A Case Report of Trauma-induced Coagulopathy in the Setting of an Isolated Humeral Shaft Fracture

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Learning Point of the Article:

Trauma-induced coagulopathy and systemic inflammatory response syndrome are rare yet devastating sequelae of long bone fractures in at-risk populations necessitating increased surveillance and rapid response efforts.

Abstract

Introduction: Humeral shaft fractures are common orthopedic injuries often resulting from high-energy trauma in young patients and low-energy trauma in the elderly. Non-operative management has traditionally been the mainstay of treatment for isolated and low-energy humeral shaft fractures, with operative management reserved for severe cases often involving neurovascular compromise. This case describes a rare, yet catastrophic complication of a humeral shaft fracture where the patient developed trauma-induced coagulopathy (TIC), resulting in amputation of the affected extremity, systemic inflammatory response syndrome (SIRS), and ultimately death.

Case Report: A 60-year-old female patient with an isolated left humeral shaft fracture was originally treated with closed reduction and splinting. The patient returned 6 days later with a dysvascular left upper extremity and subsequently decompensated over the next 2 weeks secondary to TIC and SIRS. This resulted in a left transhumeral amputation, dysvascular right upper extremity, multiorgan failure, and ultimately death.

Conclusion: This report serves as a unique clinical example of rapid decompensation related to TIC and SIRS in the setting of a humeral shaft fracture. Managing orthopedic teams must be aware of these rare sequelae of humeral shaft fracture and be prepared to respond rapidly with surgical intervention and consultation of additional services to limit associated morbidity and mortality.

Keywords: Dysvascular limb, systemic inflammatory response syndrome, trauma-induced coagulopathy, fracture, thrombosis.

Introduction

Humeral shaft fractures are common orthopedic injuries that often result from high-energy trauma in young patients and low-energy trauma, predominantly ground-level falls, in the elderly [1]. Incidence rates of these injuries vary by age and sex, with women having a higher rate of injury than men and both sexes showing peak incidence in later stages of life [1, 2]. Nonoperative treatment has traditionally been the mainstay of treatment for isolated and low-energy humeral shaft fractures [1]. This type of treatment typically involves reduction and coaptation splint application in the acute setting, followed by the transition to a fracture brace as an outpatient 7–10 days post-injury [3]. The overall complication rate of nonoperative humeral shaft fracture management has been reported at 5.3%, with the most common complications being malunion (3.95–23.2%) and radial nerve palsy (8.8–20%) [4-6]. The present case describes a rare, yet catastrophic complication of a humeral shaft fracture where the patient developed trauma-induced coagulopathy (TIC), which resulted in amputation of the affected extremity, systemic inflammatory response syndrome (SIRS), and ultimately death. The patient's legal guardian provided consent for the case data to

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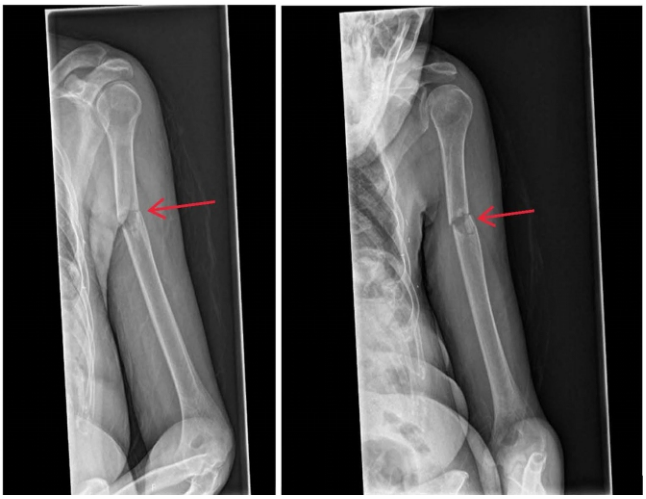

Figure 1: Sagittal (left) and coronal (right) radiographs of the initial injury demonstrating a left humeral shaft fracture. The red arrows depict the fracture site.

