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Delayed Infection Following Cranioplasty
- Review of 4 Cases -

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Currently, the accepted indications for cranioplasty are for cosmetic considerations and protection of intracranial structures. The complications of cranioplasty include delayed infection, subgaleal fluid accumulation, fracture of resin plate, and resorption of preserved autografts. The most serious complication is delayed infection. We report on four cases with delayed infection following craioplasty with discussion of possible mechanisms.

Key Words: Cranioplasty · Complication · Infection

INTRODUCTION

External decompression can be an effective treatment for acute intracranial hypertension, but the skull defect must eventually be repaired. Protection of the brain and cosmetic considerations are two important indications for cranioplasty. The complications of cranioplasty include delayed infection, subgaleal fluid accumulation, fracture of resin plate, and resorption of preserved autografts. The most serious complication of cranioplasty is delayed infection[1,11,12]. The incidence of delayed infection has been reported to be 4.5%[12]. This report covers 8 years during which 59 cranioplasties were performed at our institutions and four patients developed delayed infections. The objective of this report was to identify risk factors for delayed infection following cranioplasty.

CASE SUMMARY

There were 3 patients with methyl methacrylate (MMC) and one with autogenous bone flap. The clinical features, cause of skull defect, systemic and local signs, organisms cultured, and outcomes are shown in Table 1.

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1. Causes of skull defect

The primary diseases were severe brain contusions with depressed skull fractures in three patients and intracerebral hemorrhage due to arteriovenous malformation in one patient, and all of them underwent decompressive craniectomy. Cranioplasties were performed from 15 to 124 days after primary operation.

2. Interval between cranioplasty and delayed infection

The interval between cranioplasty and onset of delayed infection ranged from 1 to 12 months (average 5.25 months) (Table 1).

3. Previous infection

One of the four delayed-infection patients had previously had an infected craniectomy. Twelve months after the craniectomy, he underwent a cranioplasty with methyl methacrylate. Two months after cranioplasty, he had wound dehiscence with a focal abscess(Fig. 1). Organisms identified were Methicillin-Resistance Staphylococcus aureus (MRSA). Despite massive systemic antibiotics therapy, systemic and local signs were aggravated. Operative debridement was done(Fig. 2).

4. Local signs

Swelling and tenderness of the scalp flap were observed in

Table 1. Clinical summary of Delayed infection after cranioplasty

Case No.	Age/ Sex	Interval I (mon)	Cause of skull defect	Interval II (mon)	Material	Local sign	WBC	ESR	CRP	Organism	Treatment
1	F/24	15days	FCD	4	MMC	+/−	15400	51	5.19	MRSA	craniectomy
2	F/16	3	AVM	4	autograft	+/+	5200	45	8	none	craniectomy
3	M/3	1	FCD	112	MMC	−/+	16600	48	12.80	MRSA	craniectomy
4	M/546	3	FCD		MMC	+/−	18900	52	9.21	MRSA	craniectomy

*Interval I = between external decompression and cranioplasty, Interval II = between cranioplasty and infected bone flap removal, FCD = fracture compound depression, AVM = arteriovenous malformation, MMC = methyl metacrylate, Local sign = fistular and pus discharge/swelling and/or tenderness

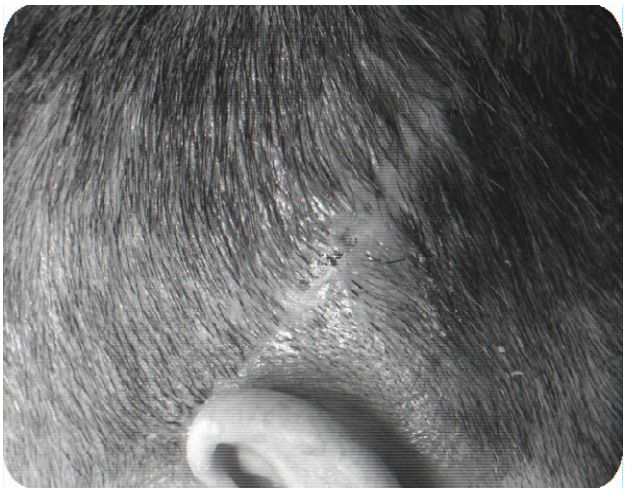

Fig. 1. A photograph showing wound erosion and serious discharge.

