

# Split Gluteus Maximus Turn-Over Flap For Sacral Pressure Ulcer

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## Abstract

Pressure ulcer is a common complication in bed-ridden patients. A case of infected sacral pressure ulcer in a 50-year-old paraplegic lady with transverse myelitis is presented. A split gluteus maximus turn-over flap was done to cover the exposed sacrum and the sacral pressure ulcer healed well.

## INTRODUCTION

Pressure sores are an ancient medical problem; even found during autopsies on Egyptian mummies. Tissue necrosis secondary to external compression, shear forces and friction cause pressure ulcers. Decubitus ulcers, derived from decumbere (Latin) meaning "to lie down", develop over bony prominences while in recumbent position. They occur in 3-5 % of patients in acute hospitalization and in 20-30% in chronic-care facilities. The incidence in the general population ranges from 5-25% and 5-8% per year in paraplegics. One-third of the patients have multiple pressure ulcers. Two thirds of the pressure sores occur in patients over 70 years<sub>1</sub>. Risk factors include increased or unrelieved pressure over bony prominences, immobility, increased moisture, impaired sensations, altered mental status, general ill health, old age, immunocompromised patients, nutritional deficiency, urinary/fecal incontinence, worn-out mattresses, hard trolleys/wheelchairs, the first two weeks of hospitalization and limited activity<sub>2</sub>.

## CASE REPORT

A 51-year-old lady presented with a chronic painless wound over her lower back of three weeks' duration. It was progressively increasing in size and depth; with foul swelling discharge soaking all her clothes and bed linen. According to the patient, she was all right about 2 months ago when she had sudden onset paraplegia along with loss of sensations in both lower limbs and urinary/fecal incontinence while doing some minor kitchen work. She was immediately rushed to civil hospital where she was managed by a medical specialist. Later on, she was referred to a tertiary care center where she was provisionally diagnosed to

be suffering from transverse myelitis; which was later on confirmed on MRI of her spine. She was managed conservatively and discharged-on-request with an advice for physiotherapy. She remained bed-ridden at home for one month during which she developed a progressively deteriorating sacral pressure ulcer. There was no fever, malaise, headache, nausea, vomiting, weight loss, spinal trauma or surgery. There was no past history of ischemic heart disease or hypertension. She was a known case of NIDDM on irregular medication and poor glycemic control.

General physical examination revealed an old bed-ridden lady with marked pallor and wasting. She was conscious, orientated with regular pulse of 100/min, BP 95/50 mm Hg and temperature 99oF. Local wound examination revealed a 5x6cm Grade IV pressure ulcer over the lower back with hypertrophied hyperpigmented margins, extending up to the sacrum and the floor was covered with yellow-black slough. Purulent foul swelling greenish discharge was soaking her clothes and bed linen. A grade 2 pressure ulcer was present on her buttocks.

**Figure 1**

Figure 1: Sacral Pressure Ulcer



Her neurological examination revealed wasting with hypotonia, hyporeflexia, power 0/5 in the lower limbs, sensory loss from L2 downward and palpable posterior tibial/dorsalis pedis pulsation. She was already catheterized and had patulous anal tone. She was managed in the intensive therapy centre. She was put on intravenous crystalloid infusions, intravenous broad-spectrum antibiotics, inotropic support, blood transfusion and enteral/parenteral nutritional supplements. Hourly posture changing was ensured. Her vital signs were monitored, intake/output was maintained and frequent bladder washes were done. The bowels were evacuated through regular enemas. The blood sugar level was managed through sliding scale control with frequent glucometer check-ups. Serial debridements/dressings were carried out. Extensive physiotherapy was started. The patient's general condition as well as her wound improved with conservative management. She was counseled for surgery and informed written consent for surgery as well as pre/postoperative photographs was taken. Mechanical bowel preparation was done to avoid any fecal contamination of the wound.

A split gluteus maximus turn-over flap was planned to cover the exposed sacrum. Under general anesthesia, the left gluteus maximus was exposed, split and mobilized over the inferior gluteal vascular pedicle. It was turned over and tunneled subcutaneously for inset into the sacral defect. Radical debridement of the ulcer was done. Lax surrounding skin was freshened up, mobilized and stitched primarily over the gluteus maximus turnover flap. Closed suction drainage of the wound was established.

**Figure 2**

Figure 2: Split Gluteus Maximus Turn-Over Flap



Strict postoperative lateral nursing was ensured to avoid any shearing damage to the flap. Oral fluids were started on the 3rd postoperative day. The sacral pressure ulcer healed well. The patient was discharged with strict compliance regarding her personal hygiene and hourly posture changes.

On a recent follow-up, her general condition had improved with power in both lower limbs 2/5 and absent sensation in left L4 to S2 dermatomes.

**Figure 3**

Figure 3: Healed Sacral Pressure Ulcer.



