Septic Emboli Resulting From Severe Trauma: A Primer on Care

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Introduction Trauma nursing requires specialized knowledge and skills. This article describes the case of a patient who was involved in a motor vehicle accident and presented to the emergency department with hypovolemic shock secondary to a splenic laceration. In the hospital, the patient experienced prolonged hypotension.
Clinical Findings The patient sustained a variety of insults to the cardiovascular, respiratory, endocrine, and musculoskeletal systems. Microbiological data and laboratory test results did not reveal the defining characteristics of sepsis or systemic inflammatory response syndrome typical of trauma patients, making it challenging to identify the source of the sepsis.
Diagnosis The patient was diagnosed with nontraumatic cerebral septic emboli, a condition that is less common in trauma patients and more common in cases of endocarditis, septic thrombophlebitis, and central venous catheter infections. The condition has a 50% mortality rate if not detected promptly and appropriate treatment administered.
Outcomes The patient survived the 4-week hospitalization owing to timely management of his conditions by the health care team and their persistence in identifying the cause of his atypical sepsis.
Conclusion To provide adequate care to trauma patients, critical care nurses require specialized knowledge of this unique population. Trauma critical care nursing should involve hands-off communication, thoughtful review of laboratory test and imaging results, and engagement in interdisciplinary care rounds.
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CE 1.0 hour, CERP A

This article has been designated for CE contact hour(s). The evaluation tests your knowledge of the following objectives:
1. Describe the events in this case where embolic events should be considered.
2. Compare the definition and clinical presentation of sepsis in a trauma patient versus a nontrauma patient.
3. Explain the nurse's role to collaborate with team members and identify reasons for a trauma patient's continued need for vasopressor support.

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This article describes the complex case of a trauma patient with multiple medical problems who experienced sepsis. Under normal circumstances, persistent problems generally resolve with good critical care and management. However, this case involved several other severe, potentially lethal conditions that required astute observation and analysis of the clinical picture to ensure optimal outcomes. Critical care nurses must be prepared to care for a patient with a rare diagnosis or unusual combinations of conditions. Nurses and the entire health care team need to consider alternative explanations for unusual observations, especially when traditional interventions fail.

The case presentation uses modified CARE guidelines to answer the following questions: What is the likely explanation for continued shock symptoms in the early days of this patient's hospitalization? What can explain the relative stability of laboratory test results in the setting of persistent hypotension? Could this be an atypical presentation of sepsis or hemorrhagic shock, and did substance abuse complicate the clinical picture? The clinical observations offered clues to help critical care nurses recognize a potentially life-threatening condition.

Case and Clinical Findings

A 45-year-old man presented to a level I trauma center's emergency department (ED) after a motor vehicle accident. The patient, who was wearing a seat belt, was driving a van that rolled over during the accident and trapped the driver inside, requiring a 30-minute extrication. Upon the arrival of emergency medical services (EMS) personnel, the patient was found to be hypoxic, with an oxygen saturation of 76%. A left-sided chest needle decompression at the scene for a suspected pneumothorax resulted in no improvement in the patient's status. Because of continued hypoxia, the patient was intubated with airway protection before arrival in the ED. The patient's medical history included smoking half a pack of cigarettes daily for the previous 6 months, using marijuana, taking over-the-counter diet pills, and taking clonazepam for sleep; the patient had an allergy to penicillin. The patient lived with his wife and was employed as a flooring contractor. At the time of the accident, the patient weighed 83.5 kg (184 lbs) and had a body mass index (calculated as weight in kilograms divided by height in meters squared) of 23.62.

Upon arrival at the ED, the patient had hypotension, decreased left-sided breath sounds, and an oxygen saturation of 70% while undergoing mechanical ventilation with a fraction of inspired oxygen of 1. Repeat needle decompression was immediately performed for suspected hemothorax or pneumothorax, with minimal improvement in symptoms, followed by a chest tube thoracostomy. Chest tube insertion yielded an immediate rush of air and 400 mL of bloody drainage. Owing to clinical signs of hemorrhagic shock, an emergent blood transfusion was initiated. In addition to the primary and secondary assessments, a focused assessment with sonography for trauma examination was completed, which was positive for fluid in the right upper quadrant. The trauma team determined that the patient was sufficiently stable to undergo immediate initial panoramic computed tomography (CT). A summary of the CT results appears in Table 1.