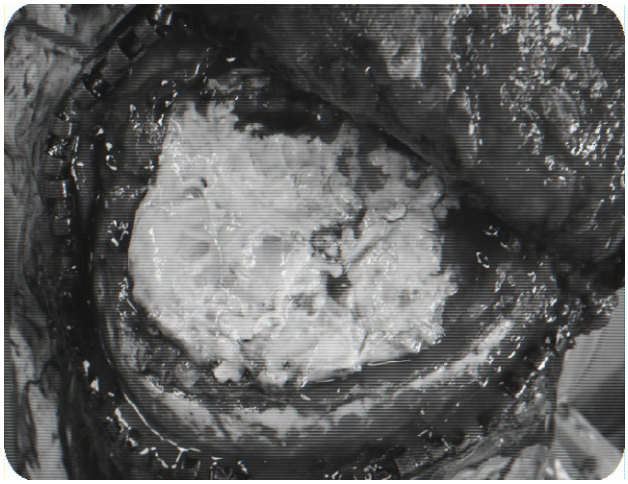

Fig. 2. Intraoperative view showing yellowish thick mass occupying epidural space.

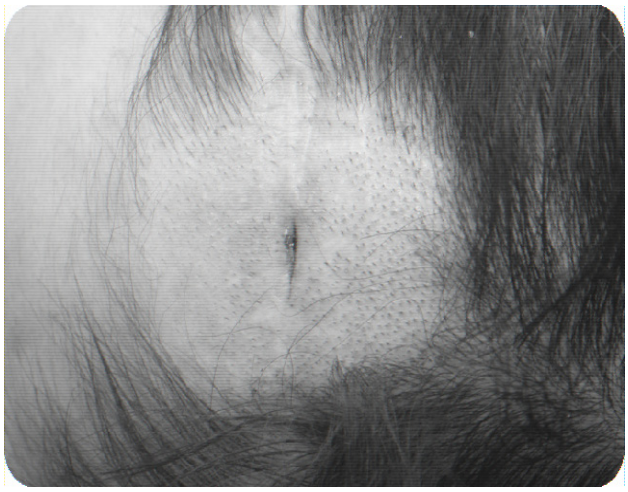

Fig. 3. A photograph demonstrating stitch stump with localized abscess.

two patients. Wound dehiscence and discharge were in three. These signs may be developed from infected galeal suture and hair folliculitis when the patients had scratched their wound (Fig. 3).

5. Systemic signs

Body temperature was normal in two cases, but elevated in the others. The C-reactive protein titer was over 5 and the ESR was over 45, in all cases. The WBC count was within normal limits (fewer than 7,000/mm^3) in one case and was elevated in the other three (over than 10,000/mm^3).

6. Treatment methods

Standard operative debridement consisted of reopening the

wound and removing of the bone flap. All visible suture material and hemostatic agents were removed. The purulent material was cultured and analyzed for anaerobic and aerobic organisms, as well as for sensitivity to various antibiotics. Necrotic and purulent debris were removed by mechanical debridement and copious irrigation with saline mixed with antibiotics. Bone edges were routinely drilled. Hemostatic agents such as Gelfoam were avoided as much as possible to reduce the amount of foreign material remaining in the wound. Patients were treated with broad-spectrum intravenous antibiotic agents until determination of the intraoperative tissue cultures and antibiotic sensitivities. A full course of systemic antibiotic agents was administered according to the recommendations of an infectious disease consultant in each case. In all cases this consisted of 2 week intravenous therapy followed by 2 to 4 weeks of oral therapy.

DISCUSSION

Postoperative wound infection was defined as purulent wound drainage, bacterial meningitis, epidural and subdural empyema, osteomyelitis, multiple stitch abscesses, or wound cellulitis[8]. Potentially devastating effects of postoperative wound infection in the central nervous system have inspired continuing interest in a better understanding of the factors leading to postoperative wound infection. Among neurosurgical operations, postoperative infections after secondary operation like cranioplasty are more stressful conditions to neurosurgeon. Since 1990, postcraniectomy infection rates have been reported from less than 1% to as high as 11%[2,3,5] and the incidence of delayed infection after cranioplasty has been reported to be 4.5%[12]. Potential risk factors for infection after cranioplasty have been identified, but relatively few attempts to verify the importance of each factor have appeared in the literatures.

