# An Acute Hemorrhage of a Chronic Ischial Tuberosity Pressure Wound Requiring Surgical Exploration: A Case Report

Patricia Stan[1], Cody Rasner[1], Joshua Levine[1], Miranda Considine[2], and Anthony Rezcallah[3]

[1]University of Minnesota Medical School Twin Cities Campus
[2]University of Minnesota Twin Cities Department of Surgery
[3]Minneapolis VA Health Care System

April 27, 2023

## Introduction

Pressure (decubitus) ulcers, also known as bedsores, are skin and soft tissue injuries that arise from constant or prolonged pressure exerted on the skin (Zaidi, 2022). An increasing number of people are affected by pressure injuries as our population grows and ages (Boyko, 2016). Pressure injuries frequently precipitate chronic wounds resulting in significant morbidity, mortality, and economic burden on both the patient and the healthcare system (Afzeli, 2020). Pressure injuries may develop in hospital and long-term care settings with orthopedic wards estimated to have the highest incidence of pressure injuries (Afzeli 2020). The reported prevalence of pressure injuries in long-term care facilities varies widely, ranging from 3.4-32.4% while hospital incidence rates range from 4% to 38% (Anthony 2019 & Afzeli 2020).

The etiology of pressure ulcers is multifactorial with risk factors including but not limited to neurologic disease, cardiovascular disease, and malnutrition (Zaidi 2022). Prolonged external pressure for as little as two hours can lead to the formation of a pressure ulcer (Zaidi 2022). Physiologic factors that incite pressure injuries include reperfusion injury and impaired lymphatic drainage (Boyko, 2016).

A number of complications may result from pressure ulcers with the most common being infection, which is often polymicrobial (Zaidi, 2022). The infection may spread to deeper tissues inciting periostitis, osteomyelitis, septic arthritis and the formation of sinuses from tissue loss (Zaidi 2022).

Stage I and II pressure ulcers are managed conservatively with appropriate wound care and elimination of causative factors, while more severe ulcers (stage III or IV) or ulcers with concomitant necrosis, osteomyelitis, or systemic infection may often require operative intervention. More severe pressure ulcers may require adjunctive procedures such as bedside debridement, negative pressure wound therapy, fecal/urinary diversion, and ultimately operative care. Since complication rates after flap reconstruction of pressure injuries are high, affecting 59% of patients (Bamba 2017), those who are poor surgical candidates should generally not undergo reconstructive procedures.

We present a unique case of a chronic ischial tuberosity pressure wound (ITPW) with multi-focal arterial and venous hemorrhage which required emergent exploration in the operating room.

## Case Presentation

*Pre-operative Course*

A 71-year-old male with a past medical history of a spinal cord injury (SCI) and resultant paraplegia with chronic tracheostomy, colostomy, suprapubic catheter and prior right above knee amputation (AKA) who

originally presented to the emergency department on Day 0 (D0) with altered mental status. On arrival, he was found to be newly anemic (Hgb: 9.7 from baseline of 12) with chronic decubitus ulcers. A stage 4 right-sided ITPW, which tunneled 10 cm toward the proximal femur, was present during initial examination (Figure 1A and B). Imaging revealed a chronic fluid collection in the left hip joint. The patient was on 81 mg aspirin prior to arrival and continued this regimen throughout his hospitalization but was not on therapeutic anticoagulation. The patient was admitted to the intensive care unit for sepsis and encephalopathy due to *C. difficile* infection, chronic osteomyelitis of the left proximal femur, and concerns for acute-on-chronic septic arthritis. He began a treatment course of fidaxomicin for the *C. difficile*infection and intravenous Zosyn for broad antimicrobial coverage given concerns for sepsis.

The patient underwent a left hip joint aspiration on D1. Blood cultures were negative and left hip synovial fluid cultures were found to grow pseudomonas aeruginosa. Orthopedic surgery performed irrigation and debridement of left hip joint with left proximal femoral resection (Girdlestone procedure) due to concern for acute on chronic septic arthritis of left hip on D3. He received one unit of pRBCs intraoperatively (and an additional unit on D5) and was started on intravenous levofloxacin for pseudomonas infection. Given the concern for possible worsening of the right ITPW, the general surgery team was consulted on D2. Upon initial evaluation by general surgery, no necrotic tissue or infectious concerns were visualized, and as such debridement was not indicated.

