JNS SPINE

CASE REPORT

J Neurosurg Spine 32:285–291, 2020

Postoperative pyoderma gangrenosum after spinal fusion with instrumentation: case report

M. Harrison Snyder, BS,[1] Leonel Ampie, MD,[1] Vernon J. Forrester, MD,[2] JoAnne C. Wilson, MSN, RN, CNRN,[1] James H. Nguyen, MD,[1] Christopher I. Shaffrey, MD,[3] and Avery L. Buchholz, MD[1]

Departments of [1]Neurological Surgery and [2]Dermatology, University of Virginia Health System, Charlottesville, Virginia; and [3]Department of Neurosurgery, Duke Health, Durham, North Carolina

Pyoderma gangrenosum (PG) is a rare inflammatory dermatosis that is most often associated with inflammatory bowel disease, but which can occur as a pathergic reaction around surgical incisions. The authors report the case of a patient who developed postoperative PG over the course of several months after undergoing extensive spinal instrumentation between the T4 and iliac levels. This is only the second such case occurring after spine surgery to be reported. The authors additionally review the literature to characterize treatment approaches and outcomes for this condition. The case highlights a potentially severe adverse effect of surgery that can be difficult to recognize and causes delays in effective treatment. It also demonstrates the importance of multidisciplinary collaboration in the effective care of patients.

https://thejns.org/doi/abs/10.3171/2019.7.SPINE19708

KEYWORDS postoperative complications; pyoderma gangrenosum; spinal instrumentation; spine surgery; dermatology; infection

PYODERMA gangrenosum (PG) is a rare dermatosis often characterized clinically by painful ulcers that rapidly progress, as well as by neutrophilic infiltrate on histology.[3,5,6,9,40] Although it may be mistaken for infection, it is believed to be mediated by immune dysfunction.[1,3,5–7,32,36,40] It most frequently occurs in individuals between the ages of 40 and 60 years, with a majority of cases occurring with comorbid conditions such as inflammatory bowel disease (IBD), rheumatoid arthritis (RA), and other inflammatory arthropathies, as well as hematological malignancies, among others.[2,3,5,9,36] However, it has also been known to occur in wounds, including those from minor trauma and surgical incisions, a phenomenon known as pathergy.[4,10,13,15,16,18,25,36,39,42,45] These cases of PG are less likely to be associated with underlying disease, and are easily mistaken for wound infection, potentially delaying diagnosis and leading to morbidity through debridement, antibiotic therapy, and other wound care such as wound vacuum therapy and hyperbaric oxygen.[12–14,25,33,36,42–44] We describe the case of a patient who developed postoperative PG (PPG) after undergoing spine surgery.

Case Report

History and Examination

A 71-year-old woman with a previous history of scoliosis and spinal stenosis corrected with surgery presented after a "popping" sound emanated from her back while bending over, followed by intense low-back pain. More than a year previously, this patient had undergone T10–iliac posterior decompression, instrumentation, and fusion, with L4–5 and L5–S1 transforaminal lumbar interbody fusion using polyetheretherketone spacers, and L1–2, L2–3, L3–4, L4–5, and L5–S1 Smith-Petersen osteotomies. Radiographs obtained at the presenting visit showed bilateral rod fracture between the L5 and S1 screws (Fig. 1). Her medical history was notable for osteoporotic arthritis, colitis (diarrhea and hematochezia after taking diclofenac), lower gastrointestinal bleeding, and obesity. She had also undergone anterior cervical discectomy and fusion at C3–7 for anterolisthesis and foraminal/canal stenosis 6 years prior. She was conservatively managed for pain with analgesics and physical therapy, to which she responded well

ABBREVIATIONS IBD = inflammatory bowel disease; PG = pyoderma gangrenosum; PPG = postoperative PG; RA = rheumatoid arthritis; RCT = randomized controlled trial; SSI = surgical site infection; vac = vacuum-assisted closure; WBC = white blood cell.

SUBMITTED June 15, 2019. **ACCEPTED** July 31, 2019.

