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REVIEW ARTICLE
Reconstructive

Postsurgical Pyoderma Gangrenosum Requiring Plastic Surgical Intervention: A Practical Review

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Summary: Pyoderma gangrenosum is a neutrophilic dermatosis characterized by immune dysfunction and pathergy. Thus, it is frequently seen in patients with underlying systemic illnesses or postoperatively. For the performance of the debridement or closure of the resultant defect, plastic surgeons are often involved in the care of pyoderma patients. However, both procedures may exacerbate the injury. Therefore, plastic surgeons must be familiar with the presentation of postsurgical pyoderma to avoid further damage and safely repair related soft tissue defects. A systematic search of the PubMed/Medline database was performed using the following keywords: "pyoderma gangrenosum" and "surgery." This online database search has identified 656 studies published between 1958 and 2022. Only reconstructed cases of postsurgical pyoderma gangrenosum were selected. Twenty-eight patients who developed pyoderma after dermatologic, plastic, orthopedic, cardiovascular, general, or obstetric surgery were included in this study. The average time to the PG presentation and diagnosis was 5.5 and 17 days, respectively. Diagnostic scoring tools were not used, and the diagnosis was primarily based on histopathology after repeated treatment failures. The patients received split- or full-thickness skin grafts, local, pedicled, and free flaps. An estimated 82.1% underwent skin grafting, whereas 42.9% underwent flap reconstruction. In addition, 21.4% got both the graft and flap. Accurate diagnosis of PSPG, prevention of further surgical injury, and timely medical management are vital for improving patient outcomes. Reconstruction can be performed, if required. However, despite the availability of different reconstructive techniques, there is no standard approach to the management of the PSPG. *(Plast Reconstr Surg Glob Open 2024; 12:e5505; doi: 10.1097/GOX.0000000000005505; Published online 19 January 2024.)*

INTRODUCTION

Incidence

Pyoderma gangrenosum (PG) is a noninfectious dermatosis characterized by ulcerative lesions with aseptic inflammation and neutrophil accumulation.[1] PG is an uncommon and morbid disease with a global incidence of three to five cases per 1,000,000[2] and a prevalence of 5.8 cases per 100,000 (USA) in the adult population.[3] PG can be observed across all age groups with a peak around ages 30–50[4–6] and patient populations with female preponderance.[2,7,8] It is a morbid disease associated with an increased risk of depression (21% PG versus 15% general population)[9] and decreased quality of life (Dermatology Life Quality Index score was 8.4 in PG versus 15 in the general population).[9]

Pathophysiology

PG was first described as "phagedenisme geometrique" more than a century ago, in 1916.[10] Nonetheless, to date, the definitive pathophysiology of PG remains elusive. The most commonly cited hypotheses revolve around neutrophil and monocyte aberrancies (chemotaxis, migration, bactericidal ability, and phagocytosis).[11] Nonetheless, the role of genetic mutations (PSTP1P1/CD2BP1) and abnormalities of the innate and adaptive immune system (increased expression of

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CD3, CD163, myeloperoxidase, TNF-alpha, IL-8, IL-17, MMP-2, MMP-9, and VEGF) has also been documented in PG.[12–14] Consequently, PG is usually observed in combination with other diseases characterized by immune dysfunction.[6,15–19]

Diagnosis

PG is considered a diagnosis of exclusion and, thus, constitutes a diagnostic challenge. According to the 2004 diagnostic framework, also known as the Su criteria, PG can be diagnosed if two major and two minor criteria are met.[20] However, Su criteria have yet to be confirmed.,[20,21] PARACELSUS Score is diagnostic for PG if the final score passes 10, irrespective of the fulfillment of the major (3) or minor (8) criteria.[22] In 2018, a validated diagnostic tool was created in Delhi.[23] Unlike other tools, it includes extreme pain as one of its minor criteria.[22] This tool is based on one major and eight minor criteria. The patient should be positive for one major and a minimum of four minor criteria to be diagnosed with PG. [23,22] (**See table, Supplemental Digital Content 1,** which displays current available clinical tools to diagnose PG. *The table is taken from Haag et al*[63] *with permission. Elsevier user license. Copyright © 2020 The Authors. Published by Elsevier Inc.* **http://links.lww.com/PRSGO/C959**.)