The wound care nurse team reassessed the right ITPW on D8 due to reports of intermittent bleeding from the wound since the patient's admission and concern for chronic hemorrhage due to a hemoglobin of 7.5 on D8 (down from 9.7 on admission). The dressings from the right ITPW were removed which caused the wound to bleed continuously despite reapplied pressure dressings. General surgery was subsequently consulted for acutely worsening bleeding from the chronic right ITPW. On exam, there was a 1-2 cm right opening over the ischial tuberosity that was persistently oozing with copious amounts of blood seen on the wound dressings. The wound depth was investigated by digital palpation and the origin of bleeding could not be found as the wound was deep and narrow, limiting visibility. The surgery team discussed conservative management with wound packing versus surgical intervention. The patient elected for operative intervention.

*Summary of Operation*

An incision was made at the opening of the ITPW distally toward the AKA stump. The wound was unpacked and burrowed deep in the thigh through several muscle layers. Some necrotic tissue was noticed without gross purulence. Three areas of arterial hemorrhage were visualized. One rather large pulsatile vessel traversed the posterior aspect of the acetabulofemoral joint and was presumed to be the medial circumflex femoral artery; this was suture-ligated with 0 PDS suture. Two other arterial vessels were suture ligated with #0 PDS suture in a figure-of-eight fashion. Approximately 8-10 additional bleeding veins were oversewn in figure-of-eight fashion with #0 PDS sutures. Various other smaller areas of hemorrhage were suture ligated with PDS suture as well. Electrocautery was used to achieve maximal hemostasis. The wound was packed with epinephrine-soaked lap pads and pressure was held for 15 minutes. Once the pads were removed from the wound, examination revealed several more areas of bleeding which were suture ligated and/or cauterized. Additionally, thrombin-soaked Gelfoam was placed in several areas that appeared to be oozing from the needle holes, and pressure applied. As the thrombin-soaked Gelfoam was removed, excellent hemostasis was achieved. At this point in time, Evista was applied to the base of the wound with tightly packed Kerlix gauze. ABD pads were placed superficially and, finally, a pressure dressing was applied. The estimated blood loss was 50 mL. Due to a borderline low hemoglobin level of 7.5 measured prior to the acute bleeding event, 2 units of pRBCs were transfused intraoperatively.

*Post-Operative Course*

Post-operatively, the patient was transferred back to the ICU for close monitoring for recurrent bleeding. The patient was continued on propofol sedation to minimize movement in order to reduce rebleeding risk. Mechanical DVT prophylaxis was utilized while pharmacologic options were held given the high risk of rebleeding. D8 post-operative hemoglobin was 9.3. The following day (postoperative day 1) the hemoglobin

was 8.9 without additional blood product administration. Post-operative packing was left in the ITPW for 48 hours following the procedure. Subsequent examination of the ITPW on D10 revealed a clean wound with no significant bleeding. The wound was redressed with Xeroform, normal saline moistened Kerlix, and covered with 2 ABD pads and a pressure dressing. Dressing changes were performed daily by the general surgery team. Daily examinations of the ITPW continued to show minimal oozing from the wound base and no evidence of active bleeding.

Hemoglobin remained stable between 8.2-9.2 with no signs of recurrent arterial bleeding. By D20, the wound exhibited signs of healing with good granulation tissue along the medial aspect, (Figure 2A and B). However, on D21, foul-smelling discharge was detected on ABD pads and a wound investigation revealed sloughing tissue and decreased granulation tissue. To address this finding, the dressing changes were increased from once a day to twice a day and switched to VASH moistened Kerlix. This change proved effective in improving the wound healing process.

On D28 wound assessment revealed a 17cm x 7.5cm x 3 cm wound with 40% necrotic tissue down the length of the center of the wound with 60% red moist granulation tissue along the sides with moderate sanguineous drainage. The patient was discharged from the general surgery team on D30, three weeks after wound exploration, with no evidence of rebleed and overall improving wound appearance. The patient was discharged with continued wound care at the patient's assisted living facility, and a general surgery clinic follow-up was scheduled 3 weeks after the discharge date.

The patient was not able to follow up in the general surgery clinic at the 3-week post-operative date. Unfortunately, he was subsequently hospitalized for dyspnea secondary to ventilator-associated pneumonia and COVID-19 pneumonia complications on 4/12. This hospitalization offered the general surgery team the opportunity to re-evaluate his wound status. Wound assessment of the right ischial tuberosity wound on 4/13 (D106) showed red, moist walls around the wound with good granulation tissue. Slough covered the deep base of the wound and over the boney prominence. There was moderate sanguineous drainage but no signs of local infection, and wound edges were intact.