INCLUDE WHEN CITING Published online October 25, 2019; DOI: 10.3171/2019.7.SPINE19708.

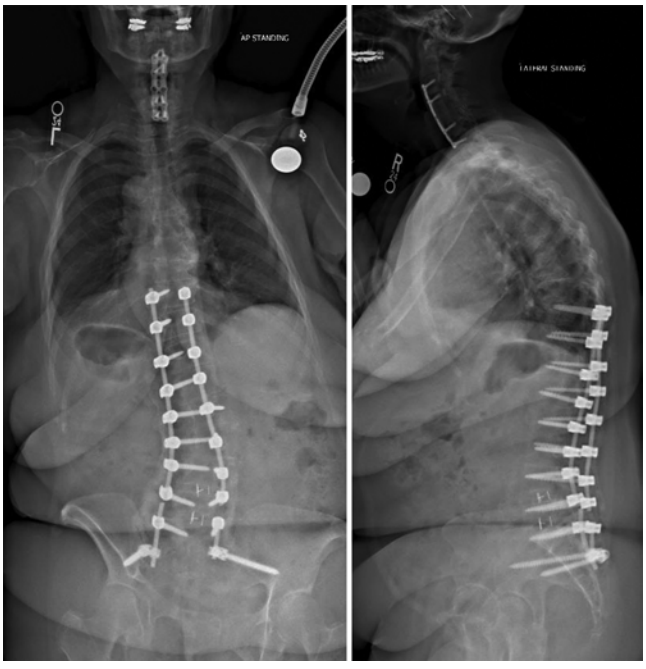

FIG. 1. Preoperative anteroposterior (**left**) and lateral (**right**) radiographs showing bilateral rod fracture between L5 and S1 screws.

initially. However, after several months she developed new symptoms, including low-back pain with radiation to the right hip and buttock, proximal thoracic pain, and a feeling of postural disturbance with anterior lean requiring use of a rolling walker, together suggesting progression of thoracic proximal junctional kyphosis. A revision was recommended, and the patient underwent extension to T4 of the T10–iliac fusion, including placement of bilateral iliac screws, instrumentation and accessory rod placement, and placement of antijunctional kyphosis tethers from T2 to T6–7 (Fig. 2).

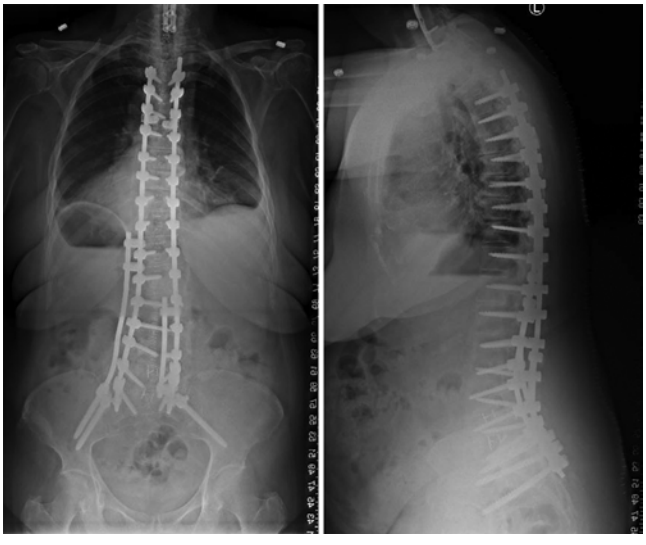

FIG. 2. Postoperative anteroposterior (**left**) and lateral (**right**) radiographs showing revision of instrumentation, including extension to T4 of the T10–iliac fusion, bilateral iliac screws, and antijunctional kyphosis tethers from T2 to T6–7.

FIG. 3. Incision at 38 (**left**) and 64 (**right**) days postoperatively, showing separate areas of dehiscence. Figure is available in color online only.

