ORIGINAL ARTICLE



Ten-year Experience of Superior Gluteal Artery Perforator Flap for Reconstruction of Sacral Defects in Tri-Service General Hospital

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Background: Despite advances in reconstruction techniques, sacral sores continue to present a challenge to the plastic surgeon. The superior gluteal artery perforator (SGAP) flap is a reliable flap that preserves the entire contralateral side as a future donor site. On the ipsilateral side, the gluteal muscle itself is preserved and all flaps based on the inferior gluteal artery are still possible. However, the dissection of the perforator is tedious and carries a risk of compromising the perforator vessels. Patients and Methods: During the period between April 2003 and March 2013, 30 patients presented to our section with sacral wounds causing by pressure sores or infected pilonidal cysts. Of a total of 30 patients, 13 were female and 17 were male. Their ages ranged from 22 to 92 years old (mean 79.8 years old). Surgical intervention was performed electively with immediate or delayed reconstruction using a SGAP flap. The characteristics of patients' age, and sex, and cause of sacral defect, co-morbidities, wound culture, flap size, perforator number, hospital stay, and outcome were reviewed. **Results:** For all operations, the length of the pedicle dissection will not exceed 1 cm because of the vascular anatomy of the SGAP, which lies adjacent to the sacral region. Due to short pedicle dissection, all SGAP flap were elevated around an hour. All flaps survived except two, which had partial flap necrosis and were finally treated by contralateral V-Y advancement flaps coverage. The mean follow-up period was 14.8 months (range 3-24). No flap surgery-related mortality was found. Conclusion: Perforator-based flaps have become popular in modern reconstructive surgery because of low donor site morbidity and good preservation of muscle. Our study shows that deep pedicle dissection is unnecessary when the surgery involves an accurate indicating perforator, adequate flap size design, and correct selection of flap utilization between tunnel and rotation. The advantages of this modification include a faster operation, less bleeding, and less trauma of the pedicle, which make the SGAP flaps an alternative choice for sacral sores coverage.

Key words: Superior gluteal artery perforator flap, sacral sore, reconstruction

INTRODUCTION

Reconstructing a sacral defect caused by pressure sores or infected pilonidal cysts is a common problem for reconstructive surgeons. Many surgical methods have been used to correct sacral sores, including primary closure, skin grafting, local random flaps, and muscle flaps. Muscle and myocutaneous flaps have been used

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successfully in pressure sore coverage¹ and are the first choice for the management of pressure sores because they provide excellent blood supply and durable coverage. On the other hand, limited shifting capacity, excessive blood loss, and sacrifice of the muscle are the major drawbacks of the procedure.¹⁻³

In the recent years, the concept of perforator-based flaps has been used for covering sacral defects. Koshima *et al.*⁴ first described a gluteal perforator flap for repairing sacral pressure sores. Then, Verpaele *et al.*⁵ introduced the superior gluteal artery perforator (SGAP) flap for covering sacral pressure sores. The SGAP flap provides an ample amount of tissue, with good vascularity, to cover large sacral pressure sores in one stage and does not sacrifice the vascularity or innervation of the underlying gluteus maximus muscle.⁵ This report presents our experience in successful reconstruction of sacral pressure sores using this SGAP flap.

PATIENTS AND METHODS

During the period of month between April 2003 and March 2013, 30 patients presented to our section with sacral wounds causing by pressure sores or infected pilonidal cysts. Of a total of 30 patients, 13 were female and 17 were male. Their ages ranged from 22 to 92 years old (mean 79.8 years old). The most common medical comorbidity found in our patients was type 2 diabetes mellitus (21 patients, 70%), followed by chronic renal insufficiency (20 patients, 67%), hypertension (19 patients, 63%), and congestive heart failure (17 patients, 57%). The causes of sacral ulcerations included pressure sores in 28 patients and infected pilonidal cysts in two patients. After admission, broad-spectrum antibiotics were prescribed and changed to specific ones if a specific organism was isolated from the wound culture.

The specific organism cultured from the wound including methicillin-resistant *Staphylococcus aureus* (11 patients, 37%), *Acinetobacter baumannii* (7 patients, 23%), *Pseudomonas aeruginosa* (6 patients, 20%), *Entamoeba coli* (6 patients, 20%), *Enterococcus faecalis* (5 patients, 17%), *Candida albicans* (3 patients, 10%), and *S. aureus* (2 patients, 7%). Local wound care was performed using wet-gauze dressing with saline-diluted iodine twice a day to achieve wound bed conditioning. Surgical intervention was performed electively with immediate or delayed reconstruction using a SGAP flap. The characteristics of patients' age, and sex, cause of sacral defect, co-morbidities, wound culture, flap size, perforator number, hospital stay, and outcome were reviewed [Table 1].