Despite the presence and efficacy of these scoring systems, they are not routinely used. None of the studies included in this review reported use of these scoring systems. Hence, PG is still (10%) misdiagnosed,[24] and there is an increasing concern in the literature regarding its overdiagnosis.[25,26]

Differential Diagnosis

Care should be taken to differentiate PG from some infectious and noninfectious disorders. In addition to more commonly seen infectious processes, such as the necrotizing fasciitis (NF) and erysipelas bullosum; sporotrichosis, blastomycosis, cryptococcosis can also mimic PG.[7,27,28-42] Some inflammatory (Henoch-Schönlein purpura,[43] granulomatosis with polyangiitis[44]); cancerous (mycosis fungoides,[45] anaplastic large-cell lymphoma[46]); and miscellaneous (iliac vein compression syndrome,[47] bromoderma[48]) disorders have similar presentation to PG, as well. One should also be able to set PG apart from pyogenic granuloma. Despite the similarities in the name, they are distinct entities.

Clinical course/findings, laboratory/biopsy results, and imaging can help with the differential diagnosis. As an example, unlike NF, PG lesions develop around postoperative day 7, skip lesions are common, and the pathergy phenomenon is present. Although both NF and PG patients might have a history of malignancy, autoimmune diseases are more associated with PG. As laboratories are usually not helpful in differentiating these two entities, wound cultures, imaging, and histology are used.[49-51,52] (**See table, Supplemental Digital Content 2,** which displays comparison of necrotizing fasciitis and PG. *The table is taken from Bisarya et al,*[52] *with permission. Creative Commons Attribution License. Copyright © 2011 The Author(s).* **http://links.lww.com/PRSGO/C960**.)

Takeaways

Question: The aim of this study is to provide a practical review of PSPG literature to plastic surgeons worldwide, to facilitate its timely recognition and prompt treatment.

Findings: This is a literature review, including 28 articles describing cases of PSPG after various surgical procedures. It provides the available up to date diagnostic criteria and treatment options for PSPG to plastic surgeons managing PSPG.

Meaning: Plastic surgeons should be aware of the characteristics of PSPG, such as its pattern of progression and symptoms, and consider reconstructive interventions cautiously to prevent further patient morbidity and mortality.

Pathergy Phenomenon

Pathergy is defined as hypersensitivity of the skin to minor trauma, resulting in the formation of nonspecific skin lesions.[53] Albeit not distinguishing for PG, this phenomenon is frequently encountered in patients with PG. As a result of the pathergy phenomenon, patients are prone to the development or aggravation of the lesions after trauma (electric current flow,[54] red tattoo dyes,[44] insect bites, biopsy,[18] surgery, etc.).[1]

PG Variants

The most frequently encountered PG subtype is the ulcerative or classic form[55] (Table 1). Classic PG most commonly develops on the lower extremity, usually in patients with inflammatory disorders.[19] Atypical or bullous PG lesions can manifest as plaques, nodules, vesicles, or bullae.[14,19,43,56,56] This PG variant is more associated with blood disorders, such as myelodysplastic disorders and monoclonal gammopathies, and blind loops.[19] Patients with IBD are also prone to develop parastomal PG. IBD is associated with pustular PG as well, mainly found on extensor surfaces.[14] A rapid expansion rate of 2 cm per day with central necrosis observed in 24–72 hours is typical for these PG types. On the contrary, vegetative or granulomatous superficial PG is characterized by slow progression and the absence of the accompanying systemic disease.[14,55]

For the first time, Cullen described postsurgical PG (PSPG) in 1924. Thus, PSPG is also called postoperative progressive gangrene of Cullen.[57] Although PSPG lesions are usually located on/around the incision due to the pathergy phenomenon, but may develop anywhere.[58] PSPG can also be the first presentation of PG, followed by further nonsurgical attacks.[47]