Postoperative Course

Approximately 1 month after surgery, new serosanguinous drainage was noted at the distal end of the incision. A possible retained stitch was removed via thumb forceps, and the drainage stopped; however, the incision resumed moderate amounts of serosanguinous drainage managed by the application of dry dressings (Fig. 3 left). By the next appointment, a new half-inch open area with light serosanguinous drainage was noted in the superior aspect of the incision between the scapulae. This was not erythematous or tender and the patient was afebrile (Fig. 3 right); a subsequent examination showed the open area unchanged from the previous appointment (Fig. 3 left). Later, a new ulcer near the middle of the wound opened with granulation tissue and slight bleeding, followed by two more 2-mm open areas thought to be areas of suture retraction. Other smaller areas opened, bringing the total openings to 5. At this point, dehiscence was suspected, and the patient underwent incision and drainage of the wound. The incision was closed with subcutaneous 2.0 Vicryl sutures and 2.0 nylon horizontal mattress sutures. During admission, blood counts and metabolic panels were within the reference ranges, including a white blood cell (WBC) count of 7990/μl.

The wound was stable for 2 weeks, after which the patient began again to notice serosanguinous drainage through the sutures that were still in place. Days later, several areas of the incision reopened (Fig. 4 left), and a left forearm intravenous access site (Fig. 4 right) and wrist arterial line site also began ulcerating; the patient was admitted again for a washout followed by placement of a wound vacuum-assisted closure (vac). Laboratory tests performed at this time again included unremarkable metabolic panels and WBC counts, but elevated C-reactive

FIG. 4. Reopening of the incision after incision and drainage (**left**), as well as a new intravenous site ulceration of the left forearm (**right**) on postoperative day 118. Figure is available in color online only.

protein (6.3 mg/dl) and erythrocyte sedimentation rate (80 mm/hr) were noted. MRI was performed and showed no concern for deep wound infection or significant fluid collection. During the admission, the patient was seen by wound care for wound vac education, and the patient was discharged.

After several weeks of wound vac therapy without improvement, the patient was referred to dermatology, the plastic surgery wound clinic, and allergy and immunology for further workup and management of the nonhealing incision, as well as the left forearm ulcer. Dermatology biopsied the left forearm site, revealing *Staphylococcus aureus* with a dense dermal infiltrate of macrophages and neutrophils. The patient was treated with a 15-day course of doxycycline. Wound clinic management consisted of cleaning, debridement, and redressing of the forearm ulcer and the back incision. During this management, the back wound continued to enlarge (Fig. 5 left), and the forearm ulcer did not heal (Fig. 5 right). The patient also developed multiple violaceous-erythematous nodules on

FIG. 5. Enlarging back wound (**left**) and forearm ulcer (**right**) on postoperative day 155. Figure is available in color online only.

FIG. 6. Cutaneous nodules with purple-red discoloration on the left flank (**left**) and left leg (**right**) on postoperative day 161. Figure is available in color online only.

the dorsal hands, upper extremities, lower extremities, and trunk (Fig. 6). Further consultation with dermatology led to suspicion for PG, and 6-week courses of topical clobetasol and oral prednisone were prescribed, resulting in significant clinical improvement (Fig. 7). As her dose of prednisone was tapered, the patient developed several new cutaneous lesions consistent with PG. She was ultimately transitioned to cyclosporine and her plaques resolved.

Discussion

PG is most frequently associated with underlying diseases, particularly IBD, autoimmune diseases, hemato-

FIG. 7. Back wound at last follow-up (postoperative day 339). Figure is available in color online only.

logical and solid tumors, and diabetes; however, PPG is notably different from classic PG because it is more likely to occur in patients with no underlying pathology (an identifiable underlying systemic illness was present between 18% and 50% of cases in other PPG literature).[14,16,21,23,26,33,35,42,43] Untreated PG can be life threatening, and patients with PG are at an increased risk of death by as many as three times compared to the general population.[5,9,18,21,24] Outside of mortality, PG may cause pain and adversely impact quality of life, predispose to secondary infection, cause disfiguring scarring, and recur.[36,38,41]

To our knowledge, this is the second case report of PPG occurring after major spine surgery, as well as the first in the neurosurgical literature. Ogon et al. describe the case of a 42-year-old man who underwent anteroposterior arthrodesis for L5–S1 spondylolisthesis; the patient developed dehiscence by postoperative day 4, with severe PPG developing by day 9.[29] This patient was successfully treated with steroids.

