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Case report

Post-traumatic infection induced scalp necrosis – An unusual complication

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ABSTRACT

Introduction: Scalp wounds usually heal very well and necrosis of scalp is very rare due to its extensive vascularity. We present a case of scalp necrosis as an unusual complication of road traffic injury.

Case report: A 50-year old male with post-traumatic quadriplegia due to C5 fracture dislocation and scalp laceration presented to us. Patient developed scalp necrosis after 5 days. Wound cultures grew *Acinetobacter baumanii*.

Conclusion: Despite extensive vascularity, post-traumatic wounds with superadded infection can rarely lead to scalp necrosis. Thorough early wound debridement can help in preventing this rare but devastating complication.

1. Introduction

Scalp has a very rich vascular supply. Necrosis of scalp is a very rare condition due to this richness of vascularity. We present an unusual case of post-traumatic scalp necrosis.

2. Case report

A 50-year old male was admitted to our trauma centre with post-traumatic quadriplegia due to C5 fracture dislocation. The patient was initially treated at another hospital and came to us after 48 h of injury. Patient had a linear lacerated scalp wound involving right fronto-parietal area, without any associated fracture of the underlying bone. The wound was contaminated with dirt particles lying inside the wound. Patient had received intravenous methylprednisolone after injury for 24 h. On 5th day, patient started developing scalp discoloration which spread over the next 2 days to involve the entire right fronto-temporo-parietal area with a clear line of demarcation suggestive of scalp necrosis (Fig. 1). Ultrasound doppler revealed thrombosis of right superficial temporal artery with normal external carotid artery flow. *Acinetobacter baumanii* was grown from the wound.

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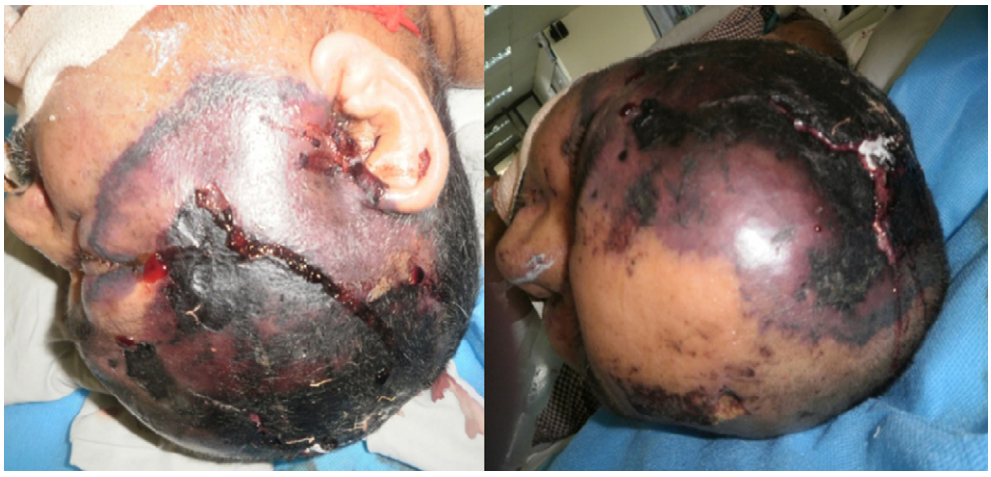

Fig. 1 – Photographs showing lacerated wound and well demarcated scalp necrosis.

3. Discussion

Scalp is a highly vascular structure. Due to this extensive vascularity, scalp wounds heal very well even in cases of avulsion injuries. Infection of scalp wounds is usually limited and easily controllable and scalp necrosis is very rare.

In the present case, scalp necrosis started 5 days after injury. At the time of presentation, there was no evidence of compromised vascularity of the scalp. Thus, direct injury to STA leading to scalp necrosis is unlikely. The possible mechanism of scalp necrosis is related to infection of the lacerated wound. There was a delay of ~48 h in the debridement of the wound after injury. Delay in the cleansing/debridement of these wounds leads to increased risk of infection. Steroid induced immunosuppression further predisposes to infection. Infection induced microvascular thrombosis, spreading in a retrograde fashion and leading to STA thrombosis can explain the development of necrosis despite the rich vascularity of the scalp.

Though, necrotizing fasciitis of scalp is a well known entity,[1] to the best of our knowledge, post-traumatic scalp necrosis has not been previously described in the literature. We report first such case of scalp necrosis occurring in a delayed manner after injury. Scalp necrosis, however, has been reported as a rare manifestation of giant cell arteritis[2] and as a complication of external carotid artery embolization for meningeal tumors.[3]

4. Conclusion

This case demonstrates that scalp lacerations which usually heal well, can develop serious complication. It also highlights the importance of early cleansing/debridement of the wound to prevent the development of this rare but devastating complication.

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