두피의 고압전기화상 환자에서의 두개골 만성골수염: 증례보고

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Chronic Osteomyelitis of Cranial Bones in a Patient with High-Voltage Electrical Burn on the Scalp: A Case Report

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High-voltage electrical burn injuries on the scalp often result in scalp and cranial bone necrosis. Repetitive debridements and rich-vascularized flap coverage of the cranium are required. However, despite successful flap coverage, chronic osteomyelitis of cranial bones may occur. Treatment of chronic osteomyelitis of cranial bones is surgical debridement of the necrotic bone with re-coverage by a well-vascularized flap. The latissimus dorsi musculocutaneous flap is suitable not only for coverage of the cranium after the burn injury, but also for treatment of chronic osteomyelitis of the skull. (*J Korean Burn Soc* 2021;24:53-59)

Key Words: Electrical burn, Scalp injuries, Free flap, Chronic osteomyelitis

INTRODUCTION

Electrical burn injuries occur more frequently in developing countries than in other countries. The incidence of electrical burn injuries is less than 5% of admissions to major burn centers in the United States. The mortality rate is 3%~15%, with approximately 1000 deaths per year in the United States [1].

Burn injuries caused by electrical currents are divided into flash burns, flame burns, contact burns, or a combination [2]. Traditionally, electrical injuries have been divided into those caused by low-voltage, (less than 1,000 V) and high-voltage, (greater than 1,000 V) [2]. The extent of damage caused by high-voltage electrical burns depends on the intensity of the heat generated and the duration

of the exposure [2].

As the electrical current flows within the body, the electrical energy is converted to heat energy, which causes thermal damage to the tissues [2]. This is because, although the duration of electrical contact is only a fraction of a second or a few seconds, extremely high temperatures are generated [3]. Intravascular thermal damage causes thrombosis, which results in progressive intravascular obstruction, leading to tissue necrosis.

The scalp is an easily accessible part of the human body for high-tension electrical wires [2]. High-voltage electrical currents can often cause deep scalp and calvarial burns [1]. Progressive scalp necrosis causes scalp defects, resulting in cranial bone exposure that would require flap coverage. In addition, progressive necrosis occurs in one or both of the calvarial tables, which is difficult to treat. The resulting damage is not caused by bone destruction but by thrombosis of its blood vessels [3,4]. The treatment of scalp and skull electrical burn injuries requires debridement of necrotic tissue and coverage of the skull. However, debridement of the injured skull is not readily performed since the protection of the brain needs to be

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Fig. 1. (A) On the day of the burn injury (PBD 0), the entry point of the electrical current in the right side of the parietal scalp. Approximately 4×3-cm-sized discolored scalp with char was noted. PBD: postburn day. (B) On PBD 45, a 12×12-cm-sized scalp defect with skull bone necrosis was found. At the central portion of the exposed parietal skull, discolored necrotic cortex was noted. (C, D) On PBD 45, from the right side of the chest, an LD MC flap was harvested. The flap size was 25×20 cm, with a central 20×5-cm elliptical skin paddle. LD MC: latissimus dorsi musculocutaneous. (E) On POD 37 after LD free flap surgery, a small wound opening was noted at posterior flap margin. Through probe examination, a 15-cm long sinus tract was found, posterior to anterior direction. POD: postoperative day. (F, G) At 4, 6, and 12 months after burn injuries, rigid endoscopic examination and saline irrigation through the wound opening were performed repeatedly. Endoscopic examination revealed no definite sequestrum or cavity along the sinus tract. (H) On POD 19 months after free flap surgery (PBD 21.5 months), exploration of the sinus tract and craniectomy were done. The arrow indicated the location of necrotic cortex and medulla of cranial bones after craniectomy. The elevated LD muscle flap overlying the bone was re-closed without tension. (I, J) On POD 2 (I) and POD 18 months (J) after craniectomy. The wound healed well. There was no recurrence of the sinus wounds. Later, a debulking procedure and hair restoration surgery were planned.

considered. Coverage of the skull requires a rich vascularized flap. Despite successful flap coverage of the scalp defect, electrically injured cranial bone may become progressively necrotic, resulting in chronic osteomyelitis. In such a case, as in this case report, another operation for surgical debridement is required. The aim of this study is to understand treatment of the scalp defect and chronic osteomyelitis of the skull caused by high-voltage electrical burn injury.

