INTRODUCTION

Migraine headache is a primary neurologic disease affecting millions of people worldwide. As a consequence, quality of life is diminished, productivity suffers (through loss of work force), and treatment costs are substantial. The occurrence rate in the general population is quite high, with women accounting for 3 of every 4 cases.

Background: Migraine headache is a primary neurologic disease affecting millions of people worldwide. As a consequence, quality of life is diminished, productivity suffers (through loss of work force), and treatment costs are substantial. The occurrence rate in the general population is quite high, with women accounting for 3 of every 4 cases.

Methods: Between January 2011 and May 2012, a total of 221 patients received hair transplants. Another 590 patients underwent hair transplantation between June 2012 and December 2016. Initially (first interval), patients were not questioned on migraine headaches in preoperative visits, but questioning was regularly done thereafter. Overall, 150 patients given transplants in the first period were surveyed by phone regarding preoperative migraine headaches. Aside from the incidental discovery, no other instances of migraine emerged. Headache origins were occipital-frontal in 2 patients, occipital-temporal in 2 patients, and occipital-temporal-frontal in the 2 others. Donor/receiver areas in hair transplantation and migraine trigger zones shared locations. Headache frequencies ranged from 4 to 8 days per month (average, 6 days), and pain scores were 5–8 (10 being highest). Duration of pain was 3–5 hours (average, 4 hours). All six patients had used various medications, such as triptans, ergot, and nonsteroidal anti-inflammatory drugs, before hair transplantation. The 1 female patient was a 32-year-old seeking treatment for alopecia, with a 6-year history of migraine headaches. The male patients presenting with androgenetic alopecia (grade 4–5 by Norwood classification) had 6- to 20-year migrainous histories.

Results: After hair transplantation, each migraine sufferer was checked once in the first month and then once every 3 months. Those who could not appear in person after the first year were evaluated by phone every 3 months. Migraine headaches had ceased in all 6 patients, none of whom used medical treatments for migraines thereafter. The postoperative improvement each patient experienced was dramatic ($P < 0.001$). Overall, the mean intensity of headaches declined from $6.6 \pm 1.47$ to 0, on an analog scale of 1–10 ($P < 0.001$); and mean headache frequency was reduced from $5.83 \pm 1.03$/month to 0/month ($P < 0.001$). Likewise, the migraine pain index fell from a mean of $149.33 \pm 19.21$/month to mean of 0/month ($P < 0.001$).

Conclusions: This report details 6 patients who experienced abatement of migraine headache symptoms following hair transplantation. The positive effects of hair transplantation on migraine headache and potential mechanisms of action are also discussed. 

(Plast Reconstr Surg Glob Open 2017;5:e1503; doi: 10.1097/GOX.0000000000001503; Published online 21 September 2017.)

Disclosure: The author has no financial interest to declare in relation to the content of this article. The Article Processing Charge was paid for by the author.
and some doctors are unaware of surgical treatment options, so referrals to plastic surgeons are uncommon. Despite the fact that medical treatment eases symptoms in many patients, drugs are not curative; and the precise pathologic mechanism of migraine has not been fully clarified.9–13 On the other hand, success rates of surgical procedures are rising. The objective of surgical intervention in migraine headache is deactivation of trigger sites by either decompression or avulsion.9–14 Although surgical treatment has proved effective in the 15 years of its use, the number of patients who cannot be completely treated is still very high.9–15 Botulinum neurotoxin,15 which has gained usage in recent years, provides temporary relief for some and helps identify candidates for surgery.15–17 Throughout the world, many men (> 60%) and women (50%) suffer from androgenetic alopecia and irreversible hair loss,18,19 which at present is frequently remedied by hair transplantation. In the past 10–15 years, a new hair restoration technique, known as follicular unit extraction, has been introduced.20–22 A patient electing hair transplantation at our clinic (~5 years ago) brought to our attention during a 6-month follow-up visit that his migraine headaches had ceased, restoring quality of life. After this first chance discovery, we initiated both retrospective and prospective investigations, documenting similar outcomes in other patients (5 males and 1 female). This report details 6 patients who experienced abatement of migraine headache symptoms following hair transplantation. The positive effects of hair transplantation on migraine headache and potential mechanisms of action are also discussed.