Vscular anatomy

The superior gluteal artery (SGA) arises from the internal iliac artery. The SGA gives off a deep branch to the gluteus medius muscle and then runs through the gluteus maximus muscle, ending in cutaneous arteries located mainly in the superolateral gluteal region. The SGA can be marked on the skin of the buttock at a place one-third of the way on a line drawn from the posterior superior iliac spine (PSIS) to the top of the greater trochanter (T) [Figure 1]. The piriformis muscle (PM) can be located topographically on the buttock skin on a line drawn between the greater trochanter and a point halfway to the sacrum. Perforators can be found in the area lateral to the SGA and above the PM.⁵ In 1993, Koshima described the detail anatomy of the SGA perforator.⁴ The length of the vessels varies from 3 to 8 cm and their diameter from 1 to 1.5 mm. These flaps can be nourished even with only one perforator. In our cases, the largest SGAP flap with one perforator is 12 $cm \times 14 cm$.

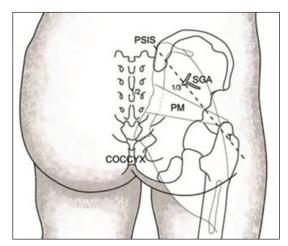


Figure 1. Stages of the operation using the superior gluteal artery (SGA) perforator flap. (a) Anatomical landmarks: The SGA emerges at the junction of the middle and medial thirds of a line drawn between the posterior superior iliac spine and the lateral border of the greater trochanter (T)

Surgical procedure

With the patient in the prone position, the anatomical landmarks are drawn and the SGA and its perforators are identified with the help of unidirectional Doppler ultrasound. The locations of the SGA perforators are situated mainly around the junction of the middle and medial third of a line drawn between the PSIS and the greater trochanter. A template of the defect helps ensure that the recipient site and donor tissue are of proper size and shape. The flap template is placed on the perforator mark; when doing so, it is essential to design the skin paddle with an extra 2 cm length for the long side of template to make the SGAP flap as an ellipse and ensure the flap easily reach the most distal point of sacral defect by rotating.

The incision is made superiorly and then continued down through skin, subcutaneous fat, and fascia to the muscle. From there, the flap is detached from the muscle until the chosen perforator is encountered, usually in the fibrous perimysium. The vessel is slowly dissected out about 1 cm in length by splitting the muscle fibers rather than by cutting under loupe magnification, and the vessel loop is placed around the perforator. The fibrous septa of the perimysium can be preserved from the dissection. Once the vessel is found suitable, the inferior border of the flap is incised and the flap is raised away from the muscle fully to form an island flap. The island flap can then be transposed into the defect. All donor sites are closed primarily. We prefer to close the donor site first to reduce the tension between the flap and defect. Suction drainage is applied under the flap and in the donor area until amount less than 10 cc is collected per 24 h. The patient remains prone or on his/her side until the flap heals.

Table 1. Characteristics of patients' age, sex, cause of sacral defect, co-morbidities, wound culture, flap size, perforator number, hospital stay, and outcome

