Case 3
A 36-year-old Thai female from Bangkok
Chief complaint: Localized alopecia on the right temporoparietal area for 2 weeks

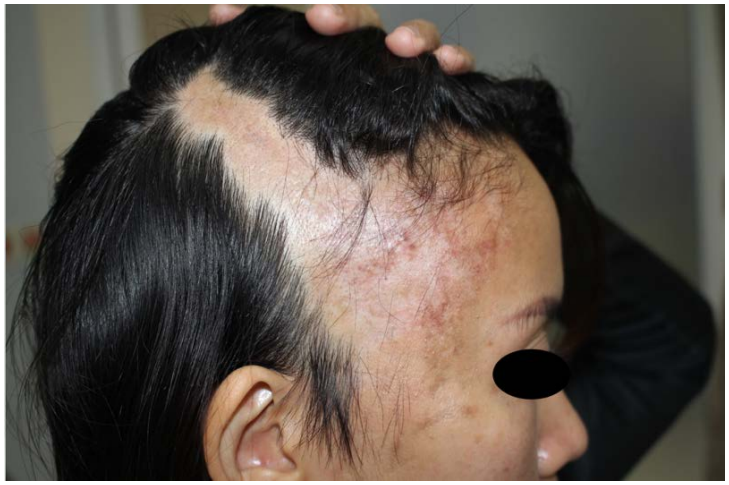

Fig 4.1

Present illness:
1 month ago, she underwent bilateral temple augmentation at an esthetic clinic by receiving the autologous fat grafting (2ml per side). Immediately, she felt flashing on the right eye and burning pain over her right temple without skin discoloration. On day1 after the procedure, the skin showed pallor and blanchable surrounding injected areas. Then, she had a necrotic skin with thin overlying eschar on the right temporoparietal area with clear discharge on day5. She was treated with ceftriaxone followed by amoxicillin/clavulanic acid. The patient developed localized alopecia that continued to deteriorate on day 14. By day 20, this deterioration showed well-demarcated non-scarring and scarring alopecia. She visited our department on day 35.

Past history: She underwent fat grafting on her face 4 months ago without any complications.

Physical examination: Unremarkable

Eye examination:
- VA: Rt 20/400 (20/80 PH)
  - Lt 20/200 (20/100 PH)
- No conjunctival injection, Cornea: clear
- A/C: no cells, Lens: clear
- No tortuous dilate artery and vein
- C/D: 0.3, Pink sharp disc

Dermatological examination: (Fig 4.1)
- Well-demarcated non-scarring and scarring alopecia on the right temporoparietal area
- Hair pull: Negative

Trichoscopy: (Fig 4.2)
- Non-scarring alopecia: Exclamation mark hairs, angulated hairs, black dots (a)
- Scarring alopecia: White patches, black dots and telangiectasia (b)

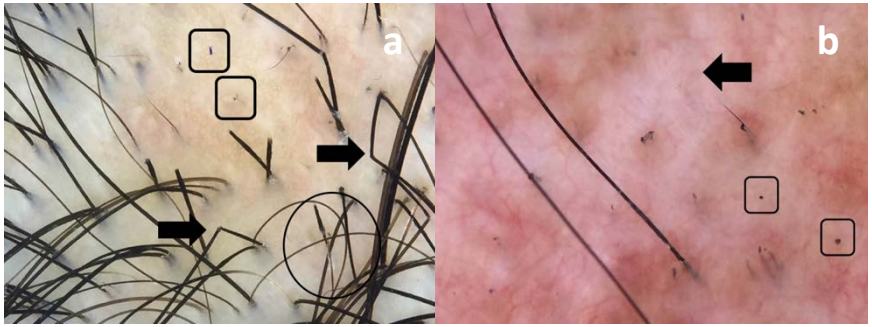

Fig 4.2

Investigation:

- Fundoscopy: WNL
- Optical coherence tomography of macula: WNL
- Fundus fluorescein angiography: Pending
- Indocyanine green chorioangiography: Pending
- Electroretinogram: Pending

Histopathology: (S18-20685, scalp) (Fig 4.3)

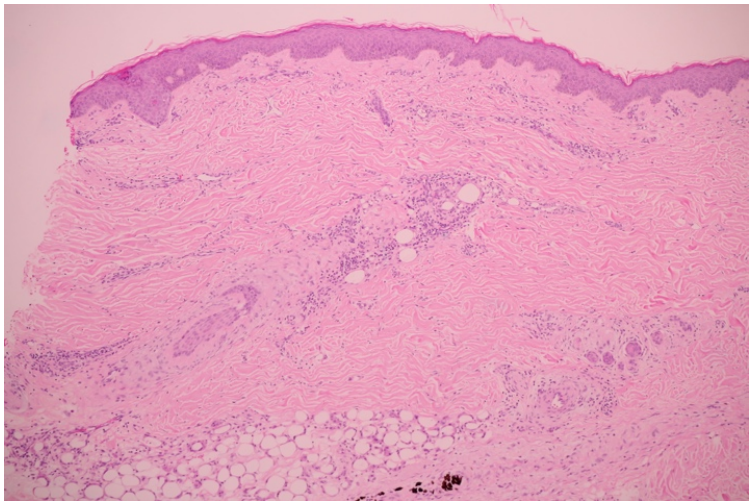

Fig4.3

- Inflammatory cells infiltrate predominately with lymphocytes and catagen hair follicles, abnormal adipocytes in the dermis