The focused assessment with sonography for trauma and CT results indicated the need for emergent intra-abdominal surgery, and the patient was taken to the operating room for definitive management of the identified intra-abdominal injuries and hemorrhagic shock. In the operating room, the patient underwent a splenectomy for an avulsed spleen (grade V splenic laceration) and hemoperitoneum with a small mesenteric hematoma at the distal jejunum. During the procedure, the surgeon also repaired a small tear in the tail of the pancreas. In addition, a 19F round drain was placed intraoperatively owing to continued oozing in the peritoneum. After surgery, the patient was admitted to the trauma intensive care unit (ICU).

Additional imaging revealed multiple musculoskeletal injuries that were assessed and treated by the orthopedics

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Table 1 Summary of panoramic computed tomography (CT) results

Hospital day 1 (before surgery)

Head: No acute intracranial process. Extensive left frontal scalp laceration with a 10-mm irregular metallic foreign body as noted on physical examination.

Chest, abdomen, and pelvis: The endotracheal tube terminates in the right mainstem bronchus. A left thoracostomy tube terminates in the left apical pleural space. Mild pulmonary contusion is present in the middle and anterior lungs; right upper lobe laceration measuring approximately 5 cm in depth. Dependent atelectasis and/or aspiration is present in the lower lobes, with possible intermixed contusion and a small pneumothorax on the left side. Mildly displaced fractures of the left first through 12th ribs are present. In addition, there is a significantly displaced multipart fracture of the left proximal humerus, primarily involving the surgical neck. Soft-tissue gas tracks up the left neck. The spleen has a significant perisplenic hemorrhage that extends into the bilateral subphrenic spaces and along the left paracolic gutter into the pelvis. Active bleeding was suspected in the peritoneum. The spleen has an overall shattered appearance with a discontinuity at the superior aspect of the spleen and partial nonenhancement, consistent with a shattered spleen injury. Haziness at the tail is related to the adjacent splenic injury. Mild haziness along the adrenal glands reflects hemorrhage. Mildly displaced fractures of the left T12-L3 transverse processes and mildly displaced fracture of the right L5 transverse process are present. Soft-tissue gas appears within the left paravertebral musculature.

Hospital day 6

Head: Interval development of a right frontal cortical hypodensity and a right parietal similar hypodensity with no acute intrahemorrhage or extrahemorrhage identified and no midline shift. These findings were interpreted as a hypoxic-ischemic injury with generalized cerebral edema. In addition, the neurosurgery service was consulted for findings suggestive of septic emboli not visualized on initial CT.

Chest, abdomen, and pelvis: Air space consolidations in the right lower lobe are new and consistent with aspiration or pneumonia. Ground-glass attenuation in the lingula and left lower lobe suggests further aspiration or pneumonia.

and spine services. Cervical and lumbar transverse process fractures were managed nonoperatively. The patient's left femur was fractured and required a left proximal tibia traction pin. The patient was placed in balanced skeletal traction for initial management with an eventual operative femur repair plan. The patient had a left proximal humerus fracture and scapula injury that were managed nonoperatively with application of a sling. A nondisplaced right nasomaxillary fracture was also identified and addressed nonoperatively. Lacerations of the patient's scalp and left upper arm were repaired while he was in the ED. All of these conditions led to a complex, multiservice team approach to the patient's care.

Interventions

On hospital day 2, the anesthesia team performed an endotracheal tube exchange because of a constant air leak in the cuff that was noticed by the nursing team. An air leak was also observed in the chest tube collection device, with a drop in blood pressure and desaturation episodes throughout the day. The patient experienced persistent hypotension with episodes of hypoxia requiring initiation of norepinephrine as vasopressor support. Two bronchoscopies were performed in an effort to correct the hypoxia. Laboratory test results for the first 8 days of the patient's stay are listed in Table 2.

