of 3 patients with contralateral SDGs. All 3 patients needed subdural drains to reverse the existing shift (1 bur hole, 1 twist drill, and 1 percutaneous insertion of a draining system; Fig. 2). Two of 3 patients whose images had demonstrated a shift of midline structures (5.9 and 6.8 mm) had unchanged GCS scores following drainage of the subdural fluid: GCS Score 8 (Motor Score 3) in 1 patient and GCS Score 14 in the other. In 2 patients with clinical neurological worsening, the GCS score improved after drainage of the SDGs. One patient had a sudden decrease in his GCS motor score (from 6 to 5), which improved after the insertion of a percutaneous drain for an ipsilateral hygroma. Dysautonomic changes in another patient's vital signs occurred (heart rate increased to 115 bpm and temperature increased to 102°F), and he became obtunded. Computed tomography studies in this patient

revealed a 20.2-mm shift due to a contralateral hygroma. This patient underwent bur hole evacuation of this SDG twice. His neurological status improved immediately after surgery (Fig. 2). The overall prognosis for an SDG was better in patients with diffuse injury than in patients with an evacuated mass, which may be simply a reflection of the fact that the prognosis of DC in our 2 published series was better in patients with diffuse injuries than in those with evacuated masses and possibly had nothing to do with the presence or absence of an SDG.^{2,3}

Conclusions

An SDG developed in almost 57% of the patients who had undergone DC and survived the 1st month of hospitalization.^{2,3} Although 3 patients harbored a contralateral SDG, 36 (92%) of 39 patients had an SDG ipsilateral to the side of DC.

Based on electron microscopy studies,^{25,63} necropsy findings,^{15,49} and the entrance of contrast material into the hygroma cavity after contrast cisternography,⁴⁶ it is hypothesized that injury to the arachnoid-dura interface layer is the central pathology in SDGs. Spillage of the CSF into the subdural space peaked at 3–4 weeks after DC, and this pathological process gradually ceased from 14–17 weeks postoperatively. There is evidence that the proximity of the hygroma fluid with the dura upregulates excitatory cytokine synthesis and neomembrane formation. It is likely that expansion of the hygroma fluid will put undue stress on the walls of parasagittal veins, resulting in fresh bleeding into the hygroma cavity.⁵⁵ In addition, bleeding from the neomembrane capillaries may contribute to the possible conversion of a benign-looking hygroma into a chronic SDH.

In our experience, high dynamic accidents and diffuse traumatic brain injuries were risk factors for an SDG following DC.

The majority of hygromas—regardless of their size or internal bleeding—gradually disappeared without surgical management. Contralateral hygromas needed more aggressive treatment because of their tendency to cause midline shift. A variety of surgical management strategies have been used for the evacuation of SDGs including bur hole evacuation, bedside subdural drains, and craniotomy.^{37,40,64,65,75,76}

Disclaimer

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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