CASE REPORT

A 37-year-old man was referred to our department because of a scalp wound caused by a high-voltage electrical burn injury. Four weeks before reference, the pa-

tient had sustained a 22,000-voltage electrical burn injury to his scalp. In the emergency room, the patient was drowsy and disoriented with respect to time, place, and person. Resuscitation was performed, and the patient recovered his orientation after three days. The entry point of the electrical current was on the right side of the parietal scalp. The exit points were on the right thumb and middle finger, right side of the face and neck, right shoulder and chest, right forearm, and right lower flank. At first, a 4×3-cm-sized discolored scalp wound was noted with visible charring at the parietal scalp (Fig. 1A).

On postburn day (PBD) 5, excision of eschar and a split-thickness skin graft (STSG) were performed on the right arm. On PBD 22, debridement and STSG were performed on the right side of the flank. The wounds of the

Fig. 1. Continued.

skin-grafted sites healed well. Other sites of the wound, except the scalp, were treated with burn dressings.

The scalp wound was approximately 4×3 cm in size on PBD 7 (Fig. 1A). The margin of the scalp wound was irregular, and a small amount of wound discharge was noted. However, after progressive occurrence of necrotic changes, the scalp wound had enlarged to 12×12 cm with cranial bone exposure at PBD 45 (Fig. 1B). From PBD 7, debridement of the necrotic scalp was performed several times over 5 weeks. The outer cortex of the cranial vault (the right side of the parietal bone) was exposed after PBD 10. It had been gradually enlarged owing to the progressive necrosis of the scalp. The outer cortex was discolored (dark brown), due to necrosis of the bone (Fig. 1B). Debridement of the discolored and necrotic outer cortex of the parietal cranial bone was also performed three times by electrical burring before the flap coverage. However, the inner cortex was preserved as it was regarded to be intact. Necrotic changes in the outer cortical bone appeared to stop at PBD 40, and healthy granulating tissue was noted at the margin of the scalp wound. Skull computed tomography (CT) revealed focal irregular areas of full-thickness bony erosions with associated overlying

soft tissue defects, involving the posterior aspect of the right parietal region at PBD 34.

Debridement of the cortical bone and coverage by the latissimus dorsi (LD) myocutaneous (MC) free flap and two STSGs were performed, under general anesthesia, on PBD 45 (Fig. 1C, 1D). The necrotic cortical bone was completely debrided. The inner cortex of the bone was intact and hence, was preserved. The LD muscle flap was 25×20 cm in size with a 20×5 cm central skin paddle, which was harvested from the right side of the chest wall (Fig. 1C, 1D) and transferred to the scalp. The thoracodorsal artery and vein were anastomosed to the right superficial temporal artery and superficial temporal vein. The raw surface of the LD muscle flap was covered by 7.5×12 cm and 7.5×9 cm STSGs, by meshed shield grafts. After the operation, the LD MC flap and skin grafts survived well. The scalp wounds healed well three weeks postoperatively. The LD donor site also healed well. And the patient was then discharged.

However, 37 days after the successful LD free flap surgery, a small opening was noted on the posterior site of the scalp wound accompanied by a small amount of discharge (Fig. 1E). No signs of acute inflammation were

Fig. 2. Enhanced brain CT and MRI at PBD 4 months (POD 2.5 months after LD MC free flap). A small sinus wound opening (the arrow tip) was noted at the posterior scalp. Suspicious of mild bone marrow edema and subtle bone enhancement without cortical destruction or periosteal reaction was found at right parietal skull. The early stage osteomyelitis was suspected.