During our literature review, we identified 7 studies that involved more than 1 patient with PPG, excluding a number of case series of peristomal PG in patients with known IBD. The studies consisted of case series, literature reviews, and systematic reviews. The number of patients involved ranged from 2 to 136, and the years of publication ranged between 1992 and 2018. The findings from these studies are summarized in Table 1. Noteworthy findings included female predominance in all studies except 1 case series of 3 patients (33%), generally favorable outcomes (2 fatalities described, 1 clearly unrelated to the PPG disease process), as well as the use of steroids in some form in nearly all cases.

PPG has been documented to occur after breast, cardiothoracic, abdominal, gynecological, orthopedic, and head/neck surgeries.[15,19,42,43,45] It can also occur as a complication of other healthcare-related procedures, such as intravenous access (as seen in this patient), central lines, and other punctures.[22,26,45] The treatment of PPG is the same as PG in general; corticosteroid therapy and cyclosporine therapy are supported best by the literature.[8,34,36,37,42,45] The only randomized controlled trial (RCT) comparing these two treatments did not show a significant difference in efficacy, and Ormerod et al. concluded that selection between these two agents may be guided by side effects tailored to the patient's medical history.[31] Cyclosporine's side effect profile includes infection, hypertension, and nephrotoxicity, while systemic corticosteroids may cause weight gain, osteoporosis, hyperglycemia, hyperlipidemia, peptic ulcer disease, infection, and psychosis.[8,20,31,34] If patients are refractory to these treatments, other therapies such as mycophenolate mofetil, infliximab, tacrolimus, or plasmapheresis may be considered.[36,37] Infliximab is the best supported of these options; an RCT involving 30 participants showed a significant difference in healing between the infliximab group and the placebo group. Eventually, 29 patients ended up receiving infliximab, with 69% experiencing clinical benefit.[8,34] Overall, the outcomes are very good once the proper treatment is initiated, with a majority of patients experiencing healing of the lesion, often without recurrence.[14,16,23,26,33–36,42,45]

Throughout the PPG literature, the common theme is that the condition is almost always first misdiagnosed as a surgical site infection (SSI); this was consistent with our experience as well.[35,42,44,45] Indeed, PPG is a rare event after surgery. Patel et al. described a rate of 3 cases in 335 patients undergoing 395 breast reconstruction procedures, for a per-patient incidence of 0.9%.[35] The relative obscurity of PPG, combined with the fact that it often occurs in patients without other medical conditions typically associated with PG, means that the majority of surgeons will not consider it early on when faced with a nonhealing surgical wound.[14,16,23,45] In addition, the common occurrence of wound infection as a postoperative complication, as well as the consequences and costs of SSIs, makes suspicion of infection as the most likely cause of a nonhealing wound appropriate.[17,27,28,30,35] Nevertheless, PPG should appear on the differential diagnosis in the setting of a nonhealing surgical incision despite antibiotic treatment, as delays in the diagnosis of PPG can lead to disfigurement, distress, and additional costs for patients.[17,23,27,28,30,42,45] Unfortunately, the treatment regimens are somewhat in opposition to one another for SSIs and PPG. Whereas debridement is a standard treatment in progressive SSI, it actually may worsen PPG due to pathergy.[11,37,42,45] On the other hand, the immunosuppressive therapies indicated in PPG can predispose to wound infection and worsen existing infection, making topical therapy a safer choice in early suspected PPG.[36,37,41]