| Patient | Sex/ Age (year) | Cause of sacral defect | Co-morbidities | Wound culture | Debridement (times) | Flap size (cm ²) | Perforator number | Hospital stay (day) | Outcome |
|---------|--------------------|------------------------------------|-------------------|---------------|---------------------|------------------------------|----------------------|------------------------|-----------------------|
| 1 | M/22 | Infected pilonidal cyst | None | SA | 1 | 168 | 1 | 12 | Good |
| 2 | M/48 | Oral cancer, bed ridden | DM | No growth | 1 | 42 | 1 | 14 | Good |
| 3 | M/62 | ICH, bed ridden | HTN | EC, PA | 2 | 56 | 1 | 18 | Good |
| 4 | F/66 | ICH, bed ridden | DM, HTN | No growth | 1 | 60 | 2 | 13 | Good |
| 5 | M/68 | Stroke, bed ridden | DM, HTN, CRI | AB | 4 | 45 | 1 | 58 | Partial flap necrosis |
| 6 | M/55 | Laryngeal cancer, bed ridden | None | MRSA, EF | 2 | 72 | 1 | 20 | Good |
| 7 | F/72 | Dementia, bed ridden | DM, CRI | AB, EF | 2 | 60 | 1 | 21 | Good |
| 8 | M/73 | Stroke, bed ridden | DM, HTN, CRI | MRSA, PA | 3 | 96 | 1 | 24 | Good |
| 9 | F/73 | Stroke, bed ridden | DM, HTN | AB | 2 | 108 | 1 | 19 | Good |
| 10 | M/28 | Infected pilonidal cyst | None | MRSA | 3 | 108 | 1 | 26 | Wound edge dehiscence |
| 11 | M/75 | Dementia, bed ridden | DM, HTN, CRI, CHF | MRSA | 2 | 72 | 1 | 23 | Good |
| 12 | M/75 | Stroke, bed ridden | HTN, CHF | EF, PA | 2 | 90 | 1 | 17 | Good |
| 13 | M/76 | Stroke, bed ridden | DM, CRI | EC | 3 | 165 | 1 | 28 | Good |
| 14 | F/77 | Stroke, bed ridden | DM, HTN, CRI, CHF | MRSA | 2 | 117 | 1 | 19 | Good |
| 15 | F/79 | Stroke, bed ridden | DM, HTN, CRI, CHF | AB, CA | 2 | 42 | 1 | 25 | Wound edge dehiscence |
| 16 | F/80 | Stroke, bed ridden | DM, CRI, CHF | SA, EF | 2 | 42 | 2 | 17 | Good |
| 17 | M/80 | Stroke, bed ridden | HTN, CRI, CHF | MRSA, PA | 2 | 84 | 1 | 22 | Good |
| 18 | F/81 | Stroke, bed ridden | DM, HTN, CRI, CHF | AB, EC | 2 | 42 | 1 | 25 | Good |
| 19 | F/82 | Parkinson's disease, bed ridden | DM, HTN, CRI, CHF | PA, CA | 2 | 70 | 2 | 21 | Good |
| 20 | M/83 | Parkinson's disease, bed ridden | DM, HTN, CRI, CHF | AB | 3 | 136 | 1 | 30 | Good |
| 21 | F/83 | Stroke, bed ridden | DM, CRI, CHF | MRSA | 2 | 56 | 1 | 17 | Good |
| 22 | M/83 | Stroke, bed ridden | HTN, CRI, CHF | No growth | 2 | 56 | 2 | 14 | Good |
| 23 | M/87 | Dementia, bed ridden | DM, HTN, CRI, CHF | AB | 2 | 42 | 1 | 17 | Good |
| 24 | F/88 | Dementia, bed ridden | DM, HTN, CRI, CHF | PA | 4 | 56 | 1 | 50 | Partial flap necrosis |
| 25 | F/89 | Dementia, bed ridden | DM, CRI, CHF | EC | 3 | 63 | 2 | 27 | Good |
| 26 | M/92 | Stroke, bed ridden | DM, CRI, CHF | MRSA, EF | 2 | 154 | 2 | 31 | Good |
| 27 | F/83 | Leukemia, bed ridden | DM, CRI, CHF | EC, CA | 3 | 80 | 1 | 28 | Good |
| 28 | M/50 | HIVD, bed ridden | HTN | MRSA | 3 | 88 | 1 | 34 | Wound edge dehiscence |
| 29 | F/72 | ICH, bed ridden | HTN | MRSA | 2 | 117 | 1 | 22 | Good |
| 30 | M/78 | Dementia, bed ridden | DM, HTN, CRI, CHF | MRSA, EC | 3 | 120 | 2 | 27 | Good |

ICH: Intracranial hemorrhage; HIVD: Herniated intervertebral disc; DM: Diabetes mellitus; HTN: Hypertension; CRI: Chronic renal insufficiency; CHF: Congestive heart failure; SA: Staphylococcus aureus; MRSA: Methicillin-resistant Staphylococcus aureus; PA: Pseudomonas aeruginosa; AB: Acinetobacter baumannii; EF: Enterococcus faecalis; CA: Candida albicans; EC: Entamoeba coli

RESULTS

The sacral ulcerations of these 30 patients successfully treated with debridement and immediate or delayed reconstruction using SGAP flaps. All flaps survived, although venous congestion was common immediately following surgery. None of our patients required conversion to a

myocutaneous rotation flap. Twenty-three flaps were found with one SGA perforator, seven flaps had two perforators. The flap size ranged between $7 \text{ cm} \times 6 \text{ cm}$ and $12 \text{ cm} \times 14 \text{ cm}$. For all operations, the length of the pedicle dissection will not exceed 1 cm because of the vascular anatomy of the SGAP, which lies adjacent to the sacral region. Due to short pedicle dissection, all SGAP flap were elevated around an hour.