CT = computed tomography, MRI = magnetic resonance imaging.

visible on the scalp or the flap. The size of the wound opening was 5 mm in diameter, and the amount of discharge along with some turbid material was approximately 2 cc. C-reactive protein (CRP) level and blood leukocyte count were within the normal range. The wound culture did not show any growth of bacteria, acid-fast bacteria, nontuberculous mycobacteria, or fungi. The brain magnetic resonance imaging (MRI) and skull CT and whole body bone scan showed signs of cranial bone osteomyelitis (OM) at the previous burn injury site (Fig. 2). Bone resorption of the outer cortex was noted, but no sequestrum was observed in skull CT and MRI (Fig. 2). According to these findings, chronic OM of the calvarial bones was suspected. Simple local wound care was performed, and intravenous antibiotics were administered initially to prepare for the operation for OM. However, the patient refused exploration, and the wound healed spontaneously after 2 weeks.

One month later, the wound recurred with a small opening and a small amount of serous discharge, similar to the previous wound. The wound culture showed growth of *Staphylococcus aureus*. Intravenous injection of vancomycin was administered for two weeks, but was stopped due to side effects. At four months PBD, the wound was examined using a probe in the operating room under local anesthesia, and rigid endoscopic exami-

Fig. 3. Non-enhanced brain CT at PBD 19 months (POD 17.5 months after LD MC free flap) showed extended low density on the right parietal skull with cortical destruction, which was compatible with the findings of chronic OM. OM = osteomyelitis.

nation through the wound opening was performed on the scalp wound. A sinus tract, approximately 15 cm long, was found in the posterior to anterior direction. A 2×3 cm undermined area was noted at the mid-portion of the sinus tract. The skull bone showed no necrotic bones or cavities along the sinus tract, with a healthy appearance (Fig. 1F). If sequestrums or infected sites had been found, surgical exploration of the scalp wound would have been done. Nevertheless, the endoscopic examination proved insufficient because of its rigidity. These episodes were repeated several times for 17.5 months after the flap surgery.

The wound recurred at 19 months PBD (postoperative day [POD] 17.5 months). At the bone lesion site, the thinnest portion of the calvarial inner table was 3-mm thick (Fig. 3). This time, surgical exploration of the scalp wound was performed by both plastic surgeons and neurosurgeons. The scalp flap (previous LD flap) was incised from the dorsal scalp wound opening to the ventral side of the scalp along the sinus tract. An approximately 15-cm-long zig-zag incision was made, and the flap was elevated from the parietal skull (Fig. 1H). The sinus tract was completely opened. Chronic bone inflammatory findings were noted at the central portion of the electrical burn wound site of the skull. A definite discolored and necrotic bone segment (sequestrum), 2×3 cm in size, was observed. Another 2×3-cm-sized discolored area on the cortical bone was noted near the sequestrum. Approximately 3 cc of serous discharge was noted in the skull cavity. Fluid removal and sequestrectomy were performed.

Fig. 4. Enhanced brain CT and MRI at POD 2 months after craniectomy (PBD 21 months). The cortical thinning was aggravated but no further thinning in inner cortex.

The discolored lesions in the outer cortex were also debrided. Further debridement (craniectomy) of suspicious necrotic diploic (medulla) bone and antibiotic irrigation were performed (Fig. 1H). However, the inner cortical bone was viable and preserved. The elevated overlying muscle flap was replaced and closed with a Hemovac suction drain (Fig. 1I). There was no flap tension or skull exposure after closing the incised wound. The drain was removed on POD 7, and the wound healed well 2 weeks later. The patient was discharged. After 18 months, no wound recurrence or inflammatory signs on the scalp were observed (Fig. 1J).

At 14 months after the craniectomy (sequestrectomy), the radiological findings of chronic OM had almost subsided. The inner cortex was intact and showed neither thinning nor progressive changes at the burn injury site. The thickness of the thinnest portion of the previous OM lesion site was well maintained with 3 mm thickness as revealed in skull CT (Figs. 3, 4, 5). However, some suspicious chronic OM findings in the skull were still observed, despite no clinical symptoms or signs of chronic OM. The infection specialist recommended observation and regular follow-up instead of using antibiotics or other treatments. The scalp and flap were good without wound recurrence for 20 months after exploration (craniectomy) of the scalp wound. Hair restoration surgery or toupee wearing on the scalp would be later recommended to the patient after the debulking procedure of the flap.