The rate of PSPG varies across different procedures. As an example, PG recurrence or exacerbation is more common after minor open surgical procedures [adjusted odds ratio (aOR), 8.65; 95% CI, 1.55–48.33] than major open procedures (aOR, 5.97; 95% CI, 1.70–21.00) and Mohs micrographic surgery/skin excision (aOR, 6.47; 95% CI, 1.77–23.61).[59]

Iatrogenic exacerbation is especially common in the case of PSPG, as PSPG symptoms can mimic postoperative pain, dehiscence, or infection, prompting unnecessary surgery. Consequently, plastic surgery is consulted

Table 1. Characteristic Features of PG Subtypes

PG Subtypes					
Subtype	**Incidence**	**Progression**	**Presentation (Main)**	**Presentation (Adjunctive)**	**Common Locations**	**Systemic Disease Association**
Classic/ulcerative	Most common (~85%)	Rapid	Painful ulcer	A violaceous undermined edge	Lower extremities	Inflammatory disorders (rheumatoid arthritis, inflammatory bowel disease, etc)
Atypical/bullous		Rapid	Painful superficial vesicles/bullae/nodules	Often coalesce and ulcerate	Head and neck upper extremities (dorsal surface of the hands, extensor aspects of the arms)	Hematological malignancy/lymphoproliferative diseases Blind loops
Pustular			Painful pustules	Background of erythema	Extensor surfaces legs upper trunk	Inflammatory bowel disease Hepatobiliary disease (vesiculopustular)
Malignant			Destructive ulceration	No violaceous edge	Upper trunk head and neck	No association
Superficial granulomatous/vegetative	Least common	Slow	Solitary superficial verrucous and ulcerative lesions/plaques	No violaceous edge	Trunk	Less frequently associated (<20%)
Parastomal		Rapid	Painful, often purulent ulcer	A raised violaceous edge	Around stomas	Inflammatory bowel disease (Crohn's > ulcerative colitis) + stoma
Postsurgical			Painful ulceration ± discharge	A violaceous edge Coalescing dehiscence	Peri-incisional Breast/abdomen (most common) lower extremity Chest	Less frequently associated

to perform definitive debridement or to achieve wound closure. Many reports in the literature demonstrate the omission of further debridements after the plastic surgeon recognizes the PG pattern.[45,46] However, PG patients might need reconstructive intervention by plastic surgery. Nonetheless, these interventions should be carried out meticulously, as PSPG can also affect/recur on the skin grafts[60,61] and flaps.[62]

Therefore, timely and correct diagnosis and management of PG by plastic surgeons is imperative to prevent further patient morbidity and mortality. Unfortunately, despite the availability of PG reviews[63] in the literature, a comprehensive review of the literature to facilitate the recognition of PSPG and available treatment options for plastic surgeons is still missing. Thus, in this study, we reviewed the peer-reviewed literature to aid in the timely diagnosis and management of PSPG by plastic surgeons.

METHODS

The following PubMed search was mapped: (pyoderma gangrenosum[MeSH Terms]) AND (surgery[MeSH Terms]) "pyoderma gangrenosum"[MeSH Terms] AND ("surgical procedures, operative"[MeSH Terms] OR "general surgery"[MeSH Terms]). This strategy identified 656 studies published between 1958 and 2022.

The inclusion criteria were case reports, case series, or correspondences published in English and describing definitively diagnosed cases of PSPG treated with reconstructive techniques. In addition, articles were included if they represented the hospital course in detail and reconstructive surgery was performed after the establishment of PG diagnosis. Moreover, cases of parastomal PG were excluded due to the irrelevance of surgical techniques to plastic surgery.