**SURGICAL TECHNIQUE**

All procedures were performed under local anesthesia. First, the donor area was infiltrated with lidocaine, using a tumescent solution to then inflate the scalp. This maneuver facilitates graft collection and implantation. Grafts were collected via punch (0.8–0.9 mm), anchored to a micromotor. For each graft, the punch was inserted into scalp to an average depth of 4–4.5 mm, at a micromotor rate of 3,000–5,000 cycles/min. The sharp punch tip was advanced under loose areolar tissue. In this way, hair follicles buried in fatty substrate were easily released. Subcutaneous veins and nerves in the donor field invariably were destroyed. The average length of collected grafts was 5 ± 0.5 mm. Once graft collection was complete, the receiver field was cultivated and local anesthesia was administered in the same manner as that in the donor field. A sterile razor was used to open recipient channels (length, 7 mm; width, 1 mm). Secured in a clamp, the same razor served to create pores in the receiver field (~50 pores/cm²). The average pore depth was 5 mm, incising scalp to level of periosteum. Grafts were then implanted manually, one by one. Vein and nerve damage within subcutis of receiver field was also typical.23 After transplantation, the average vascularization period of grafts is 8 days. A total of 2,500 grafts were placed in our first patient. Graft totals in the other male patients ranged from 3,000–4,500 (Figs. 1–3). Grafts were collected via micromotor method, using temporal (right and left) and occipital scalp as donor.

![Fig. 1](image_url). The images of a male patient, 57 years old, who had migraine headache for 20 years. A, B, Preoperative appearances. C, D, Postoperative 2-year appearances.
sites (Fig. 4A, B). Inflammation, proliferation, maturation, and remodeling phases of wound resolution proceeded without incident in receiver areas. The fact that thousands of microscopic tissue losses are sustained at donor locations is not unexpected. As receiver channels are developed through microincisions, cutaneous and subcutaneous structures are typically destroyed as well (Fig. 5C). All patients eventually recovered from transplant procedures, without long-term sequelae or complications. Hair growth in all patients was substantially complete within 6–8 months. Patient characteristics and methods between years 2011 and 2016, a total of 811 patients underwent hair transplantation by follicular unit extraction method. The vast majority (98%) were men (n = 796), the ages ranging from 32–57 years (average, 45 years). The 6 patients reported had suffered migraine headaches for 6–20 years. Our Research Ethics Committee granted approval for this retrospective clinical study, with consent of all patients involved. Between January 2011 and May 2012, a total of 221 patients received hair transplants. Another 590 patients underwent hair transplantation between June 2012 and December 2016. Initially (first interval), patients were not questioned on migraine headaches in preoperative visits, but questioning was regularly done thereafter. Overall, 150 patients given transplants in the first period were surveyed by phone regarding preoperative migraine headaches. Preoperative and postoperative surveys are given below. Aside from the 1 incidental discovery, no other instances of migraine emerged. Headache origins were occipital-frontal in 2 patients, occipital-temporal in 2 patients, and occipital-temporal-frontal in the 2 others. Donor/receiver areas in hair transplantation and migraine trigger zones shared locations. Headache frequencies ranged from 4–8 days per month (average, 6 days), and pain scores were 5–8 (10 being highest). Duration of pain was 3–5 hours (average, 4 hours). All 6 patients had used various medications, such as triptans, ergot, and

Fig. 2. The images of a male patient, 47 years old, who had migraine headache for 8 years. A, B, Preoperative appearances. C, D, Postoperative 3-year appearances.