Many factors affect the delayed infection after cranplasty. First, previous infection due to penetrating open head injury appears to be an important risk factor for subsequent infection. Previous studies reported that the infection rate in patients with previously infected craniotomy was high[12]. Recently, Kim et al.[7] reported cranioplasty should be repaired as soon as possible, because early cranioplasty can lower the infection rate. In our case, cranioplasty was performed 15 day after surgical debridement of penetrating frontal head injury at other institute. Four months after the cranioplasty, an infection as detected that involved a purulent discharge originating in the frontal sinus. Many investigators have reported the benefits of delaying cranioplasty. Yamaura et al.[13] noted that all of their infected patients had undergone cranioplasty within 3 months of external decompression. In case of infection after the initial surgery, the importance of delaying cranioplasty is even more obvious. Rish et al.[12] found that when cranioplasty was performed within 1 year of contamination of the initial wound, the incidence of infection was formidably high(56%). Above all, penetrating injuries involved the frontal sinus and CSF leakage are important risk factors for delayed infection. Despite adequate administration of antibiotics, the incidence of delayed infection might not be lowered in penetrating injuries involving the frontal sinus.

Second, introduction of foreign body like MMC during the drilling of bone is also risk factors for delayed infection. Bacteria attached to the surface of a foreign body can remain alive and cause recurrent infection[6]. Mollian et al.[8] reported that place ment of foreign bodies may increase the risk of postoperative infection. Park et al.[9] reported that cranioplasties using refrigerated autogenous bone flaps showed shorter operative times, better cosmetic results, and lower rates of complications than those using MMC. To be suitable for cranioplasty, the materials must meet several criteria. It must be biologically inert, nonreabsorbable, nonantigenic, relative inexpensive, readily available, radiolucent, and sterilizable[1,10]. Physically, it should be lightweight and strong to withstand trauma[1,10]. Popular current cranioplasty materials include autogenous bone, metals (tantalum, stainless steel, aluminum), and plastics (methyl methacrylate, polyethylene, silastic). Considering all of the above criteria, autogenous bone is probably the best material presently available[1,11]. In our study, cranioplasties using MMC were associated with higher rates of delayed infection than were those using autogeneous bone. Because the foreign bodies such as bone fragements and artificial materials appear to be an causative factors for subsequent infection[10], adequate and meticulous irrigation is an important factor to prevent delayed infection due to introduction of foreign body during the drilling.

Third, bacteria newly introduced from the environment (seborrheic skin and scalp) may cause a first infection. Because scar

tissue is less resistant to infection, secondary infection more likely arises from bacteria that have been dominant within the wound for a long period[8]. Jeffery et al.[5] reported that the majority of neurosurgical wound infection patients were infected by skin organisms, mostly by one of the staphylococci or by Propionibacterium acnes. In our cases, three patients had staphylococci infections.

Finally, wound infection was associated with a purulent discharge from fistula, which developed when the galeal suture became a focus of infection after the patients had scratched their wounds[6]. The stitch stump represents a substantial risk factor for postoperative delayed infection. When a stitch abscess is identified, it seems to be logical to recommend aggressive managements to treat it.

Traditionally, the management of delayed infection following cranioplasty has consisted of operative debridement and removal of devitalized bone flaps[4]. Within the limits of possibility, preservation of bone flap has the advantange of treating infection, but, we suggest that aggressive operative debridement with removal of the bone flap and antibiotic irrigation to remove all dead tissue, debris, suture, and foreign materials is desirable.

CONCLUSION

We suggest that thinned scalp due to multiple operations with seborrheic dermatitis and/or folliculitis eczematosa may be a the risk factor for delayed infection following cranioplasty. No matter how subtle the systemic signs, late infection warrants surgical debridement and antibiotic chemotherapy as soon as possible. Despite debridement and chemotherapy, it appears very difficult to completely prevent cases of delayed infection, but the incidence of bone flap removal can be minimized if the risk factors are kept in mind.

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