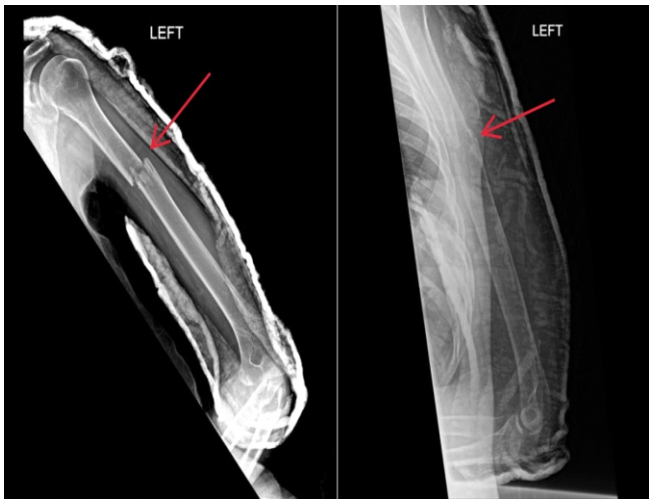

Figure 2: Radiographs following closed reduction and splinting of left humeral shaft fracture. The red arrows depict the fracture line.

be deidentified and submitted for publication.

Case Report

A 60-year-old African American woman with a medical history including epilepsy, intellectual disability, cerebral palsy, breast cancer status post left-breast mastectomy now in remission, chronic pancreatitis, and tobacco smoking presented to the emergency department (ED) with a left transverse humeral shaft fracture (Fig. 1). The injury was sustained after a seizure-induced fall from standing height. Neurovascular examination in the ED demonstrated palpable pulses and entirely intact motor and sensory function throughout the left arm. Based on this stable neurovascular status, a nonoperative approach of left humeral closed reduction and coaptation splinting was conducted by the orthopedic resident on-call (Fig. 2 and 3). Neurovascular examination was unchanged post-reduction and post-coaptation splint application. The patient was then discharged home from the ED with her mother (primary caregiver) with instructions to follow-up in the orthopedic surgery clinic the following week.

The patient returned to the ED 6 days later by her mother for increasing pain, left-hand swelling, and discoloration of the left hand. The patient was found to have a dysvascular left upper extremity (LUE), with a cool hand, swelling, lack of pinprick bleeding, and absent radial and ulnar pulses. On neurologic exam, the LUE was insensate including the axillary, radial, ulnar, and median nerve distributions. The patient was unable to form a composite fist, as well as flex, extend, adduct, or abduct her digits. Computed tomography (CT) angiography of the LUE was performed, which demonstrated intraluminal irregularities within the left axillary artery, abrupt partial and total occlusions along the left brachial artery, and the original transverse humeral fracture to be mildly displaced with moderate surrounding edema (Fig. 4). These arterial occlusions were noted to be proximal to the level of the fracture site. The patient's mother stated that over the prior 2 days, the arm had become dusky purple, swollen, and painful. She denied removing or augmenting the patient's dressing or splint in any way during that time. Emergent surgical intervention was performed by the vascular surgery team, consisting of a left brachial, radial, and ulnar artery thrombectomy, left brachial artery stenting, and axillary artery stenting. Open reduction and internal fixation of the humerus and prophylactic forearm fasciotomy including carpal tunnel release were performed by the orthopedic team during the same anesthesia event (Fig. 5). At the conclusion of this procedure, radial and ulnar Doppler signals were present at the level of the wrist.

Approximately 6-hours postoperatively, Doppler signals were lost in the affected limb and the patient was brought back to the operating room emergently. The vascular surgery team again performed left brachial, radial, and ulnar artery thrombectomies, with catheterization of the left radial and ulnar arteries, angioplasty of the left radial and ulnar arteries with standard balloons and left axillary artery angioplasty with stenting.