Fig. 5. Enhanced skull base bone CT and brain MRI at POD 1 year after craniectomy (PBD 33 months). There was no interval change in focal low density in the right parietal skull. No further thinning of the inner cortex was noted. The finding of chronic OM was nearly subsided.

DISCUSSION

The severity of the injury caused by high-voltage electrical burn depends on the voltage, duration, location, and resistance of tissues to the current [2]. According to Joule's law, heat generated is defined as amperage-squared times resistance [5]. The resistance capacity of tissues to electric current in increasing order is nerves, vessels, muscles, skin, tendons, fat, and bone [6]. The bone is the tissue with greater electrical resistance and heat production than other tissues [2,4]. The large surface and vascular diploe (medulla) in the skull decrease electrical resistance, allowing a large amount of electrical current to flow [4]. As a result, necrosis of one or both of the calvarial tables may occur.

The generally accepted treatment for full-thickness scalp burns with an underlying skull injury, is skull preservation with satisfactory soft tissue coverage [6]. Adequate surgical debridement, removal of dead bone mainly in the outer cortex, and selection of appropriate flaps are the mainstay of management [2]. According to this strategy, complete removal of the necrotic tissue is necessary before coverage of the scalp and skull defects. However, debridement of the cranial bone is different from that of other bones because the skull is relatively thinner than most other bones. Aggressive debridement of the cranial bone can cause exposure of the dura mater or cerebrum, requiring more complex surgery [6].

Bone debridement is performed until fine punctate bleeding points are visible [3]. However, accurately as-

sessing the degree of bone damage caused by electrical injury is challenging because the distinction between dead bone and living bone is not definite [3,6]. If bone viability is suspected, more observation is needed instead of aggressive debridement [3]. Consequently, several instances of serial debridement of the necrotic cranial bone are advisable [3,6]. Some authors performed only superficial curettage of the outer table [6] with serial debridement and delayed reconstruction [3].

There are many reports about treatments of chronic osteomyelitis of the cranium. Previously, the classic management of deep electrical burns to the scalp and skull was conservative treatment, involving leaving devitalized bone exposed until sequestration was complete, before performing reconstruction [4,6]. On the contrary to this, some studies support early debridement and closure of scalp burn injury wounds to avoid infection [3]. Another approach involving primary excision of the eschar and rotational scalp flap coverage has been used, without regard for the degree of bone devitalization [7].

Some authors recommend delayed bone debridement until the line of demarcation appears [3]. According to them, it takes approximately 7~10 days to develop a clear zone of demarcation. Serial debridement and delayed reconstruction are advised [3]. Consequently, free flap coverage should be performed at least 3 weeks after the injury. This method is similar to the method we have used. In our case, serial debridement of the scalp and skull bone was performed 45 days after the burn injury.

There are various types of surgery for treatment of scalp defects. Traditionally, for coverage of the exposed skull, trephination and STSG have been performed [2,3].

Usage of negative pressure wound therapy before STSG has increased lately. Nowadays, free flaps have been frequently used for coverage of exposed skull bones. For large-sized wounds of the skull, free flaps are preferable to local flaps. The soft tissue flap must be highly vascularized for the coverage of exposed bones, especially in electrical burns.

If flap coverage is performed successfully, the wound heals well, with no discharge or inflammatory signs in the scalp. In our case, despite a vascularized free muscle transfer covering the bone, chronic osteomyelitis developed in the skull, with subsequent bone resorption [6] (Figs. 3, 4). In a sense, chronic OM of the skull by high-voltage electrical burn on the scalp is inevitable, because complete and perfect debridement of the necrotic skull bone is practically impossible at the time of flap coverage, as previously described. Another study also reported that all patients suffered from calvarial osteomyelitis during treatments of high-voltage electrical burns on the scalp in 11 patients [1]. They reported that it was difficult to treat calvarial osteomyelitis due to a high relapse risk. They recommended timely surgical debridement of scalp and calvaria.