Diagnosis: Alopecia associated with scalp necrosis secondary to accidental embolization with autologous fat grafting

Treatment: Reassure and 5% minoxidil apply bid

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Discussion:

Autologous fat injection has become a crucial method in aesthetic medicine since it can correct various facial problems. The temple is one of well-accepted regions for fat grafting. However, some patients suffer from its serious complication, such as blindness and cerebral fat embolism.[1-4]

The anatomical sites of fat compartments, the plane of injection and the volume of augmentation are an important technique for fat-grafting in order to avoid complications and enhance patient satisfaction. Currently, there is no consensus on fat-grafting techniques in the temple. Huang et al.[5] demonstrated that an entry site was inserted at the medial side of the head between the junction of the hairline and the temporal line. Moreover, fat grafting should be placed into the loose areolar tissue layer (the deep plane) and the subcutaneous fat layer (the superficial plane). The former is composed of the upper temporal compartments (UTC) and the lower temporal compartments (LTC). The latter is composed of the lateral temporal-cheek fat compartments (LTFC) and the lateral orbital fat compartments (LOFC) of the temple.[5] The average optimal volume for augmentation in the temporal area was 5.9 mL with range of 2.0–10.0 mL per side.[3]

Superficial temporal artery (STA) supplies the temple. It runs within temperoparietal fascia (TPF) and finally bifurcates into the frontal (anterior) and parietal (posterior) branches at occurs 2-4 cm above the zygomatic arch in the majority of the individuals (60–88 %). Due to the variations of STA, it may run more superficial or deeper the surface of the TPF through

the subcutaneous fat layer and the loose areolar tissue layer, respectively.[6,7] Even fat grafting was placed into the recommend plane, the risk for vascular compromise can occur. In addition, intravascular injection in the STA can cause blindness due to anastomosis between the STA and supratrochlear and supraorbital artery which connect to ophthalmic artery. Retrograde embolization of the fat grafting, as the injected bolus may overcome arterial pressure and move against the direction of blood flow into the ophthalmic artery.[8,9] Autologous fat (47.9%) was the most common filler type to blindness.[8]

Arterial occlusion due to intra-arterial injection usually presents with an immediate skin blanching with sharp pain distal to obstruction. The involved site will develop reticulated erythema, purpura and ulceration and consequently, scarring.[10] Delayed onset arterial occlusion secondary to external compression by the injected material can also occur.[11] However, venous occlusion occurs either by accidental intravenous injection or by placing a large amount of the filler material in a small area leading to venous compression.[12] It has a more delayed presentation with continuous, dull aching pain, swelling and bruising of the skin.

Regarding the surgical literature, vascular obliteration from persistent external pressure such as prolonged head immobilization in the operation leads to tissue hypoxia and premature termination of the anagen phase.[13] Hair loss with sharp demarcation from the surrounding scalp occurs 2-3 weeks after the operation and permanent alopecia has also been reported in some cases.[14] Similar to alopecia secondary to fat grafting, intravascular injection into deep vessels causes partial- or full-thickness skin necrosis, resulting in tissue hypoxia and subsequent alopecia. The severity of local anoxia determines the extent of injury to the follicles and the prognosis.

The histopathological findings depend on a period of biopsy. In the early phase, vascular thrombosis and adnexal necrosis may be observed in the dermis as well as majority of hair follicles are in the catagen or telogen phase with or without trichomalasia, and melanin pigment in collapsed fibrous root sheaths. In the late phase, scarring alopecia may be shown.[15] Spontaneous resolutions probably occurred by given adequate collateral circulation from anastomoses within the temporal region. Partial or complete hair regrowth should be observed within 12 months.

We herein present a case of 36-year-old woman underwent bilateral temple augmentation with autologous fat injection. Biopsy on day 35 demonstrated non-scarring alopecia with near-total shift to catagen/telogen. The diagnosis was alopecia associated with scalp necrosis secondary to accidental embolization with autologous fat grafting. Necrosis of the right temporal scalp in this case was caused by intravascular injection into parietal branch of STA which supplies this scalp region. The patient also had opthalmic complication which is ongoing in the investigation. Our case was treated by 5% minoxidil solution and follow-up is ongoing.

In conclusion, this is the first case report of acute alopecia with scalp necrosis secondary to accidental embolization with autologous fat grafting. Patient counseling before performing

the procedure for this rare complication is recommended. Proper anatomical knowledge and accurate injection technique are essential to avoid potential severe complications.

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