On post-operative day 1, the patient was transferred to the critical care unit following signs of undifferentiated shock with respiratory insufficiency, persistent rising lactate, thrombocytopenia, and anemia requiring packed red blood cell (pRBC) transfusion. On post-operative day 2, LUE pulses were again lost. Following this, the patient decompensated with

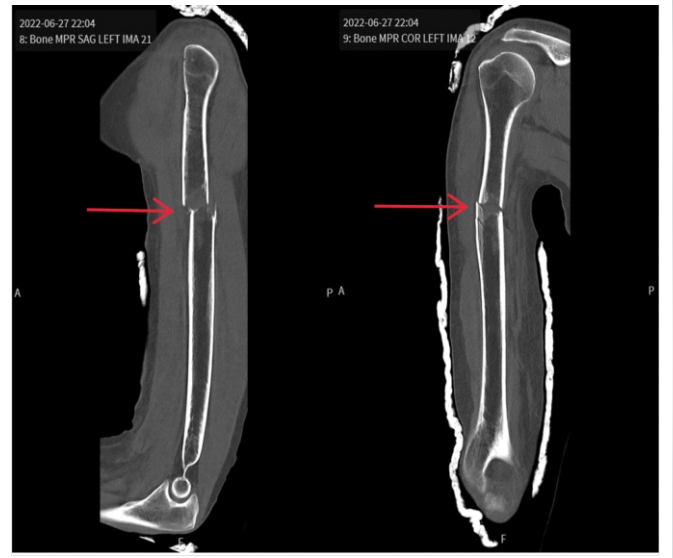

Figure 3: Sagittal (left) and coronal (right) computed tomography generated images of the left upper extremity following closed reduction and splinting. The fracture is well approximated. The red arrows depict the fracture line.

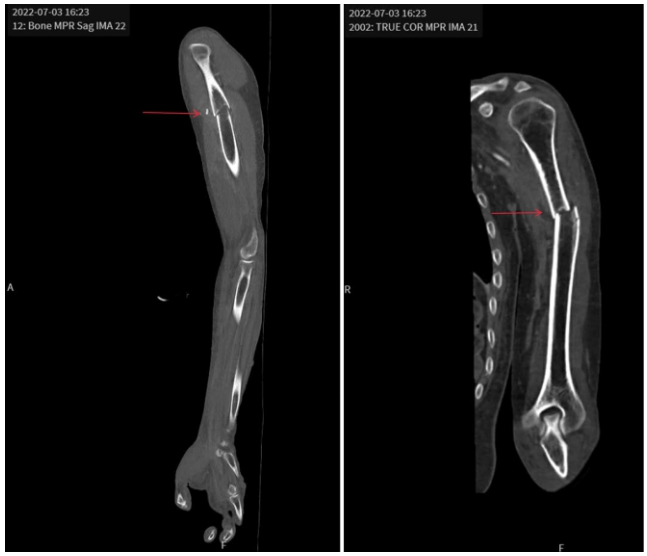

Figure 4: Sagittal (left) and coronal (right) computed tomography generated images of the left upper extremity on presentation to the emergency department with new onset loss of neurovascular function. The red arrows depict the fracture line.

progressive left arm necrosis, disseminated intravascular coagulation (DIC), septic shock, and multiorgan failure. She was transfused two units of fresh frozen plasma (FFP) and two additional units of pRBCs. At this point, the orthopedics and vascular teams conducted a left transhumeral amputation following family consent. The wound was closed with a plan to return to the operating room in 1–2 days for repeat debridement and closure.

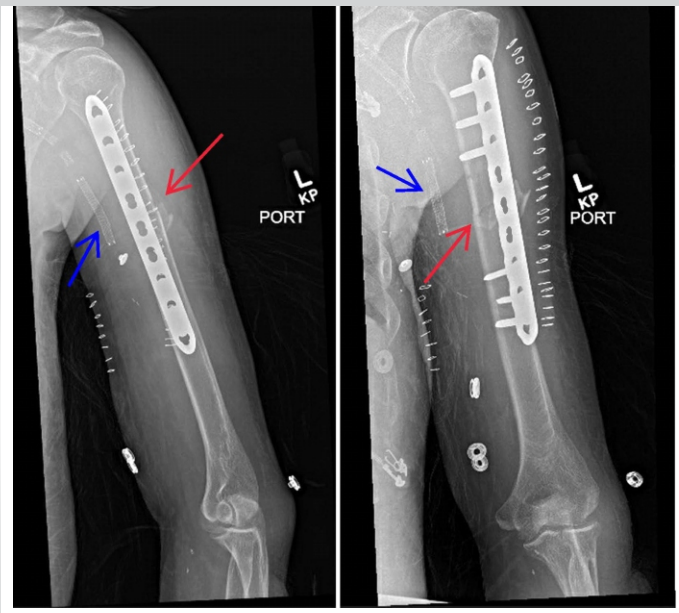

Figure 5: Post-operative sagittal (left) and coronal (right) radiographs following open reduction internal fixation of a left humeral shaft fracture. The fracture line is shown by the red arrows. The left brachial artery stent is shown by the blue arrow. A bone fragment is appreciated at the posterolateral aspect of the fracture line.