The occurrence of PPG is likely less of a consideration in neurosurgery due to the patterns displayed by the disease. PG most commonly occurs on the lower extremities; the trunk is less frequently involved, while involvement of upper extremities and the head/neck area is more rare, being affected in an estimated 7%–8% of cases.[5,18] The occurrence of PPG somewhat follows these trends, as the most commonly implicated surgeries include breast, cardiothoracic, abdominal, plastic, and gynecological procedures.[14,42,43,45] As stated previously, only 2 cases (including the current patient) of PPG after spine surgery have been described, and we are not aware of the existence of any case reports describing PPG after cranial surgery.[29] However, because spine surgery has become a significant part of neurosurgical practice, this rare complication should be considered when surgical wounds are slow to heal. Tolkachjov et al. suggest presenting clinical features that should raise suspicion for PPD: severe pain with a nonhealing wound or dehiscence, presentation around postoperative day 7, a violaceous border, rapid evolution, refractoriness to antibiotics and debridement, as well as negative wound culture.[42] An additional clue to the diagnosis and possible confirmatory test is pathergy, in which a patient develops ulceration around sites of known trauma such as invasive lines (similar to what occurred with the intravenous site in the case of our patient).[11,13,18] We recommend consultation with dermatology early during the course of treatment of a nonhealing surgical incision that is refractory to standard treatment, as the additional perspective and experience of specialists may assist in earlier diagnosis and guide treatment. Based on our review of the literature, we would recommend continuing antibiotics to cover possible current and future SSI while beginning a trial of topical corticosteroids should PPG be suspected.[9,14,31,34,36,37,41,45]

TABLE 1. Summary of studies on PPG involving more than 1 patient

Authors & Year	Type of Study	No. of Pts	Median Age in Yrs (range), % Female	Comorbid Conditions	Procedures Undergone	Treatment	Outcomes
Long et al., 1992	Case report & literature review	16	49 (16–68), 62.5% female	9 idiopathic, 3 IBD, 1 MCG, 1 prostate cancer	6 abdominal, 4 skin, 3 cardiac, 2 vascular (extremity), 1 breast	15 steroids (systemic, intralesional, or topical)	15 healed
Ouazzani et al., 2007	Case series	2	34.5 (21–48), 100% female	2 idiopathic	2 breast	2 systemic steroids	2 healed
Ferrandiz-Pulido et al., 2009	Case series	3	49.7 (47–54), 33.3% female	2 idiopathic, 1 IBD	1 breast, 1 cardiac, 1 abdominal	1 steroids, 2 steroids + chlorhexidine wraps + silver sulfadiazine	2 healed, 1 fatality (unrelated to PG, w/in 24 hrs)
Kikuchi et al., 2010	Case series	2	23 (18–28), 100% female	2 idiopathic	2 abdominal	1 steroids + skin graft, 1 cosmetic reconstruction	2 healed
Tolkachjov et al., 2016	Systematic review	140	50 (13–90), 72% female	90 idiopathic, 20 hematologic, 13 rheumatologic, 12 IBD, 4 solid tumor	41 breast, 38 abdomen, 22 lower extremity, 13 chest, 7 head/neck, 5 upper extremity, 3 miscellaneous (including cervical, anal, & vascular access site), 11 multiple sites	Treatment included systemic steroids in 110 of 129 cases (85%) w/ available data; see table included in original review study (Tolkachjov et al., 2016) for details on other agents used & treatment combinations	Not specified
Patel et al., 2017	Case series	3	49.3 (41–56), 100% female	3 breast cancer	3 breast	2 steroids + cyclosporine, 1 steroids + cyclosporine + graft	3 healed
Ebrad et al., 2018	Systematic review	31	56.5 (16–82), 58% female	4 diabetes, 3 IBD, 1 RA, 1 colon cancer	8 knee replacement, 7 trauma, 5 bone infection, 5 arthroscopy, 3 hip replacement, 1 spine,* 1 hand, 1 foot	22 steroids, 3 steroids + vac, 2 steroids + vac + hyperbaric oxygen, 2 steroids + polyclonal IG, 1 steroids + cyclosporine, 1 steroids + vac + hyperbaric + cyclosporine + azathioprine	30 healed, 1 fatality

IG = immunoglobulin; MCG = monoclonal gammopathy; Pts = patients.
* Ogon et al., as previously discussed.