Fig. 3. Three-year postoperative male patient, 43 years old, who had migraine headache for 10 years.
nonsteroidal anti-inflammatory drugs, before hair transplantation. The 1 female patient was a 32-year-old seeking treatment for alopecia, with a 6-year history of migraine headaches. The male patients presenting with androgenetic alopecia (grade 4–5 by Norwood classification) had 6- to 20-year migrainous histories. Patient characteristics and treatments are summarized in Table 1. Ages, genders, and symptoms of these patients were obtained from demographic records at first presentation. Once the first patient came forward, a migraine headache questionnaire was routinely requested of every patient, documenting headache frequency, duration, and severity. A migraine headache index was computed as (frequency: days/mo) × (duration in 24-hour period) × (pain score: 0–10), using survey responses and other collected data (Table 2). Quantifiable parameters were expressed as mean ± SD. To compare preoperative and postoperative variables, a 2-tailed t test was applied, setting significance at \( P < 0.05 \).

PREOPERATIVE SURVEY

1. Have you had headaches?

2. Did you get a migraine diagnosis? Who made the diagnosis?

3. How long do you have migraines?

4. How many migraine attacks do you have in a month?

5. How long does this pain last?

6. Does anyone have migraines in the family?

7. Do you have aura?

8. Do you have symptoms associated with migraine?

9. How would you classify the severity of your pain between 1–10?

10. Where exactly does migraine pain start?

   [Behind the left eye, behind the right eye, the back of both eyes, left temporal side, right temporal side, in both temporal sides, above the left eyebrow, above the right eyebrow, above both eyebrows, left back of head, right back of head, both sides of the head.]

POSTOPERATIVE SURVEY

Does your pain continue?

Did you have any attack after surgery? Yes/No

If yes, is there a change in frequency?

When did your pain disappear? Immediately after, 1 month after, 6 months after.

Did you take medication? Yes/No

Has your quality of life improved?

RESULTS

After hair transplantation, each migraine sufferer was checked once in the first month and then once every 3 months. Those who could not appear in person after the first year were evaluated by phone every 3 months. Migraine headaches had ceased in all 6 patients, none of whom used medical treatments for migraines thereafter. The postoperative improvement each patient experienced was dramatic \( (P < 0.001) \). Overall, the mean intensity of headaches declined from 6.6 ± 1.47 to 0, on an analog scale of 1–10 \( (P < 0.001) \), and mean headache frequency was reduced from 5.83 ± 1.03/mo to 0/mo \( (P < 0.001) \). Likewise, the migraine pain index fell from a mean of 149.33 ± 19.21/mo to mean of 0/mo \( (P < 0.001; \) Table 3).
It is currently conceded that migraine headaches are not fully treatable through symptomatic medical intervention, and the possibility of better results by surgical means is echoed in recent studies. In the past 15 years, various authorities (primarily Guyuron) have conducted numerous studies (e.g., anatomic, retrospective, prospective/randomized) aimed at surgical (or sham) treatment of migraine headaches. Based on the 5-year follow-up data, it would appear that surgery is indeed effective, but the underlying pathophysiology of migraines remains unclear. Related theories have implicated neuronal mediators, cortical neuronal hyperexcitability, peripheral and central activation of trigeminal nerves, and abnormal sensitization of the nociceptive system, the latter reflecting central activation of trigeminal nerves, and abnormal sensitization and abnormal excitability. However, the cause of neuropeptide release is not entirely understood. Related theories have implicated neuronal mediators, cortical neuronal hyperexcitability, peripheral and central activation of trigeminal nerves, and abnormal sensitization of the nociceptive system, the latter reflecting periaqueductal gray matter dysfunction.