Twenty-five sacral wound (83%) healed completely with no further operations to achieve defect coverage. Two flaps (7%) developed partial necrosis, which were finally treated by contralateral V-Y advancement flaps coverage. Three minor complications of partial dehiscence of the wound edge (10%) were finally managed by delayed primary closure. All the five flaps with wound dehiscence were infected by methicillin-resistant *S. aureus* (three patients, 60%), *A. baumannii* (two patients, 40%), and *C. albicans* (one patient, 20%). The mean debridment was 2.3 times (range 1-4) with average hospital stay 24.9 days (range 12-58). All donor sites were closed primarily and healed without complication. No surgery-related mortality was found in this report. No recurrence was observed during the 14.8-month follow-up period (range 3-24 months).

CASE REPORTS

Case 9

A 73-year-old man with a history of stroke was referred with a sacral Grade IV pressure sore. After debridement, a SGAP flap measuring 8 cm \times 12 cm based on one perforator was moved into the defect beneath the skin strip and inset without any tension. The donor site was closed primarily. The patient was discharged to a nursing home uneventfully 2 months later [Figure 2].

Case 29

A 72-year-old woman with intracranial hemorrhage with bed ridden developed a sacral grade IV pressure sore. After debridement, a 13 cm × 7 cm SGAP flap was designed to cover the defect. The SGAP was taken from the zone immediately adjacent to the defect with a tunnel to place the SGAP flap base centrally on the perforators. The flap was sutured into the flap and the donor site was closed primarily [Figure 3]. The patient remains recurrence-free at 8 months follow-up.

DISCUSSION

The common causes of sacral defects include pressure sores in paraplegic patients and infected pilonidal cysts in ambulatory patients.⁶ Delayed wound coverage of a sacral defect can cause progressive infections and wound pain. Therefore, surgical debridement and subsequent wound reconstruction remain the best treatments for most patients with a sacral defect.^{7,8} The gluteus maximus myocutaneous flap has been the most popular technique for closure of a sacral defect, thanks to its reliability and short learning curve for surgeons. However, this flap has several shortcomings.

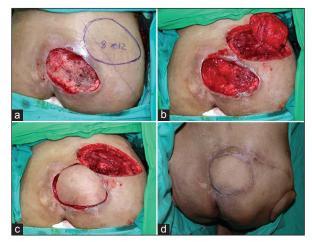


Figure 2. (a) Planning of an $8 \text{ cm} \times 12 \text{ cm}$ superior gluteal artery perforator (SGAP) flap for a $7 \text{ cm} \times 11 \text{ cm}$ sacral pressure sore coverage. (b) The SGAP flap was raised on one perforator. Note that the pedicle was still encased in its fibrous septum and the pedicle length did not exceed 1 cm. (c) The flap was tunneled into the defect from the flap donor site, and the thickness of subcutaneous fat provided ample length to tunnel the flap to the defect without further pedicle dissection. (d) The postoperative result 8 weeks after surgery

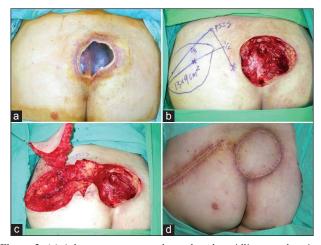


Figure 3. (a) A large pressure sore located at the midline sacral region. (b) Planning of an 13 cm \times 9 cm superior gluteal artery perforator flap for sacral pressure sore coverage. (c) The flap was raised on one perforator, which was still encased in its fibrous septum. (d) The postoperative result 6 weeks after surgery

It requires the surgeon to sacrifice a functioning muscle and has potential risks of walking instability in an ambulatory patient. Other disadvantages include a bulky appearance, limited distance of flap transposition and unnecessary blood loss when splitting the muscle. ¹⁻³ An additional problem is that, in some instances, the suture line of bilateral gluteus maximus myocutaneous flaps lies exactly at the maximal pressure point, resulting in frequent wound dehiscence during recovery.

In 1993, Koshima *et al.*⁴ found 20-25 perforators supplying the entire gluteal region and used gluteal perforator flaps to

cover sacral pressure sores. The beauty of this flap is that a large, safe flap can be raised unilaterally with minimal bleeding and will leave the muscle intact with little donor site morbidity. Verpaele *et al.*⁵ described the use of the SGAP flap, based on the perforator arising from the SGA that penetrates the gluteus maximus muscle, to reconstruct a large midline sacral defect. According to an anatomical study by Ahmadzadeh *et al.*,⁹ a mean of 5 ± 2 cutaneous perforators arising from the SGA can be found in the gluteal area. All these perforators are myocutaneous, passing through the gluteus maximus muscle or the gluteus medius muscle. The diameter of the superior gluteal perforators ranges from 0.6 to 1.0 mm.⁹ In our series, the mean size of the SGAP flaps was 80.1 cm² (range 42-168 cm²) and the maximum flap size supplied by one SGAP could reach 12 cm × 14 cm.