### DISCUSSION

External pressure more than arterial capillary pressure of 32 mm Hg and venous capillary closing pressure of 8-12 mm Hg impedes blood flow and results in irreversible tissue damage secondary to vascular occlusion and tissue anoxia. Tissue damage is directly proportional to external pressure and its duration. Constant external pressure over 70 mm Hg for 2 hours produces irreversible ischemic changes in animal model studies. The greatest pressure points in the supine patient include sacrum, heel and occiput; at 40-60 mm Hg, chest/knees at 50 mm Hg in prone position and ischial tuberosities at 100 mm Hg when sitting<sup>3</sup>. Therefore pressure ulcers occur on sacrum, ischial tuberosity, trochanters, heels, malleoli, pretibial area, patella, elbow and occiput. Pressure ulcer causes a tip of iceberg phenomenon with an inverted cone damage being more severe in muscle than in skin and subcutaneous layers. Pressure ulcers are usually infected with proteus, staphylococcus, streptococcus, pseudomonas, E.coli and bacteroides. Complications of pressure sores include autonomic dysreflexia, osteomyelitis, pyarthroses, sepsis, amyloidosis, recurrence, anemia, urethral fistula, renal failure and malignant transformation.

Barczak et al. staged pressure ulcers as: Stage 1: Erythema of intact skin > 1 hour after pressure relief. Stage 2: Blister/ulcer into dermis with or without infection. Stage 3: Subcutaneous destruction into muscle with or without infection. Stage 4: Involvement of bone or joint with or without infection<sup>4</sup>. Pressure ulcer can be prevented by early detection of patients at risk and Braden Scale is the most widely used tool for predicting development of pressure ulcers in medically and cognitively impaired elderly in acute-care, home-care and institutional-care settings. Risk assessment is done in six areas (Sensory perception, Skin moisture, Activity, Mobility, Nutrition, and Friction/shear). Score is assigned from one (highly impaired) to four (not impaired). Scores below 16 indicate high risk while scores above 16 indicate moderate to no risk<sup>5</sup>.

Avoidance of pressure is the single most important factor in prevention of pressure ulcer. Hourly posture changes when lying and half-hourly when sitting significantly reduces incidence. High-tech constant low pressure, alternating pressure mattresses, overlays and seating cushions are beneficial in high risk patients. Aims of management include adequate pressure relief, protection of vulnerable sites and treatment of underlying causes if possible. Poor prognostic factors include obesity, functional impairment, urinary/fecal incontinence, recurrent pressure ulcer, recent institutional

discharge, poverty and advanced age, living alone, illiteracy, co-morbidity, poor compliance and poor personal hygiene. Successful preoperative management includes adequate wound sepsis control (bacterial load <10<sup>5</sup>), positive nitrogen balance status, urinary tract infection control, urinary/fecal incontinence management (Foley catheterization & colostomy), extensive physiotherapy to minimize muscle spasticity and correction of nutritional/electrolyte deficiencies (serum albumin >3.5gm %). Stage I and II pressure ulcers are treated conservatively while stage III and IV resistant pressure ulcers require flap reconstruction. Conservative management includes antibiotics, serial dressing, physiotherapy, nutritional support, pressure relief and management of primary illness; but is not always successful. If a superficial ulcer does not reduce by 30% after two weeks, management should be reviewed. Our patient started improving on conservative management and reconstruction was planned because the defect was large.

Surgical management includes serial dressing/debridements, locoregional skin/myocutaneous flaps and osteotomy (total/partial) if osteomyelitis is present. In the 1930s, Davis, Greeley and Kostrubala pioneered plastic reconstructive procedures for covering pressure ulcers by using local fascia or muscle-fascia flaps. Reconstructive soft-tissue transfer techniques successfully achieve early wound closure, coverage of exposed hardware and decreased rates of chronic osteomyelitis. Skin graft is not a good option in pressure bearing areas because of doubtful viability and unpredictable outcome. Flap selection for back reconstruction is based on defect size, site, extent, tissue availability, previous surgery or radiotherapy. Random pattern skin flaps can be used for small sacral ulcers, are easy to elevate, muscle is not sacrificed and they can be re-elevated but lack bulk with limited rotational arc and doubtful tip vascularity. Tissue expansion is a useful technique providing sensate skin of similar color, texture and thickness. Myocutaneous/muscle flaps have good vascularity, withstand infections, obliterate dead space and are the best choice in paraplegics<sup>6</sup>. Gluteus maximus is a quadrilateral muscle (Type III flap) with dual blood supply from the superior and inferior gluteal arteries<sup>7</sup>. Turnover gluteus maximus<sup>8,9</sup>, sliding, segmental, split, bilateral V-Y myocutaneous advancement flaps are all excellent variants for coverage of sacral defects<sup>10,11,12</sup>. Split-turnover flap based on either of the vascular pedicles preserves most of the muscle in ambulatory patients. Keeping this in view, split-turnover flap was done in our patient who was recovering from her neurological deficit. Microsurgical free flaps

requiring plastic surgery facilitate to cover defects where local tissue is not available. Preoperative planning, single stage procedure, adequate debridement, dead space obliteration, meticulous hemostasis, tension-free wound closure and proper postoperative nursing care lead to excellent results. Complications include seroma, hematoma, wound dehiscence, infection and flap necrosis.

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