Table 1. Migraine Headache and Hair Transplantation Patient Records

| Patient Number | Sex | Disease Duration (y) | Age (y) | Drugs Preoperative | Follow-Up (mo) | Headache Satisfaction (%) | Medication | Symptoms Reduction (%) |
|---------------|-----|----------------------|---------|------------------|---------------|-------------------------|------------|------------------------|
| 1             | Female | 6 | 32 | Triptans + nonspecific | 58 | 100 | None | 100 |
| 2             | Male | 29 | 57 | Triptans + nonspecific | 36 | 100 | None | 100 |
| 3             | Male | 8 | 47 | Triptans + nonspecific | 26 | 100 | None | 100 |
| 4             | Male | 10 | 43 | Ergot + triptans | 21 | 100 | None | 100 |
| 5             | Male | 12 | 46 | Triptans + nonspecific | 18 | 100 | None | 100 |
| 6             | Male | 6 | 42 | Ergot + triptans | 6 | 100 | None | 100 |

O, occipital; F, frontal; T, temporal.
Non-specific: anti-inflammatory drugs.

DISCUSSION

It is currently conceded that migraine headaches are not fully treatable through symptomatic medical intervention, and the possibility of better results by surgical means is echoed in recent studies. In the past 15 years, various authorities (primarily Guyuron) have conducted numerous studies (e.g., anatomic, retrospective, prospective/randomized) aimed at surgical (or sham) treatment of migraine headaches. Based on the 5-year follow-up data, it would appear that surgery is indeed effective, but the underlying pathophysiology of migraines remains unclear. Related theories have implicated neuronal mediators, cortical neuronal hyperexcitability, peripheral and central activation of trigeminal nerves, and abnormal sensitization of the nociceptive system, the latter reflecting periaqueductal gray matter dysfunction. Given these influences, meningeal inflammation ensues, activating trigeminal nerves and perivascular sensory fibers. Vasodilation is then triggered through release of calcitonin gene-related peptide, substance P, and neurokinin A, all residing in trigeminal neurons. Sensitization and abnormal excitability. However, the cause of neuropeptide release is not entirely understood. Related theories have implicated neuronal mediators, cortical neuronal hyperexcitability, peripheral and central activation of trigeminal nerves, and abnormal sensitization of the nociceptive system, the latter reflecting periaqueductal gray matter dysfunction.

Table 2. Migraine Headache Index

| Patient Number | Pain Frequency (d/mo) | Scale of Pain (with 10 being Highest) | Pain Duration (h/24) | Pain Index before Hair Transplantation | Pain Index after Hair Transplantation |
|---------------|----------------------|--------------------------------------|---------------------|--------------------------------------|--------------------------------------|
| 1             | 4                    | 8                                    | 5                   | 160                                  | 0                                    |
| 2             | 5                    | 7                                    | 4                   | 140                                  | 0                                    |
| 3             | 6                    | 7                                    | 4                   | 168                                  | 0                                    |
| 4             | 8                    | 5                                    | 3                   | 120                                  | 0                                    |
| 5             | 7                    | 6                                    | 4                   | 168                                  | 0                                    |
| 6             | 5                    | 7                                    | 4                   | 140                                  | 0                                    |