**Discussion**

Arterial bleeding from a deep pressure ulcer is a rare occurrence. To our knowledge, no such cases have been reported in the available literature. The exact reason for this patient's acute bleeding event remains uncertain, necessitating further discussion and investigation of potential underlying causes.

With iatrogenesis being the fifth leading cause of death in the world, a potential complication of wound care must not be overlooked (Peer 2018). Although acute life-threatening hemorrhage from chronic pressure ulcers is not common, exploring the potential causes and treatment options is critical to reduce the morbidity and mortality of patients affected by chronic ulcers. This patient's acute bleeding event may have been precipitated by iatrogenic trauma during routine wound care. Standard wound care in deep wounds is frequently supplemented by the use of wooden adjuvants, such as cotton swab applicators. Medical personnel often blindly sweep, debride, and pack deep wounds with rigid objects, therein increasing the risk of damaging nearby structures. Bearing the nature of this patient's wound in mind, being approximately 10 cm deep (Figure 1B) and with a poorly visualized base, the aforementioned iatrogenic injury during routine dressing changes is not an unlikely cause of his acute bleeding complication.

Separately, this patient's acute bleeding event may have been a result of necrotic tissue creating friable areas in the vasculature and ultimately resulting in a multi-focal hemorrhage. The patient's ITPW harbored a chronic infection. Chronically infected wounds may increase the risk of hemorrhage particularly when the necrotic tissue is not appropriately debrided (Yoshikawa 2002). This patient arrived at the hospital with metabolic encephalopathy and septic arthritis and was ultimately found to have a pseudomonal infection of his ITPW. Provided this history, the potential for a chronic smoldering infection in the ITPW with resultant necrotic and friable tissue is high.

Although clinical suspicion is often sufficient to diagnose vascular injury with external hemorrhage, deter-

mining the exact source of external bleeding is difficult when the vessel cannot be visualized. Soft signs of hemorrhage include a history of prehospital blood loss, diminished pulse, proximity to the large vessel or bony injury, and ipsilateral neurologic deficit. Hard signs include paresthesia, pulselessness, paralysis, pain, pallor, and poikilothermia (the six P's). Hard signs indicate the need for urgent surgical intervention. Soft signs require further workup such as an ankle-brachial index, ultrasound, or angiography as the gold standard. In emergent acute hemorrhage, immediate bleeding control and rapid restoration of blood flow is the primary goal and extensive diagnostic workup should not delay treatment (Liu 2019).

When faced with acute hemorrhage, treatment options include conservative management, operative management, or endovascular repair. Operative treatment includes vascular suture or ligation, blood vessel prostheses, and grafts made of synthetic materials or the greater saphenous vein. Embolization, balloon dilation, and stent implantation are the main endovascular techniques (Liu 2019). Arterial ligation is suitable for most vascular injuries. A retrospective analysis found that embolization offers an effective and safe alternative to conventional operative care of traumatic arterial injuries (Whigham 2002). Although open surgical repair has been the gold standard for treating vascular trauma, the application of endovascular techniques may be more favorable in appropriately selected patients (Liu 2019).

In this case, the patient's source of bleeding appeared to be multifocal from mixed arterial and venous sources. If a single, clearly identified arterial source of bleeding was identified, endovascular techniques such as embolization may have been appropriate. However, endovascular treatments are not an effective treatment for multifocal venous bleeding, so open operative care was the treatment of choice.

During the operation, the patient's artery, likely the medial femoral circumflex artery, was ligated. Ligation of this artery was necessary to stop the arterial hemorrhage and avoid hypovolemic shock. The medial femoral circumflex artery is the main source of oxygenated blood to the head and neck of the femur. Disruption of this vessel is a well-known inciting factor for avascular necrosis of the femoral head and neck. Although compromising this patient's medial femoral circumflex artery was necessary to save the patient's life, this patient now has a risk of developing avascular necrosis. This potential complication must be addressed and investigated during follow-up in clinic. If the patient has evidence of avascular necrosis, such as increasing hip pain, an MRI will be warranted.