Deeply dissecting the perforator vessel from the muscle should obtain a pedicle length of 8.5-10 cm, giving the flap an impressive mobility and the possibility of covering large defects with a unilateral flap.⁵ A long pedicle can be raised if a lateral perforator is chosen, providing the flap a large arc of movement, which allows undamaged tissue to be used from a distant nontraumatized zone in certain cases. In small defects, they included the perforator vessels in the central portion of the flap design, and the flap was tunneled into the defect from the flap donor site. However, in some very large defects, the margins of the defect need to be included in the flap and the flap was inset by rotating.

In our series, however, we planned the flap to be 2 cm longer than the defect around the predetermined perforator vessels to make the SGAP flap as an ellipse fit the sacral defect. In this way, the most distal point of sacral defects is easy to be covered by the long side of the SGAP flaps. In contrast to previous studies, 5,7-12 we found no need to dissect more than 1 cm of length of the pedicle in our 30 patients. The dissection of the pedicle takes time and extreme care should be taken to avoid injuring the perforator vessel; pedicle dissection is the rate-determining step of the SGAP flap for closure of a sacral sore. For SGAP flaps with more than one perforator vessel, Verpaele et al.5 suggested that choosing a lateral perforator creates the longest possible pedicle to raise the flap from the distant nontraumatized zone and allows the surgeon to inset the flap with minimal torsion to the vascular pedicle.

We choose the medial perforator vessel as a pivot point to rotate the SGAP flap and found no need to skeletonize the perforator. Although rotating the SGAP flap applies torsion to the pedicle, none of the 11 rotating SGAP flaps failed because of a kink at the pedicle. No study has described SGAP flap death from pedicle torsion.^{5,7-12} Raising the SGAP flap adjacent to the defect does not influence wound

healing postoperatively after adequate wound debridement. Multiple perforator vessels will restrict flap mobility. A lower number of perforators allow the flap to move more easily and farther. Using only one perforator may preserve the viability of the flap. The additional perforator vessels do not increase the viability of the flap but provide additional stretch between the flap and defect. Ligation of the marginal perforators may release the tension without compromising the SGAP flap.

We think there are several reasons why our SGAP flaps could cover the sacral sores without needing a long pedicle dissection.

- 1. Because the SGA is often found one-third of the way down the line drawn from the PSIS to the greater trochanter, the perforator vessels will be close to the sacral sore;
- A longer flap design with an extra 2 cm length in the long side of template to make the SGAP flap as an ellipse ensures that the flap easily reaches the most distal point of sacral defect by tunneling or rotating;
- Minimal undermining of the defect and primary closure
 of the donor site places the defect toward the flap and
 decreases the tension between the flap and defect;
- 4. The bulk of subcutaneous fat of the SGAP flap provides additional length to increase flap mobility;
- The furthest end of the sacral sore could be covered by rotating the SGAP flap up to 180°, making a long pedicle dissection to advance the flap unnecessary.

However, there are several limitations in our study. First, our patients are not consecutive. The SGAP flaps are relative technique-demanded and meticulous dissection of the perforators is to achieve a good surgical result. The flaps are not preferred by most of the physician surgeons. Second, the major defect of this article is a small case number. We still need more experience of the modified flaps in the future.

CONCLUSION

Sacral sore management is a difficult issue in plastic surgery, and flaps must be chosen carefully. The SGAP flap provides a large, bulky, and safe fasciocutaneous flap to cover sacral pressure sores. The flap also has advantages of minimal blood loss, mild donor site morbidity, and preservation of muscle function. Like other perforator flaps, pedicle dissection needs a meticulous dissection technique to avoid damaging the perforator vessels, especially for inexperienced surgeons. Our study shows that deep pedicle dissection is unnecessary when the surgery involves an accurate indicating perforator, adequate flap size design, and correct selection of flap utilization between tunnel and rotation. We found that the SGAP flap elevation is

easier, faster, and safer. Because raising the SGAP flap is no longer a technique that demands a steep learning curve, we recommend the SGAP flap as a good alternative choice in the management of sacral sores which could not be covered with primary closure or local fasciocutaneous flap.

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CONFLICTS OF INTEREST

None declared.

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