Greater occipital nerve domain, both auriculotemporal and zygomaticotemporal branches of trigeminal nerve may be destroyed bilaterally from excessive grafting. Receiver areas for hair grafts are usually frontal area, apex, and vertex (Fig. 5). In these regions, approximately 50 pores (average depth, 5–6 mm) are opened in each square centimeter, damaging veins, nerves, and soft tissues (Fig. 5). Depending on the nature of hair loss, the auriculotemporal and zygomaticotemporal branches of trigeminal nerve, the distal branches of supraorbital and supratrochlear nerves (extending toward frontal area), the distal branches of occipital nerve at vertex, and the arteries and veins accompanying these nerves may be damaged as well. In grafts incorporating 0.4 mm² of tissue (skin surface area), roughly 15 cm² of tissue is lost for every 3,000 grafts procured from donor areas (graft radius, r = 0.4 mm; area [πr²] = 3.14 × 0.4 × 0.4 × 3,000 grafts = 1,500 mm² = 15 cm²). Such extensive tissue loss may alter occipital skin and subcutis, which then tightens. Hair transplants are composite in nature, combining skin, connective tissue, aponeurosis, and loose areolar tissue, so donor side tissue losses impact both appearance and contours. The genetics of hair in occipital region differs from that in frontal region and apex. Hair follicles are capable of self-regulating responses to androgens through expressions of 5-alpha reductase and androgen receptors. The latter elements quantifiably differ in areas of alopecia and hair-bearing scalp. Accordingly, occipital hairs maintain resistance to male androgens when transplanted to vertex, and scalp hairs transplanted from vertex to forearm regress at the same pace as donor site. Both tissue destruction and genetic underpinnings may account for differences within receiver fields. It is at these sites where migraine-triggering neurotransmitters are released, thus serving as points of neuromodulation. After hair transplantation, blood circulation increases substantially in both donor and receiver areas, whereas disrupted vascular networks and fibrofatty tissues no longer
Contribute to vasospasm. As confirmed by Guyuron et al., transecting the zygomaticotemporal branch of trigeminal nerve and repositioning temporal soft tissues minimizes the potential for neural coaptation, diminishing migraine headache recurrence. Hair transplantation may affect receiver and donor areas in a similar manner. Given the enhanced regional blood flow, growth factors are apt to penetrate faster, facilitating wound recovery and maturation. Regenerating nerves also undergo change, especially myelin sheaths, leading to improved sequencing of neurotransmitter release and propagation and stem cells conveyed via perifollicular fat of composite grafts aid in tissue recovery. Trigger sites of migraine headaches in occipital, frontal, and temporal regions that occupy donor tissue recovery. Pain frequency $5.83 \pm 1.47$ $0 \pm 0$ $< 0.0001$

Table 3. Comparison of Preoperative and Postoperative Symptoms

| Characteristics | Before | After | $P$ |
|-----------------|--------|-------|-----|
| Pain frequency  | $5.83 \pm 1.47$ | $0 \pm 0$ | $< 0.0001$ |
| Pain scale      | $6.66 \pm 1.03$ | $0 \pm 0$ | $< 0.0001$ |
| Pain duration   | $4 \pm 0.3$ | $0 \pm 0$ | $< 0.0001$ |
| Pain index      | $149.33 \pm 19.21$ | $0 \pm 0$ | $< 0.0001$ |

Conclusions

The seemingly curative effect of hair transplantation on migraine headaches has no clear explanation as yet. However, clinical and experimental studies of larger populations are surely forthcoming. Results were decisive in these few patients. It is remarkable that both alopecia and migraine headache may be easily remedied through a simple and easily applied procedure. As a result of this study, hair plantation in bald migraine patients may be an alternative to migraine treatment. In nonbald patients, the mechanism of action of hair transplantation may be a guide for migraine treatment. Previously, various mechanisms of central and peripheral origin have been emphasis in the treatment of migraine. In recent years, however, the focus has been on the origin of peripheral nerves. This study supports that the main cause of migraine originates from peripheral nerves. This study shows that migraine symptoms may improve in patients who do not need hair transplantation when they are damaged in the transcutaneous peripheral nerve, both in donor and recipient areas, as in hair transplantation. Our work will be a reference for such a study on patients in the future.