Data Extraction

Author name, specialty, country, publication year, study type, and level of evidence data were collected. Additionally, patient-related (age, sex, comorbidities, preceding surgery, follow-up, number of PSPG/all patients), PSPG presentation-related (location, presentation, time-to-diagnosis), PG diagnostic criteria, and treatment-related [first/final treatment, number of debridements, reconstructive techniques (graft, flap), time-to-healing] data were extracted. (**See table, Supplemental Digital Content 3,** which displays study- and patient-related data about the surgically treated cases of PSPG after dermatologic and plastic surgery. **http://links.lww.com/PRSGO/C961**.) (**See table, Supplemental Digital Content 4,** which displays summary of the PSPS-related information on the surgically treated cases of PSPG after dermatologic and plastic surgery. **http://links.lww.com/PRSGO/C962**.) (**See table, Supplemental Digital Content 5**, which displays a summary of (patient and study) data on surgically treated cases of PSPG after general, orthopedic, obstetrics, or cardiovascular/vascular surgery. **http://links.lww.com/PRSGO/C963**.) (**See table, Supplemental Digital Content 6**, which displays a summary of the PSPG diagnosis and treatment-related data after general, orthopedic,

obstetrics, or cardiovascular/vascular surgery. http://links.lww.com/PRSGO/C964.)

RESULTS

Twenty-eight articles published between 1997 and 2021 were selected. Twelve were published in the USA, three in Germany,[48,64,65] two in Canada,[66,67] Japan,[39] and Belgium,[55,68] and one in France,[69] Italy,[70] Australia,[71] New Zealand,[72] and Singapore.[73] The studies comprised seven correspondences/letters, one case series, and twenty case reports. The level of evidence was low (IV and V). These studies included 51 patients, of whom 28 underwent reconstruction after PSPG development. Most (19) patients were women. The mean age of the patients was 52 (median, 49.5) years. Four (14.3%) patients had diagnosed/suspected inflammatory bowel disease.[8,19,74] Another common comorbidity was rheumatoid[75]/unspecified[48,69] arthritis or positive rheumatoid factor.[76] Other concomitant pathologies with immune dysfunction were antiphospholipid antibody syndrome, idiopathic thrombocytopenic purpura, myelofibrosis,[34] acute/chronic renal failure,[34,75] factor XIII deficiency,[48] platelet dysfunction,[48] and hypothyroidism.[77] Other considerable comorbidities were cancers (breast, skin, lymphoma) and a history of abortions.[69,78] No underlying pathology could be determined for one of the patients with previous abortions.[78]

The median/mean time to the PG presentation and diagnosis was 5.5/13.9 and 17/19 days, respectively. The majority (23; 82.1%) of the patients received split- or full-thickness skin grafts. Additionally, 42.9% (12 of 28) of them underwent flap reconstruction. These techniques were used together in six (21.4%) patients.

Plastic Surgery

The characteristic findings of PSPG after breast surgery are its pattern of progression, 4 days to 6 weeks latency of symptom onset, sparing of the nipple-areolar complex, and rapid response to immunomodulatory medicines.[68] Usually, the early postoperative incision is typical. However, gradually, small dehiscences develop, which later merge.[68] Though pain can be variable, it is usually severe and present within the first postoperative week.[68]

All plastic surgery patients presented with PSPG within the first postoperative week (median, 5). (**Supplemental Digital Content 3**, http://links.lww.com/PRSGO/C961) On average, patients were usually diagnosed with PSPG on postop day 19. Meanwhile, they underwent a median of two debridements. One patient had multiple debridements.[79] One patient was intubated[80] and one had implant exposure.[68] Additionally, one patient plans to repeat the surgery due to cosmetic disfigurement related to post-PSPG scarring.[19] The most frequently performed reconstructive surgery was skin grafting (split[19,77,79,80] or full[66,70] thickness). Some of these grafts were combined with temporary homograft,[80] bilaminate neodermal matrix/Integra,[19] and local advancement flaps.[68] Patient follow-up ranged between 8 and 104 weeks. (**Supplemental Digital Content 4**, http://links.lww.com/PRSGO/C962.)

Dermatologic Surgery

Four articles described PSPG development in 7–210 days, mainly after skin cancer/lesion excision surgery.[8,69,74,81] The mean age of these patients was 66.8 years. The main presenting symptom was pain.[69,74,81] (**Supplemental Digital Content 3**, http://links.lww.com/PRSGO/C961.) Patients received skin grafts 2,[8] 4,[69] or 14[81] weeks after diagnosis. The latest patient first received radial left forearm free-flap four weeks after a definitive diagnosis. However, after flap failure, the patient underwent skin grafting as a secondary procedure.[81] One patient was admitted to the intensive care unit and developed pneumothorax.[81] (**Supplemental Digital Content 4**, http://links.lww.com/PRSGO/C962.)