Four days after the initial operation, the patient developed right upper extremity pulselessness and ischemia with a dusky right hand, no radial artery doppler signals, and minimal signal from the ulnar artery. Goals of care were discussed with the family and a plan to limit care escalation, including a do not resuscitate order, was made.

On post-operative day 5, the patient was diagnosed with noninfectious SIRS with multiorgan failure. Due to the established goals of care, no further surgical intervention was performed. Over the course of the next few days, the patient experienced worsening conditions including acute renal failure, DIC, severe inflammatory shock, multiorgan failure, and respiratory failure. Nineteen days after the initial presentation, the patient died due to multiorgan failure and DIC.

Discussion

This case describes a rare, yet catastrophic complication following a closed humeral shaft fracture: TIC resulting in amputation and ultimately death. This case is unique in the literature due to the patient's rapid decline following her initial injury and treatment. Multiple case reports involving arterial thrombi following humeral shaft fractures exist in the literature; however, no such reports describe rapid clinical deterioration to this extent following closed management of a humeral shaft fracture.

To evaluate the etiology of the patient's coagulopathy and rapid clinical decline, the possible causative factors of acute trauma, iatrogenic injury at the time of fracture reduction, and late-onset

TIC must be evaluated. Based on clinical presentation and radiological data collected during the patient's initial presentation to the ED, acute trauma and iatrogenic injury are unlikely to be the primary causative factors. This conclusion is based on the patient's intact neurovascular status both before and after fracture reduction and splinting. Further, CT scans of the LUE (Fig. 3) demonstrate that the fracture was appropriately reduced, and the attending radiologist noted minimal signs of soft tissue trauma in their impression of the scan. When the patient returned to the ED with the neurovascular compromise of the LUE, a CT scan demonstrated multiple intraluminal arterial blockages, blockages extending proximally from the fracture site, and moderate edema (Fig. 4). Notably, the fracture was only mildly displaced without signs of direct impingement on surrounding neurovascular structures. Based on this presentation, the pathogenesis of the patient's dysvascular limb was unlikely to be caused by her acute trauma but instead by later onset coagulopathic sequelae stemming from a continued pro-inflammatory response multiple days after the initial injury and reduction [7]. After this point, the patient developed profound coagulopathy manifesting in DIC and hemolytic anemia requiring daily transfusions of pRBC and FFP. This clinical example is pathognomonic for late-onset TIC.

TIC, also described as acute traumatic coagulopathy, refers to a maladaptive impairment of hemostasis occurring after a traumatic injury [8]. TIC is divided into two recognized states of disease progression, early and late TIC, with early TIC involving a hypo-coagulable state which transitions into a hypercoagulable state seen in late TIC [8]. Complications include shock, hypoperfusion, a thromboembolic state, and multi-organ failure. The etiology of the condition is multifactorial but involves a mixture of endotheliopathy following trauma, fibrinolysis dysregulation, and platelet hypofunction [8].

Examples of vascular complications following humerus fractures exist in the literature with causative factors including the fracture itself as well as both operative and nonoperative treatment modalities. Cases involving coagulopathy and vascular injury following humeral shaft treatment can be stratified by arterial or venous involvement. Venous thrombosis following humeral fracture is uncommon but has been described in several reports [9,10]. Arterial involvement, depending on etiology, is comparatively rarer. Most studies discussing a relationship between humeral fracture and arterial blockage cite acute vessel impingement or coagulopathy as the cause of the blockage, with most cases being resolved with anti-thrombotic or surgical intervention [11-14]. Coagulopathy occurring days or weeks after the initial fracture is exceedingly rare with only a single published report with characteristics mirroring some aspects of the present case [15]. In this report, a patient who had previously healed from a nondisplaced humeral neck fracture and regained complete active range developed vascular claudication, paresthesia, and absent distal pulses 2 months after treatment [15]. In this case, the patient was treated conservatively and made a full recovery with no lasting complications. No other reports of late-onset coagulopathy or systemic inflammatory response in the setting of acute humeral shaft fractures have been reported at this time.