REFERENCES

1. Stewart WF, Schechter A, Rasmussen BK. Migraine prevalence: a review of population-based studies. Neurology. 1994;44:S17–S23.
2. Betul B, Mustafa E, Necdet K, et al. Migraine incidence in 5 years: a population-based prospective longitudinal study in Turkey. J Headache Pain. 2015;16:103–113.
3. Abu-Arefeh I, Razak S, Sirvabam B, et al. Prevalence of headache and migraine in children and adolescents: a systematic review of population-based studies. Dev Mol Child Neurol. 2010;52:1088–1097.
4. Abu-Arefeh I, Russell G. Prevalence of headache and migraine in school children. BMJ. 1994;309:765–769.
5. Split W, Neuman W. Epidemiology of migraine among students from randomly selected secondary schools in Lodz. Headache. 1999;39:494–501.
6. Rozen TD. Migraine prevention: what patients want from medication and their physicians (a headache specialty clinic perspective). Headache. 2006;46:750–753.
7. Young WB, Hopkins MM, Shechter AL, et al. Topiramate: a case series study in migraine prophylaxis. Cephalalgia. 2002;22:659–663.
8. Peres MF, Silberstein S, Moreira F, et al. Patients’ preference for migraine preventive therapy. Headache. 2007;47:540–545.
9. Guyuron B, Varghais A, Michelow BJ, et al. Corrugator supercilii muscle resection and migraine headaches. Plast Reconstr Surg. 2000;106:429–434; discussion 435.
10. Guyuron B, Tucker T, Davis J. Surgical treatment of migraine headaches. Plast Reconstr Surg. 2002;109:2183–2189.
11. Guyuron B, Kidger JS, Davis J, et al. Comprehensive surgical treatment of migraine headaches. Plast Reconstr Surg. 2005;115:19–21.
12. Guyuron B, Reed D, Kidger JS, et al. A placebo-controlled surgical trial of the treatment of migraine headaches. Plast Reconstr Surg. 2009;124:461–468.
13. Guyuron B, Kidger JS, Davis J, et al. Five-year outcome of surgical treatment of migraine headaches. Plast Reconstr Surg. 2011;127:605–608.
14. Guyuron B, Tucker T, Davis J. Surgical treatment of migraine headaches. Plast Reconstr Surg. 2003;112:164–170.
15. Janis JE, Dhanik A, Howard JH. Validity of the peripheral trigger point theory of migraine headaches: single-surgeon experience using botulinum toxin and surgical decompression. Plast Reconstr Surg. 2011;128:123–131.
16. Poggi JT, Grizzell BE, Helmer SD. Confirmation of surgical decompression to relieve migraine headaches. Plast Reconstr Surg. 2008;122:115–22; discussion 123.
Ors • Hair Transplantation in Migraine Headache Patients