PPG is a rare, but perplexing and potentially devastating, complication of surgery. While its initial presentation is incredibly similar to postoperative infection, features such as failure to heal after antibiotic treatment and debridement, as well as disproportionate pain and violaceous borders, may help to set it apart from the more common SSI. Early recognition and initiation of appropriate treatment may help to prevent complications of the disease, and can spare the patient additional procedures and treatments that could be otherwise unhelpful or detrimental.

References

1. Adachi Y, Kindzelskii AL, Cookingham G, Shaya S, Moore EC, Todd RF III, et al: Aberrant neutrophil trafficking and metabolic oscillations in severe pyoderma gangrenosum. **J Invest Dermatol 111:**259–268, 1998
2. Ashchyan HJ, Butler DC, Nelson CA, Noe MH, Tsiaras WG, Lockwood SJ, et al: The association of age with clinical presentation and comorbidities of pyoderma gangrenosum. **JAMA Dermatol 154:**409–413, 2018
3. Ashchyan HJ, Nelson CA, Stephen S, James WD, Micheletti RG, Rosenbach M: Neutrophilic dermatoses: pyoderma gangrenosum and other bowel- and arthritis-associated neutrophilic dermatoses. **J Am Acad Dermatol 79:**1009–1022, 2018
4. Berger N, Ebenhoch M, Salzmann M: Postoperative pyoderma gangrenosum in children: the case report of a 13-year-old boy with pyoderma gangrenosum after hip reconstruction surgery and a review of the literature. **J Pediatr Orthop 37:**e379–e383, 2017
5. Binus AM, Qureshi AA, Li VW, Winterfield LS: Pyoderma gangrenosum: a retrospective review of patient characteristics, comorbidities and therapy in 103 patients. **Br J Dermatol 165:**1244–1250, 2011
6. Braswell SF, Kostopoulos TC, Ortega-Loayza AG: Pathophysiology of pyoderma gangrenosum (PG): an updated review. **J Am Acad Dermatol 73:**691–698, 2015
7. Brooklyn T, Dunnill G, Probert C: Diagnosis and treatment of pyoderma gangrenosum. **BMJ 333:**181–184, 2006
8. Brooklyn TN, Dunnill MGS, Shetty A, Bowden JJ, Williams JDL, Griffiths CEM, et al: Infliximab for the treatment of pyoderma gangrenosum: a randomised, double blind, placebo controlled trial. **Gut 55:**505–509, 2006
9. Brooklyn TN, Williams AM, Dunnill MGS, Probert CS: T-cell receptor repertoire in pyoderma gangrenosum: evidence for clonal expansions and trafficking. **Br J Dermatol 157:**960–966, 2007
10. Callen JP: Pyoderma gangrenosum. **Lancet 351:**581–585, 1998
11. Callen JP, Jackson JM: Pyoderma gangrenosum: an update. **Rheum Dis Clin North Am 33:**787–802, vi, 2007
12. Contreras-Ruiz J, Kresch-Tronik NS, de la Cruz-Garcia MI, Mercado-Ceja S, Lozano-Platonoff A: Delayed diagnosis of pyoderma gangrenosum: a case study. **Ostomy Wound Manage 54:**32–36, 2008
13. Davis MDP, Alexander JL, Prawer SE: Pyoderma gangrenosum of the breasts precipitated by breast surgery. **J Am Acad Dermatol 55:**317–320, 2006
14. Ebrad S, Severyns M, Benzakour A, Roze B, Derancourt C, Odri GA, et al: Pyoderma gangrenosum after orthopaedic or traumatologic surgery: a systematic revue of the literature. **Int Orthop 42:**239–245, 2018
15. Esnault P, Dompmartin A, Moreau A, Caraes B, Leroy D: Recurring postoperative pyoderma gangrenosum. **Int J Dermatol 34:**647–650, 1995
16. Ferrandiz-Pulido C, Bartralot R, Fuente MJ, Heras C, Bassas P, Aparicio G, et al: Postoperative pyoderma gangrenosum: diagnostic value of 16s ribosomal RNA sequencing and review of the literature. **Clin Exp Dermatol 34:**598–602, 2009