Orthopedic Surgery

PSPG manifested in six patients after total knee arthroplasty,[65,73,76] knee arthroscopy,[49] intramedullary nail osteosynthesis,[64] or internal fixation.[65] (**Supplemental Digital Content 5**, http://links.lww.com/PRSGO/C963) One of the patients had a previous episode of PSPG.[72] Three patients developed a recurrence of PG. One patient experienced recurrence after appendectomy.[79] The other episodes occurred during the current treatment course due to inadequate immunosuppression.[64,82]

Patients presented with fever, local signs of inflammation, and erythema usually 5 days after the surgery.[49,64,65,73,76,82] Most patients underwent multiple debridements. Four patients developed local (large-scale wound breakdown,[76] soft tissue/joint capsular defect,[49] infectious hematoma,[65] and flap autologous skin partial loss[65]) and systemic (multi-organ failure[64]) complications. Five patients were treated with a skin graft and flap. The following flaps were used: medial gastrocnemius muscle,[49,73] free latissimus dorsi,[65,76] and rectus abdominis flap.[82] One patient had surgery with a combination of free parascapular, latissimus dorsi, and pedicled medial gastrocnemius muscle flaps after 2 weeks of steroid therapy.[73] (**Supplemental Digital Content 6**, http://links.lww.com/PRSGO/C964.) Other patients underwent flap surgery 2 weeks,[49] 2 days,[73] and 2.5 weeks[76] after definitive PG diagnosis.

General Surgery

We identified one article reporting a PSPG case after a general surgery procedure.[55] This patient developed fever and swelling 5 days after an inguinal hernia repair. (**Supplemental Digital Content 5**, http://links.lww.com/PRSGO/C963.) In the hospital, he was also diagnosed with low-grade lymphoma. Septic shock complicated his hospital course. Eventually, his groin defect was skin grafted with a positive outcome.[55] (**Supplemental Digital Content 6**, http://links.lww.com/PRSGO/C961.)

Gynecology/Obstetrics

All four patients developed PSPG after 2–7 days after C-section. Pain, erythema, swelling, and fever were observed in these patients. All were treated with skin grafts.[39,72,78,83] (**Supplemental Digital Content 5**, http://links.lww.com/PRSGO/C963.) One patient also required

a jejunostomy and a local flap.[72] (**Supplemental Digital Content 6**, **http://links.lww.com/PRSGO/C964**.)

Cardiac Surgery

These patients presented with a fever, cellulitis symptoms, drainage/pus, and ulceration. The hospital course of one patient was complicated by cardiovascular instability, respiratory insufficiency, acute renal failure, and the need for transfusion.[48] All patients eventually underwent tissue transfer. The first patient had a combination of omental transfer with split-thickness skin graft 2.5 weeks after cardiac catheterization.[34] The other transfers were latissimus dorsi flap[48] and pectoralis muscle flaps 2 weeks after steroid initiation.[48] (**Supplemental Digital Content 5**, **http://links.lww.com/PRSGO/C963**; **Supplemental Digital Content 6**, **http://links.lww.com/PRSGO/C964**.)

Parastomal PG

Parastomal PG is a substantial contributor to the morbidity of stoma patients.[84] It presents similarly to other parastomal diseases, such as stitch abscess, infection, contact, and irritant dermatitis.[26,85] However, the presence of undermined borders, rapid ulceration (does not start as erosions), and avoidance of sites priorly affected by PG are typical for parastomal PG. On the other hand, parastomal PG is less likely if the disease develops in a parastomal area without adequate wound care and cannot be controlled with adequate immunosuppressive therapy.[26] In addition to wound care, the treatment primarily consists of topical/systemic steroids and biologics. Additionally, dapsone, minocycline, and intravenous immune globulin were reported to be effective in parastomal PG.[86] Surgical intervention usually involves stoma closure, revision, or relocation.[86]

DISCUSSION

Recognition of PSPG is essential because it may lead to flap/tissue loss due to arterial thrombosis or as a part of the efforts to clear the "infected/necrotic" tissue.[87] In most articles included in this study, the diagnosis was made based on histopathology, persistently negative tissue cultures/stains, unresponsiveness to antibiotics/debridement,[88] and further deterioration. (**See table, Supplemental Digital Content 7**, which displays PG diagnostic criteria used in the included studies. **http://links.lww.com/PRSGO/C965**.)