The treatment of chronic skull OM involves surgical debridement and coverage by a well vascularized flap. Complete debridement of necrotic tissue and bone with appropriate antibiotics is necessary [1]. If previous coverage was with a skin graft, flap coverage is desirable. If previous coverage was with a flap, surgical treatment is elevation of the former flap and removal of all necrotic bone and granulation tissue, and then returning of the flap [8], which is similar to our method. Full thickness

Fig. 6. (A) Bone scan on PBD 11. The early phase of osteonecrosis of the right upper posterior parietal skull is shown. (B) Bone scan at PBD 8 months, POD 6.5 months after LD MC free flap. More aggravated osteomyelitis findings were observed in the circular periphery of the right upper parietal skull. (C) Bone scan at POD 1 year after craniectomy (PBD, 31 months). Improvement of remaining osteomyelitis in the right parietal skull is shown.

skull defect may be treated by cranioplasty. If surgery is successful, the wound heals well, and the clinical signs of chronic OM will subside.

The LD MC flap was resistant to infection in this patient, even enduring chronic OM for up to 19 months [3]. In case of a thin flap, such as only a skin flap, the flap might be partially necrotic and perforated at the mid-portion of the flap by the chronic OM, which would be more difficult to treat. However, this LD MC flap was sufficiently thick, redundant, and well-vascularized; thus, it was resistant to infection by chronic OM. Consequently, no additional flap surgery was required after exploration (craniectomy) of chronic OM wounds.

In our case, 19 months after debridement (craniectomy) of the necrotic skull bone, there was no wound recurrence. The radiological findings of chronic OM were much improved, and skull CT showed no further thinning of the skull inner table (Fig. 6C) at the previously infected sites. Despite this, a long-term follow-up for recurrence is recommended. Some authors reported that electrical scalp burn wound recurred three years after wound healing by chronic OM of the skull in a child [8].

After free flap operation, the patient wanted debulking of the flap for esthetic shaping. However, from our experience, it is desirable that the debulking procedure of the bulky flap is delayed until there are no signs of chronic OM. Thereafter, a toupee or hair graft could be worn.

In conclusion, high-voltage electrical burn on the scalp is treated by repetitive surgical debridements and coverage by a rich vascularized flap. Despite successful flap

coverage, chronic OM of the skull may occur. The treatment of chronic OM is surgical debridement and well vascularized flap coverage. The LD muscle free flap is suitable not only for coverage of the skull, but also for the treatment of chronic OM of the skull.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

REFERENCES

1. Yudoyono F, Sutiono AB, Arifin MZ. High-voltage electrically head injury presenting underlying calvarial osteomyelitis: Single Indonesian tertiary hospital experience. *J Surg Surgical Res* 2016;2:10-3.
2. Jeyakumar P, Hussain AT, Ahamed AR. Reconstruction of extensive post-electric burn scalp defects with exposed bones: A study of 12 cases. *Ann Plast Surg* 2018;81:39-44.
3. Naik AA, Math M, Morab JV. Scalp reconstruction-post-electric burns. *Medica* 2019;8:34.
4. Worthen EF. Regeneration of the skull following a deep electrical burn. *Plast Reconstr Surg* 1971;48:1-4.
5. Grassner L, Bierschneider M, Strowitzki M, Grillhösl A. Different sequelae of electrical brain injury - MRI patterns. *Burns* 2017;43:e7-e10.
6. Rockwell WB, Bodily KD. Fate of free muscle transfer covering chronically infected burned skull. *J Burn Care Rehabil* 2001;22:288-91.
7. Luce EA, Hoopes JE. Electrical burn of the scalp and skull. Case report. *Plast Reconstr Surg* 1974;54:359-63.
8. Gümüş N, Coban YK, Reyhan M. Cranial bone sequestration 3 years after electrical burn. *Burns* 2006;32:780-2.