17. Gabriel A, Gupta S, Orgill DP: Challenges and Management of Surgical Site Occurrences. **Plast Reconstr Surg 143 (1S):**7S–10S, 2019
18. Hadi A, Lebwohl M: Clinical features of pyoderma gangrenosum and current diagnostic trends. **J Am Acad Dermatol 64:**950–954, 2011
19. Hobman JW, Wright AL, Sharpe DT: Bilateral pyoderma gangrenosum complicating minimal access cranial suspension face lift. **Aesthet Surg J 26:**440–442, 2006
20. Hoes JN, Jacobs JW, Verstappen SM, Bijlsma JW, Van der Heijden GJ: Adverse events of low- to medium-dose oral glucocorticoids in inflammatory diseases: a meta-analysis. **Ann Rheum Dis 68:**1833–1838, 2009
21. Hoff NP, Gerber PA: Pyoderma gangrenosum associated with chronic polyarthritis. **CMAJ 183:**1746, 2011
22. Inan I, Myers PO, Braun R, Hagen ME, Morel P: Pyoderma gangrenosum after totally implanted central venous access device insertion. **World J Surg Oncol 6:**31, 2008
23. Kikuchi N, Hanami Y, Miura T, Kawakami Y, Satoh M, Ohtsuka M, et al: Pyoderma gangrenosum following surgical procedures. **Int J Dermatol 49:**346–348, 2010
24. Langan SM, Groves RW, Card TR, Gulliford MC: Incidence, mortality, and disease associations of pyoderma gangrenosum in the United Kingdom: a retrospective cohort study. **J Invest Dermatol 132:**2166–2170, 2012
25. Lindberg-Larsen R, Fogh K: Traumatic pyoderma gangrenosum of the face: pathergy development after bike accident. **Dermatology 218:**272–274, 2009
26. Long CC, Jessop J, Young M, Holt PJ: Minimizing the risk of post-operative pyoderma gangrenosum. **Br J Dermatol 127:**45–48, 1992
27. Magill SS, Edwards JR, Bamberg W, Beldavs ZG, Dumyati G, Kainer MA, et al: Multistate point-prevalence survey of health care-associated infections. **N Engl J Med 370:**1198–1208, 2014
28. Merkow RP, Ju MH, Chung JW, Hall BL, Cohen ME, Williams MV, et al: Underlying reasons associated with hospital readmission following surgery in the United States. **JAMA 313:**483–495, 2015
29. Ogon M, Sepp NT, Wimmer C, Behensky H: A surgical wound infection? **Lancet 356:**1652, 2000
30. Olsen MA, Tian F, Wallace AE, Nickel KB, Warren DK, Fraser VJ, et al: Use of quantile regression to determine the impact on total health care costs of surgical site infections following common ambulatory procedures. **Ann Surg 265:**331–339, 2017
31. Ormerod AD, Thomas KS, Craig FE, Mitchell E, Greenlaw N, Norrie J, et al: Comparison of the two most commonly used treatments for pyoderma gangrenosum: results of the STOP GAP randomised controlled trial. **BMJ 350:**h2958, 2015
32. Ortega-Loayza AG, Nugent WH, Lucero OM, Washington SL, Nunley JR, Walsh SW: Dysregulation of inflammatory gene expression in lesional and nonlesional skin of patients with pyoderma gangrenosum. **Br J Dermatol 178:**e35–e36, 2018
33. Ouazzani A, Berthe JV, de Fontaine S: Post-surgical pyoderma gangrenosum: a clinical entity. **Acta Chir Belg 107:**424–428, 2007
34. Partridge ACR, Bai JW, Rosen CF, Walsh SR, Gulliver WP, Fleming P: Effectiveness of systemic treatments for pyoderma gangrenosum: a systematic review of observational studies and clinical trials. **Br J Dermatol 179:**290–295, 2018
35. Patel DK, Locke M, Jarrett P: Pyoderma gangrenosum with pathergy: a potentially significant complication following breast reconstruction. **J Plast Reconstr Aesthet Surg 70:**884–892, 2017