No article referenced the use of PG diagnostic tools/scores. Usually, the diagnosis was made either after a dermatology[73]/plastic surgery/rheumatology[71] consultation or after a multidisciplinary discussion of the case.

Due to the risk of pathergy, some authors are against reconstruction with skin grafting or flaps.[87] However, others showed skin grafting could have positive outcomes with concomitant corticosteroid treatment until the donor and recipient graft sites are recovered.[55] Additionally, preoperative steroid or immunosuppressive (Infliximab, cyclosporine) therapy and weaning over 6 months is deemed useful in patients with a history of PSPG.[81,89,90] Despite not being recommended by Canzoneri et al, this option looks valid because skin grafts usually fail without immunosuppressive treatment.[91,92] Although almost complete graft take (90%) can be achieved without steroids, recurrence of PG and involvement of the donor site can be observed.[93] In the studies included in this review, local, pedicled, and free flaps have been used successfully to manage PSPG, usually with concomitant steroid treatment.

The steroid therapy (0.5–2 mg/kg/day) can be administered locally or systemically based on the symptom severity.[94,95] Using steroids may seem counterintuitive given steroids are associated with wound healing problems. However, in PSPG, the benefit of wound disease stabilization outweighs the possibility of steroid side effects. Furthermore, immunomodulators [tacrolimus, cyclosporin, azathioprine, minocycline, doxycycline, dapsone, thalidomide, and TNF-a inhibitors (etanercept, adalimumab, ustekinumab, infliximab)] and supplements, such as vitamin A, can be used to offset these adverse effects or as adjunctive or alternative treatment.[19,94–98] Among them, 4–5 mg/kg/day ciclosporin demonstrated similar efficacy as did 0.75–1 mg/kg/day oral prednisolone therapy.[96]

Other adjunctive treatment options include hyperbaric oxygen, negative pressure wound therapy, and intravenous immunoglobulin (IVIG).[66,70,79] Negative pressure wound therapy is hypothesized to modulate the inflammatory process and was reported to improve postoperative day 1 pain. However, it was exclusively recommended in cases with skin graft placement.[95] Hyperbaric oxygen is usually started with a preemptive diagnosis of NF/wound infection. However, it also helps facilitate PG wound closure.[70,71,77] The efficacy of the IVIG in PG is hypothesized to be related to immunomodulation by its effect on the macrophages, T, and B cells.[99] Schintler et al used IVIG (2g/kg x 1) in combination with high-dose steroid therapy (prednisolone 100 mg), V.A.C. Instil, and cyclosporin A (200 mg) to achieve stabilization of severe PSPG.[100] Patel et al combined IVIG (2g/kg over 3 days) with prednisone (80 mg), cyclosporin A (4 mg/kg/day), human cadaveric allograft, Integra, and skin graft with IVIG cover. However, split skin graft failure was still observed.[98] Furthermore, the use of dermal regeneration templates,[19,101] human cryopreserved placental membrane,[102] and structural placental allograft[81,102] were reported. However, large-scale studies are needed to evaluate their efficacy in the management of PSPG.

CONCLUSIONS

Due to the high morbidity and overuse of health care, every surgeon should be aware of the possibility of PSPG, despite its low incidence rate. Prompt recognition of PSPG, prevention of further surgical injury, and timely medical management are crucial in improving patient outcomes. A presentation of atypical and treatment-resistant wound infection should heighten suspicion for PSPG, especially in a patient with a history of immune dysfunction. Definitive reconstruction should ideally take place after stabilization of the PSPG with a 2-week (or more) course of steroid/immunomodulator therapy.

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