17. Dirnberger F, Becker K. Surgical treatment of migraine headaches by corrugator muscle resection. Plast Reconstr Surg. 2004;114:652–657; discussion 658.
18. Gan DC, Sinclair RD. Prevalence of male and female pattern hair loss in Maryborough. J Invest Dermatol Symp Proc. 2005;10:184–189.
19. Otberg N, Finner AM, Shapiro J. Androgenetic alopecia. Endocrinol Metab Clin North Am. 2007;36:379–398.
20. Poswal A Donor sealing: a novel method in hair transplant surgery. Indian J Dermatol. 2006; 51:55.
21. Woods R, Campbell AW. Chest hair micrografts display extended growth in scalp tissue: a case report. Br J Plast Surg. 2004;57:789–791.
22. Rassman WR, Bernstein RM, McClellan R, et al. Follicular unit extraction: minimally invasive surgery for hair transplantation. Dermatol Surg. 2002;28:720–728.
23. Randall VA, Thornton MJ, Messenger AG. Cultured dermal papilla cells from androgen-dependent human hair follicles (e.g. beard) contain more androgen receptors than those from non-balding areas of scalp. J Endocrinol. 1992;133:141–147.
24. Neumann S, Dumbell TP, Leslie T, et al. Inflammatory pain hypersensitivity mediated by phenotypic switch in myelinated primary sensory neurons. Nature. 1990;384:360–364.
25. Woolf CJ. Phenotypic modification of primary sensory neurons: the role of nerve growth factor in the production of persistent pain. Philos Trans R Soc Lond B Biol Sci. 1996;351:441–448.
26. Burstein R, Jakubowski M. Unitary hypothesis for multiple triggers of the pain and strain of migraine. J Comp Neurol. 2005;493:9–14.
27. Waer G, Moskowitz MA. Therapeutic implications of central and peripheral neurologic mechanisms in migraine. Neurology. 2003;61:S9–S20.
28. Goadsby PJ. Pathophysiology of headache. In: Silberstein SD, Lipton RB, Solomon S, eds. Wolf’s Headache and Other Head Pain. 7th ed. Oxford, England: Oxford University Press; 2001:57–72.
29. Feindel W, Penfield W, McNaughton F. The tentorial nerves and localization of intracranial pain in man. Neurology. 1960;10:555–563.
30. Liu-Chen LY, Gillespie SA, Norregaard TV, et al. Co-localization of retrogradely transported wheat germ agglutinin and the putative neurotransmitter substance P within trigeminal ganglion cells projecting to cat middle cerebral artery. J Comp Neurol. 1984;225:187–192.
31. Edvinsson L, Brodin E, Jansen I, et al. Neurokinin A in cerebral vessels: characterization, localization and effects in vitro. Regul Pept. 1988;20:181–197.
32. Goadsby PJ, Edvinsson L, Ekman R. Vasoactive peptide release in the extracerebral circulation of humans during migraine headache. Ann Neurol. 1990;28:183–187.
33. Ebersberger A, Averbuck B, Messinger K, et al. Release of substance P, calcitonin gene-related peptide and prostaglandin E2 from rat dura mater encephali following electrical and chemical stimulation in vitro. Neuroscience. 1999;89:901–907.
34. Goadsby PJ, Hoskin KL. The distribution of trigeminovascular afferents in the nonhuman primate brain Macaca nemestrina: a c-fos immunocytochemical study. J Anat. 1997;190:367–375.
35. Hoskin KL, Zaganis AS, Goadsby PJ. Stimulation of the middle meningeal artery leads to Fos expression in the trigemino-cerebral nucleus: a comparative study of monkey and cat. J Anat. 1999;194:579–588.
36. May A, Goadsby PJ. The trigeminovascular system in humans: pathophysiological implications for primary headache syndromes of the neural influences on the cerebral circulation. J Cereb Blood Flow Metab. 1999;19:115–127.
37. Dimitriadou V, Buzzi MG, Theoharides TC, et al. Ultrastructural evidence for neurogenically mediated changes in blood vessels of the rat dura mater and tongue following antidiromic trigeminal stimulation. Neuroscience. 1992;48:187–203.
38. Uddman R, Edvinsson L, Ekman R, et al. Innervation of the facial cerebral vasculature by nerve fibers containing calcitonin gene-related peptide: trigeminal origin and co-existence with substance P. Neurosci Lett. 1985;62:131–136.
39. Ols S, Ozkose M, Ols S. Follicular unit extraction hair transplantation with micromotor: eight years experience. Aesthetic Plast Surg. 2015;39:589–596.
40. Thornton MJ, Laing I, Hamada K, et al. Differences in testosterone metabolism by beard and scalp hair follicle dermal papilla cells. Clin Endocrinol (Oxf). 1993;39:633–639.
41. Itami S, Kurata S, Takayasu S. 5 Alpha-reductase activity in cultured human dermal papilla cells from beard compared with reticular dermal fibroblasts. J Invest Dermatol. 1990;94:150–152.
42. Sawaya ME, Price VH. Different levels of 5 alpha-reductase type I and II, aromatase, and androgen receptor in hair follicles of women and men with androgenetic alopecia. J Invest Dermatol. 1997;109:296–300.
43. Boudou P, Reygagne P. Increased scalp skin and serum 5 alpha-reductase reduced androgens in a man relevant to the acquired progressive kinky hair disorder and developing androgenetic alopecia. Arch Dermatol. 1997;133:1129–1133.
44. Orentreich N. Autografts in alopecias and other selected dermatological conditions. Ann N Y Acad Sci. 1959;83:463–479.