36. Patel F, Fitzmaurice S, Duong C, He Y, Fergus J, Raychaudhuri SP, et al: Effective strategies for the management of pyoderma gangrenosum: a comprehensive review. **Acta Derm Venereol 95:**525–531, 2015
37. Reichrath J, Bens G, Bonowitz A, Tilgen W: Treatment recommendations for pyoderma gangrenosum: an evidence-based review of the literature based on more than 350 patients. **J Am Acad Dermatol 53:**273–283, 2005
38. Ruocco E, Sangiuliano S, Gravina AG, Miranda A, Nicoletti G: Pyoderma gangrenosum: an updated review. **J Eur Acad Dermatol Venereol 23:**1008–1017, 2009
39. Samuels LE, Kaufman MS, Kussmaul WG, Brockman SK: Pyoderma gangrenosum and myelofibrosis after coronary artery bypass grafting. **J Thorac Cardiovasc Surg 113:**795–797, 1997
40. Su WP, Davis MD, Weenig RH, Powell FC, Perry HO: Pyoderma gangrenosum: clinicopathologic correlation and proposed diagnostic criteria. **Int J Dermatol 43:**790–800, 2004
41. Thomas KS, Ormerod AD, Craig FE, Greenlaw N, Norrie J, Mitchell E, et al: Clinical outcomes and response of patients applying topical therapy for pyoderma gangrenosum: a prospective cohort study. **J Am Acad Dermatol 75:**940–949, 2016
42. Tolkachjov SN, Fahy AS, Cerci FB, Wetter DA, Cha SS, Camilleri MJ: Postoperative pyoderma gangrenosum: a clinical review of published cases. **Mayo Clin Proc 91:**1267–1279, 2016
43. Tolkachjov SN, Fahy AS, Wetter DA, Brough KR, Bridges AG, Davis MDP, et al: Postoperative pyoderma gangrenosum (PG): the Mayo Clinic experience of 20 years from 1994 through 2014. **J Am Acad Dermatol 73:**615–622, 2015
44. Wall LB, Stern PJ: Pyoderma gangrenosum. **J Hand Surg Am 37:**1083–1085, 2012
45. Zuo KJ, Fung E, Tredget EE, Lin AN: A systematic review of post-surgical pyoderma gangrenosum: identification of risk factors and proposed management strategy. **J Plast Reconstr Aesthet Surg 68:**295–303, 2015

Disclosures

Dr. Shaffrey reports direct stock ownership in NuVasive; being a patent holder for NuVasive, Medtronic, Zimmer Biomet, and SI Bone; and being a consultant to NuVasive, Medtronic, and SI Bone. Dr. Buchholz reports being a consultant to Medtronic, Siemens, and NuVasive.

Author Contributions

Conception and design: Buchholz, Snyder, Ampie, Nguyen, Shaffrey. Acquisition of data: Snyder, Ampie, Forrester, Wilson, Nguyen. Analysis and interpretation of data: Snyder, Ampie, Forrester, Shaffrey. Drafting the article: Snyder. Critically revising the article: Buchholz, Snyder, Ampie, Forrester, Wilson. Reviewed submitted version of manuscript: Buchholz, Snyder, Ampie, Wilson, Shaffrey. Approved the final version of the manuscript on behalf of all authors: Buchholz. Administrative/technical/material support: Wilson. Study supervision: Buchholz, Shaffrey.

Correspondence

Avery L. Buchholz: University of Virginia Health System, Charlottesville, VA. alb8jp@virginia.edu.