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Acute autologous bone flap infection after cranioplasty for postinjury decompressive craniectomy

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ABSTRACT

Background: Acute bone flap infection is a devastating complication after cranioplasty for postinjury decompressive craniectomy. We aim to identify the risk factors of autologous bone flap infection.
Methods: We enrolled 151 patients undergoing 153 cranioplasties in the 4-year retrospective study. Autologous bones stored at −75 °C were used in the cranioplasties. Acute bone flap infection was defined as the onset of infection ≤14 days after cranioplasty. The epidemiological data of patients and details of the cranioplasty procedure were recorded.
Results: Acute bone flap infection was identified in five of the 153 cranioplasties, accounting for 3.3% of all episodes. Three of the 5 infected patients and five of 143 uninfected patients presented with dysfunction of subgaleal drainage comparatively, which was significantly different ($p = 0.001$). Statistical analysis of the cranioplasty procedures and subsequent results of the two patient groups revealed the following significant findings: the duration of operation ($p = 0.03$) and the length of hospital stay after cranioplasty ($p < 0.001$).
Conclusions: Dysfunction of subgaleal drainage and long operative duration of cranioplasty are risk factors of acute autologous bone flap infection. Regarding the prolonged hospital stay in complicated patients, better surgical techniques should be implemented in order to eliminate the risks of infection.

Introduction

Decompressive craniectomy is widely performed to treat severe traumatic brain injury (TBI) with refractory intracranial hypertension. The removed cranial bone flaps are traditionally preserved in the deep freezers or in subcutaneous tissues for delayed cranioplasties.[1–4] Autologous bone flaps are preferred over newly developed reconstructive materials, because the former are more convenient, less costly, offer excellent fit, and permit bone growth. However, the use of cryopreserved bone flaps is reported to have a high infection rate of up to 25.9%.[5] A few risk factors have been identified, such as the prolonged bone preservation period and whether or not the skull is autoclaved before replacement, but the results of some studies are in opposition with these findings.[5–7] Therefore, the risk factors of post-surgical infection remain unclear. In this study, we retrospectively collected patients undergoing cranioplasty and analysed clinical features, operative details, and neuro-imaging findings to determine possible factors that are predictive of autologous bone flap infection.

Materials and methods

Patient selection

From January of 2005 to December of 2008, 320 patients underwent decompressive craniectomy for TBI at Kaohsiung Chang Gung Memorial Hospital, the medical center in southern Taiwan. Of these, 172 patients returned to the hospital for cranioplasty. The following patients were excluded: (1) patients who underwent vault reconstruction using alloplastic substitutes; (2) those with active systemic infection or local scalp infection; (3) those who had already received therapeutic antibiotics for any cause. A total of 151 patients undergoing 153 cranioplasties were enrolled for the analysis. The patients' charts were reviewed retrospectively after approval by the Institutional Review Board of Chang Gung Memorial Hospital.

Craniectomy and cranioplasty techniques

The patients with TBI underwent standard hemicraniectomy covering the frontotemporoparietal region. Following removal, the bone flaps were wrapped using two layers each of sterile plastic coverage and waterproof fabrics. The flaps were then placed into 2 plastic bags, transferred to the bone bank at our institute within 2 h, and stored in a deep freezer at −75 °C for periods ranging from 13 days to 245 days.

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Cranioplasty was performed if the presence of slack brain and medical recovery allowed to reconstruct. The bags containing the bone flaps were retrieved from the freezer immediately prior to the surgery. The bone flap was washed in sterilised saline solution containing chloramphenicol (1 g/500 ml). After the wound was reopened, the flap was fixed in its original position by using wires or titanium plates with screws. Then, Hemovac closed-suction drain was placed in the subgaleal space and tunnelled under the scalp to an exit point off the incision line. The volume and contents of drainage were recorded every 8 h by the nursing staff. Dysfunction of subgaleal drainage was identified on the basis of one the following clinical evidences: (1) the subgaleal drain was obstructed immediately following the procedure; (2) the volume of subgaleal drains was less than that of most cases (95%) in the first 8 h after cranioplasty. Post-operative single cefazolin with or without gentamicin was intravenously administered as prophylaxis.

Diagnosis and management of acute bone flap infection

The criteria for the diagnosis of acute bone flap infection are as follows: (1) the onset of infection is less than 14 days after cranioplasty, (2) the accumulation of pus or infected fluid in the subgaleal layer with or without involvement of epidural and subdural spaces, and (3) extensive infection necessitating the removal of the bone flap. Some patients have evidence of superficial wound infection and turbid discharge. Computed tomography (CT) scans usually present subgaleal fluid with perilesional enhancement. When acute bone flap infection is highly suspected, surgical debridement is carried out. Abscess cultures and swab cultures from skull flaps are performed in operation rooms. Empiric antibiotics are prescribed before or after the debridement and adjusted according to results of the cultures.