The definitive treatment for pressure wounds is often achieved via a skin flap or through primary intention. Although both of the options may be used to definitively treat this patient in the future, neither is appropriate in the setting of a chronic infection with evidence of necrotic tissue in the wound. Once the patient's infection has cleared, necrotic tissue from his wounds has been debrided, and evidence of granulation tissue is noted, discussion of definitive treatment options may be appropriate.

**Conclusion** :

The number of immobilized persons is increasing given the continued advent of life-prolonging medical interventions in a growing population. As such, the prevalence of pressure ulcers continues to grow. Here we report a unique case of acute hemorrhage of a chronic right ischial tuberosity pressure wound, which necessitated emergent operative exploration and suture ligation of 8-10 vessels to successfully achieve hemostasis. Acute arterial hemorrhage of a chronic pressure wound is a rare but potentially fatal complication, which may require emergent operative interventions. We highlight this potential complication to promote appropriate wound care and prompt recognition of hemorrhagic chronic pressure wounds with the goal of reducing morbidity and mortality.

**Abbreviations:**

AKA: above knee amputation

ITPU: ischial tuberosity pressure ulcer

IT: ischial tuberosity

**Declarations:**

*Availability of data and materials* :

The data for this case report are located at the Veteran Affairs Hospital, Minneapolis, Minnesota, United States.

*Acknowledgments* : Not applicable.

*Funding* : There was no financial support for this work.

*Author Information* :

University of Minnesota Medical School, Minneapolis, Minnesota

University of Minnesota Twin Cities Department of Surgery

Minneapolis VA Medical Center

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

*Competing interests* :

The authors declare that they have no competing interests.

**References**

1. Zaidi SRH, Sharma S. Pressure Ulcer. 2022 Aug 9. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan–. PMID: 31971747.
2. Boyko TV, Longaker MT, Yang GP. Review of the Current Management of Pressure Ulcers. Adv Wound Care (New Rochelle). 2018 Feb 1;7(2):57-67. doi: 10.1089/wound.2016.0697. PMID: 29392094; PMCID: PMC5792240.
3. Afzali Borojeny L, Albatineh AN, Hasanpour Dehkordi A, Ghanei Gheshlagh R. The Incidence of Pressure Ulcers and its Associations in Different Wards of the Hospital: A Systematic Review and Meta-Analysis. Int J Prev Med. 2020 Oct 5;11:171. doi: 10.4103/ijpvm.IJPVM_182_19. PMID: 33312480; PMCID: PMC7716611.
4. Anthony D, Alosoumi D, Safari R. Prevalence of pressure ulcers in long-term care: a global review. J Wound Care. 2019 Nov 2;28(11):702-709. doi: 10.12968/jowc.2019.28.11.702. PMID: 31721671.
5. Peer RF, Shabir N. Iatrogenesis: A review on nature, extent, and distribution of healthcare hazards. J Family Med Prim Care. 2018 Mar-Apr;7(2):309-314. doi: 10.4103/jfmpc.jfmpc_329_17. PMID: 30090769; PMCID: PMC6060929.
6. Thomas T. Yoshikawa, Nigel J. Livesley, Anthony W. Chow, Infected Pressure Ulcers in Elderly Individuals, *Clinical Infectious Diseases* , Volume 35, Issue 11, 1 December 2002, Pages 1390–1396, https://doi.org/10.1086/344059.
7. Liu JL, Li JY, Jiang P, Jia W, Tian X, Cheng ZY, Zhang YX. Literature review of peripheral vascular trauma: Is the era of intervention coming? Chin J Traumatol. 2020 Feb;23(1):5-9. doi: 10.1016/j.cjtee.2019.11.003. Epub 2019 Dec 25. PMID: 32014343; PMCID: PMC7049612.
8. Whigham CJ Jr, Fisher RG, Goodman CJ, Dodds CA, Trinh CC. Traumatic injury of the internal mammary artery: embolization versus surgical and nonoperative management. Emerg Radiol. 2002 Oct;9(4):201-7. doi: 10.1007/s10140-002-0226-7. Epub 2002 Aug 17. PMID: 15290563.

**Figure Legends** :

**Figure 1.** (A) Appearance of right ischial tuberosity pressure wound on D1 prior to operative intervention. (B) Inserted ruler showing depth of wound at 10 cm.

**Figure 2.** (A) General appearance of right ischial tuberosity wound measuring 17cm x 7.5cm x 3cm taken 20 days after operative suture ligation due to acute hemorrhage. (B) Inserted ruler showing the depth of the wound at 3 cm.