A Case of Postoperative (Pressure) Alopecia

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Postoperative (pressure) alopecia is the temporary or permanent loss of hair that occurs following a prolonged immobilization during and/or after general anesthesia and intubation. The cause is considered to be pressure-induced ischemia. Although not an uncommon condition, it is frequently overlooked as a minor complication relative to a causative surgical procedure. We report a 22-year-old woman who was presented with focal hair loss of two weeks' duration after a 9-hour operation for a left brachial plexus injury. Histopathologic examination showed that nearly all terminal follicles were in catagen phase with marked atrophic change of subcutaneous fat. (Ann Dermatol 12(3) 218~221, 2000).

Key Words : Postoperative alopecia, Pressure alopecia

Postoperative alopecia is a minor and self-limiting complication preceded by a prolonged period of unconsciousness, most commonly a surgical procedure under general anesthesia. Localized pressure-induced ischemia is the likely cause[1,2]. Since Abel originally described five cases of pressure alopecia in 1960, not a few have been reported including several cases in Korean literature[3,4]. In spite of its characteristic history and clinical features, it sometimes needs to be differentiated from other forms of focal hair loss of which management and prognosis may be different. In spite of its minor and self-limiting nature, this preventable complication may be disturbing to both patient and physician.

Herein we report a case of postoperative alopecia that showed clinically a close resemblance to alopecia areata and trichotillomania, but histopathologically characteristic features consistent with pressure alopecia.

Received January 3, 2000.
Accepted for publication June 7, 2000.
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* This paper was presented at the 51st annual meeting of the Korean Dermatological Association on October 12, 1999.

CASE REPORT

A 22-year-old woman was referred from the Department of Neurosurgery for the evaluation of focal hair loss of two weeks' duration. She had an operation 20 days previously for her traumatic left brachial plexus injury caused by an automobile accident. She was deliberately positioned to give the most exposure for left brachial plexus, with her head turned to the right during the operation. Total operation and anesthesia time were eight-and-a-half and eleven hours, respectively. No antiseptic solution or other medication touched the scalp. She initially developed an erythematous tender nodule in the right parietal scalp a few days after the operation, then followed by developing an ovoid patch of hair loss on that lesion. Her health was otherwise excellent. The lesion was a relatively well-demarcated 1.5×1.2cm-sized ovoid alopecic patch with some broken hairs. There were also vellus hairs on the periphery of the lesion with a central zone of dusky brown discoloration overlying the slightly indurated subcutaneous papule(Fig. 1). Hair pull test revealed increased plucking, and abnormal anagen hairs and a couple of broken hairs were observed on hair mount. A transverse section of the biopsy specimen showed that nearly all terminal follicles were in catagen phase and there was no evidence of follicular disruption or atrophy(Fig. 2). Subcuta-

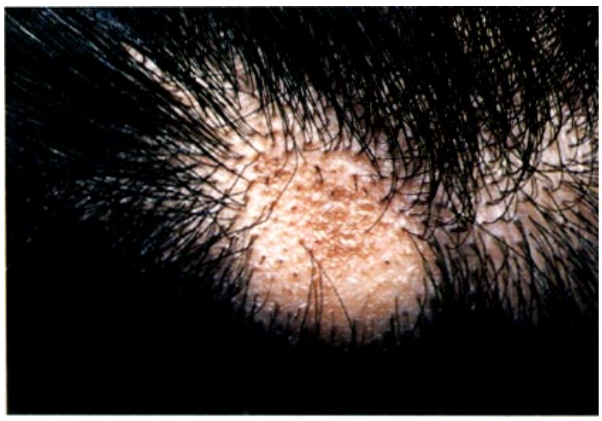

Fig. 1. An ovoid patch of hair loss with fragmented and vellus hairs, and with a central dusky brown discoloration.

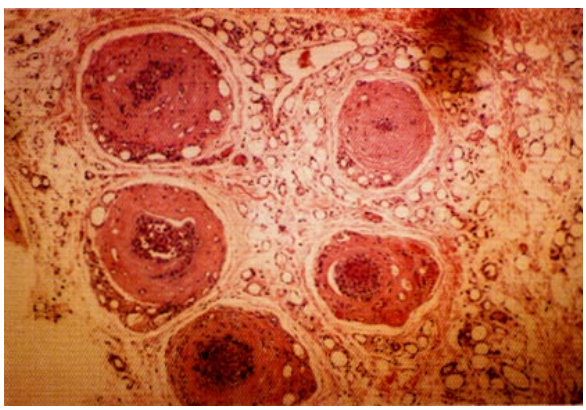

Fig. 3. Transverse section at the level of dermal/fat junction showing catagen follicles embeded in the atrophic subcutaneous fat(H & E, ×40).

neous fat atrophy were also prominent associated with infiltrate of foamy cells(Fig. 3). In a vertical section, mild inflammatory infiltrates were observed in the papillary and upper reticular dermis but it was not centered around hair follicles. Routine laboratory and radiological studies were within normal limits or negative. No treatment was given. She has noted hair growth for 6 months and that was nearly complete in one year. Given the histological findings and clinical history a diagnosis of postoperative alopecia was made.