Conclusion

This case report of TIC following a closed humeral shaft fracture is an important addition to the literature as it represents an exceptionally rare, yet catastrophic complication, that orthopedic surgeons should be aware of.

Clinical Message

To reduce patient morbidity and enhance survival rates, a heightened awareness of complex neurovascular sequelae stemming from bone fractures is paramount in the early intervention of such pathology.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given the consent for his/ her images and other clinical information to be reported in the journal. The patient understands that his/ her names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Conflict of interest: Nil **Source of support:** None

References

1. Iglesias-Rodríguez S, Domínguez-Prado DM, García-Reza A, Fernández-Fernández D, Pérez-Alfonso E, García-Piñeiro J, et al. Epidemiology of proximal humerus fractures. J Orthop Surg 2021;16:402.

2. Ekholm R, Adami J, Tidermark J, Hansson K, Törnkvist H, Ponzer S. Fractures of the shaft of the humerus. An epidemiological study of 401 fractures. J Bone Joint Surg Br 2006;88:1469-73.

3. Sarmiento A, Kinman PB, Galvin EG, Schmitt RH, Phillips JG. Functional bracing of fractures of the shaft of the humerus. J Bone Joint Surg Am 1977;59:596-601.

4. Westrick E, Hamilton B, Toogood P, Henley B, Firoozabadi R. Humeral shaft fractures: Results of operative and non-operative treatment. Int Orthop 2017;41:385-95.

5. Zhang BS, Li WY, Liu XH, Wei J, He L, Wang MY. Comparative results of non-operative and operative treatment of humeral shaft fracturesf. Beijing Da Xue Xue Bao 2017;49:851-4.

6. Mahabier KC, Vogels LM, Punt BJ, Roukema GR, Patka P, Van Lieshout EM. Humeral shaft fractures: Retrospective results of non-operative and operative treatment of 186 patients. Injury 2013;44:427-30.

7. Kleinveld DJB, Hamada SR, Sandroni C. Trauma-induced coagulopathy. Intensive Care Med 2022;48:1642-5.

8. Duque P, Calvo A, Lockie C, Schöchl H. Pathophysiology of trauma-induced coagulopathy. Transfus Med Rev 2021;35:80-6.

9. Heyer JH, Parker RL, Lynch T, Parry T, Neviaser AS. Rate of venous thromboembolism after surgical treatment of proximal humerus fractures. Arch Orthop Trauma Surg 2021;141:403-9.

10. Sawyer GA, Hayda R. Upper-extremity deep venous thrombosis following humeral shaft fracture. Orthopedics 2011;34:141.

11. Thorsness R, English C, Gross J, Tyler W, Voloshin I, Gorczyca J. Proximal humerus fractures with associated axillary artery injury. J Orthop Trauma 2014;28:659-63.

12. Modi CS, Nnene CO, Godsiff SP, Esler CN. Axillary artery injury secondary to displaced proximal humeral fractures: A report of two cases ((Hong Kong). J Orthop Surg Hong Kong 2008;16:243-6.

13. Wenger JD, Olsson CJ. Acute limb ischemia after a proximal humeral epiphyseal fracture: Intraoperative findings of an illustrative vascular lesion. J Shoulder Elbow Surg 2011;20:e1-3.

14. Mahajan NP, Kumar GS, Sakhare V. Closed Humerus shaft and distal humerus fractures with associated brachial artery thrombosis in adult patients and its management-a case series. J Orthop Case Rep 2021;11:110-3.

15. Gallucci G, Ranalletta M, Gallucci J, De Carli P, Maignon G. Late onset of axillary artery thrombosis after a nondisplaced humeral neck fracture: A case report. J Shoulder Elbow Surg 2007;16:e7-8.

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