Clinical data collection

Epidemiologic data was documented, including prior scalp wound or central nervous system (CNS) infection after craniectomy, time interval between craniectomy and cranioplasty, and length of hospital stay after cranioplasty. "Multiple operations" was defined as 2 or more operations in which incisions were made along the wound of craniectomy prior to cranioplasty. Previous CT scans were reviewed for fractures crossing paranasal sinuses or mastoid air cells. After discharge, all the patients were followed up at the outpatient department.

Statistical analysis

All statistical analyses were conducted using SPSS version 12.0 (SPSS Inc., Chicago, IL). Continuous variables were assessed using the Student's *t*-test or Mann–Whitney *U*-test. Categorical variables were analysed using the chi-square test or Fisher's exact test. A *p*-value of 0.05 or less was considered statistically significant.

Results

The 151 patients receiving 153 cranioplasties included 108 male and 43 female subjects with a mean age of 41.2 (SD 17.8) years. Acute autologous bone flap infection occurred in 5 of the 153 cranioplasties. The incidence of infection was 3.3%. The mean time interval from cranioplasty to the onset of bone flap infection was 10.4 days (range, 4–13 days). The microorganisms responsible for infection in 4 of the 5 patients were *Staphylococcus aureus*, *Serratia marcescens*, *Citrobacter diversus*, and *Staphylococcus epidermidis*. The other one had prior administration of empirical antibiotics and the pus culture did not exhibit growth. The characteristics of 5 patients with acute bone flap infection are summarised in Table 1.

Comparisons of the baseline clinical features between 153 cranioplasties with or without acute bone flap infection are listed in Table 2. Differences in the gender ($p = 0.14$) and mean age ($p = 0.68$) were not statistically significant. There were no differences in the mechanisms of trauma and underlying diseases between the 2 groups. Prior CT scans for traumatic insults revealed no significant intergroup differences in the presence of fractures crossing paranasal sinuses ($p = 1.00$) or mastoid air cells ($p = 1.00$). The previous complications of decompressive craniectomy in these 2 groups did not show statistically significant difference in surgical wound infection ($p = 1.00$) or CNS infection ($p = 1.00$). Further, no significant difference was observed in the presence of multiple operations before cranioplasty ($p = 0.07$). The mean time interval between craniectomy and cranioplasty was 78.2 (SD 45.8) days in non-infected patients and 85.6 (SD 67.8) days in infected patients ($p = 0.70$).

The characteristics of cranioplasty procedures in these 2 groups are compared in Table 3. The mean duration of cranioplasty was 3.7 (SD 1.3) and 5.0 (SD 1.0) hours in the non-infected and infected groups, respectively ($p = 0.03$). The mean blood loss in the cranioplasty procedure was 313.1 (SD 321.2) ml in the non-infected group and 290.0 (SD 185.1) ml in the infected group ($p = 0.87$). The usage of prophylactic antibiotics after operation was divided into monotherapy (cefazolin) or dual therapy (cefazolin and gentamicin), and there was no statistical difference ($p = 1.00$). The mean length of prophylactic administration did not show statistically intergroup difference ($p = 0.85$). Amongst the 153 cranioplasties, dysfunction of subgaleal drains was found in 5 of the 143 non-infected patients and 3 of the 5 infected patients. The incidence of dysfunction of subgaleal drains was 3.5% and 60.0% in the non-infected and infected patients, respectively ($p = 0.001$). The mean length of the indwelling drain was 3.2 (SD 1.4) days and 2.8 (SD 0.8) days in the non-infected and infected groups, respectively ($p = 0.49$). CSF drainage from the subgaleal drain was recorded in 11 of the 148 cranioplasties without subsequent acute bone flap infection and in 1 of the 5 cranioplasties with subsequent infection ($p = 0.34$).

The mean hospital stay after cranioplasty was 10.3 (SD 9.1) days in the non-infected group and 116.8 (SD 110.3) days in the infected group ($p < 0.001$).

Table 1
Summary of 5 patients with acute autologous bone flap infection after cranioplasty.

Case No.	Gender, age (years)	Interval of craniectomy-cranioplasty (days)	Operative duration of cranioplasty (h)	Dysfunction of subgaleal drain	Time to acute infection (days)	Findings	Infecting organism
1	M, 68	39	6	Yes	11	Subgaleal abscess	*Serratia marcescens*
2	F, 57	195	6	Yes	13	Subgaleal abscess, intracerebral abscess	*Citrobacter diversus*
3	F, 17	44	4	No	13	Subgaleal abscess, wound dehiscence	*S. aureus*
4	F, 28	41	5	Yes	4	Subgaleal abscess	No growth in culture
5	M, 20	109	4	No	11	Subgaleal abscess	*S. epidermidis*