DISCUSSION

Postoperative alopecia most likely occurs after

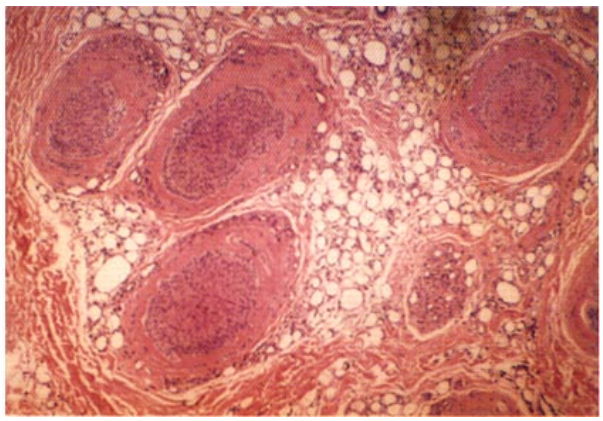

Fig. 2. Multiple catagen follicles showing thickened vitreous layer and fibrous root sheath, and degenerating cells within epithelial column in a transverse section just above the junction of the dermis and subcutis(H & E, ×40).

lengthy anesthesia associated with hypotension, blood loss or continued pressure over an area of the scalp. The cause of postoperative alopecia is a prolonged localized pressure to the scalp during and after operative procedures, which results in focal tissue ischemia[1,5,6]. The prime factor, therefore, appears to be a prolonged period of profound unconsciousness because the original injury would probably be quite painful to a conscious or sleeping patient[5]. The downward force of the head localized to one area over a number of hours damages the blood vessels of the scalp by direct traumatic crushing action or by blanching out the normally dense scalp vasculature and causing ischemia. The resultant ischemia may cause temporary cessation of follicular activity leading to loosening and depilation.

Initial signs and symptoms that may occur within the first postoperative week include focal pain, inflammation, and edema followed in some cases by an exudative or crusted eruption and ulceration[1]. The symptoms would abate preceding the defluvium by several days. But some patients might not note these prodromes before developing alopecia[7]. Defluvium would occur asymptomatically and quite rapidly, often within a day, and is usually complete within three to twenty-eight days following surgery. As seen in our case, there is occasional hyperpigmentation of the hairless area[1]. The appearance of gross ulceration presaged permanent alopecia, however, in some cases, was not preceded by gross ulceration[2].

The histopathologic findings depend upon when in

the evolution of the lesion the specimen is obtained[8,9]. Early in the course of the disease, before hair loss is complete, vascular thrombosis, inflammation and destruction may be seen in the dermis. A prominent feature of obliterative vasculitis appears after the onset of alopecia[1]. Once the alopecia develops, nearly all terminal follicles will be in the catagen or telogen phases. Focal vascular and tissue necrosis is present along with an associated chronic inflammatory infiltrate. It does not seem to be centered around hair follicles, but is usually associated with foci of vascular and tissue necrosis. Fat necrosis is often found, associated with infiltrate of foamy macrophages and mononuclear cells[9]. Fat atrophy, as seen in our case, could also be a possible finding. These additional histological changes render differentiation with alopecia areata and trichotillomania which also show multiple catagen follicles[10]. Our biopsy specimen did not demonstrate the definite feature of fat necrosis but morphologic changes in individual fat cells were conspicuous. These changes include hyalinized membrane associated with foamy cells, which are thought as a process of necrosis.

The differential diagnosis of postoperative alopecia may be variable as the clinical features develop[1]. The presence of exudative and later a crusted eruption immediately following operation might suggest pyoderma, kerion, seborrheic dermatitis, contact dermatitis, or herpes simplex[8]. The history and clinical features coupled with results of Wood's light examination, potassium hydroxide examination, and cultures provide the necessary diagnostic information. The strict localization to the pressure site following a lengthy operation and the lack of prior history of scalp disease would help to indicate the correct diagnosis. After the defluvium alopecia areata is a particular concern, because it may develop concomitantly or purely coincidentally with postoperative alopecia under stressful condition. The appearance that clinically the rather sudden loss of hair over a circumscribed area of scalp and usually complete, and the scalp otherwise appears normal, is identical with that of alopecia areata. Differentiation would be impossible from inspection if the area of baldness were located over or near the pressure site. Aside from its characteristic histological features, alopecia areata differs in that it sometimes has exclamation point hairs, no prodromes, more than one spot may develop, and regrowth is not a rule[5].

The prognosis of postoperative alopecia is generally favorable, with regrowth occurring within twelve weeks in most cases and with a low incidence of permanent alopecia. Permanent alopecia, defined as no hair regrowth by one year[11], may develop. The duration of the pressure is closely correlated with the likelihood of permanence of the hair loss. Lawson[2] described that permanent alopecia occurred in patients whose heads had been stationary for more than 24 hours. But there was a report of permanent alopecia resulting from the use of head strap during the 6 -hour anesthesia[7], so the duration may not be the sole factor that determines the permanency. The severity of antecedent scalp symptoms also seems to have no correlation with the possibility of permanency.

Postoperative alopecia is probably a preventable condition. Frequent repositioning of the head in cardiac surgery patients every 30 minutes both intraoperatively and during recovery until extubation has been reported in a prospective study with a reduced incidence of postoperative alopecia[2]. In this aspect, postoperative alopecia may be simply described as a pressure sore on the scalp. The simple act of repositioning of the head, therefore, is the key to eliminate the alopecia.

A prospective search revealed an incidence of 14 percent of 230 cases relative to a retrospective search of 7 percent of 225 cases following open-heart surgery, which means we had missed some of the cases before we actively sought them[2]. This finding may be attributed to the fact that postoperative alopecia can be overlooked as a minor complication relative to the causative surgical procedure because it cannot be a presenting symptom during the postoperative period. It is possible that these patients were not aware of scalp discomfort because of their poor condition necessitated analgesics and sedatives. Relatively short and minor surgery, as in our case, could also result in a postoperative alopecia. Not being an uncommon condition and disturbing to both patient and physician, postoperative alopecia therefore needs to be underscored among surgeons, anesthesiologists as well as dermatologists. That the burden of protecting the patient from postoperative alopecia falls primarily to medical attendants in the operation field implicates the significance of role of the dermatologist in reminding them of the iatrogenic nature of

this preventable complication.

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