On hospital day 4, the patient remained persistently hypotensive despite dual vasopressor support with norepinephrine and epinephrine. These drugs stimulate β-adrenergic responses, causing inotropic effects such as increased heart contractility for rate and blood pressure. The patient's lactic acid level was 5.2 mmol/L, the white blood cell count was 7.0/μL, and other electrolyte levels were within normal ranges. An adrenal replacement was initiated as a treatment for suspected bilateral adrenal hemorrhage suggested on admission CT. A toxicology screen was obtained to explore whether the hypoxia was related to substance use that might be contributing to the newly elevated serum lactate level and acidosis without signs of infection and sepsis. A basic immunoassay was completed and was positive for amphetamines, fentanyl, benzodiazepines, and tetrahydrocannabinol. Although fentanyl and benzodiazepines were used for sedation and pain control, the results did not yield explanatory information.

On hospital day 5, vasopressor requirements decreased, but these agents were still needed to maintain a mean arterial pressure (MAP) greater than 60 mm Hg per hospital protocol. No apparent reason for the hypotension was evident. Metabolic acidosis persisted, and values improved, with a pH of 7.22 and a lactate level of 8.5 mmol/L. With an elevated lactate level, the arterial base deficit (–10) is a helpful prognostic indicator in trauma patients and is associated with increased mortality and significant injuries and complications.[1] Blood cultures grew gram-negative bacilli, as shown in the microbiological data in Table 3. A regimen of cefepime, metronidazole, and vancomycin was initiated.

Table 2 Laboratory test results

Analyte	Day 1	Day 2	Day 4	Day 5	Day 6	Day 7	Day 8
Sodium, mEq/L	140	143	143	144	143	143	146
Potassium, mmol/L	3.6	4.3	4.7	4.5	4.2	4.1	4.2
Chloride, mEq/L	111	117	115	112	113	115	116
Carbon dioxide, mEq/L	22	21	17	15	19	22	22
Blood urea nitrogen/ creatinine, mg/dL	18/0.9	20/1.1	27/1.2	30/1.1	32/0.7	44/0.7	44/0.7
Glucose, mg/dL	178	91	100	126	173	210	164
Adjusted ionized calcium, mmol/L	.90	1.10	1.07	1.12	1.16	1.24	1.08
Magnesium, mg/dL	1.9	1.8	1.9	2.1	2.3	2.3	2.3
Phosphorus, mg/dL	3.2	3.3	2.1	2.9	1.7	1.0	3.0
Lactic acid, mmol/L	1.8	2.6	5.2	8.5	4.5	2.5	1.6
pH	7.33	7.25	7.24	7.22	7.37	7.45	7.47
White blood cell count, cells/μL	11.1	7.5	7.0	7.0	11.9	19.2	21.8
Red blood cell count, cells/μL	4.02	3.06	2.89	2.89	2.51	2.59	2.68
Hemoglobin (arterial), g/dL	12.6	9.0	8.3	7.4	7.4	7.3	7.6
Platelet count, platelets/μL	181	175	219	120	59	46	47
Clotting factors PT/INR/aPTT, s						17.9/1.5/32.4	
Interpretation of heparin platelet antibody						–0.058	
Cortisol, μg/dL			>120				
Hemoglobin A_{1C}, %			5.3				

Abbreviations: aPTT, activated partial thromboplastin time; INR, international normalized ratio; PT, prothrombin time.

Table 3 Microbiological laboratory values and cultures

Hospital day	Blood	Sputum	Tissue
5	*Escherichia coli*	Heavy *Escherichia coli*	
10		Heavy *Stenotrophomonas maltophilia* Heavy *Acinetobacter baumannii*	
10		Bronchoscopy specimen: >100 000 CFU/mL *Klebsiella aerogenes* >100 000 CFU/mL *Stenotrophomonas maltophilia* 50 000-100 000 CFU/mL *Achromobacter species*	
11			Tissue anaerobes Heavy *Prevotella bivia* Tissue *Trichosporon asahii*

Abbreviation: CFU, colony-forming unit.

Diagnosis

On hospital day 6, the patient continued to receive 2 vasoactive agents and showed persistent hemodynamic instability. The nursing staff attempted to wean the patient off the medications, but the instability continued, and the MAP measurements fell below the minimally acceptable level. These efforts were communicated to the full patient care team. The cardiology service was consulted because of the development of atrial fibrillation with a rapid ventricular response in the absence of a history of cardiac arrhythmia. Cardioversion was performed, and an amiodarone infusion was initiated. Two-dimensional

echocardiogram was unremarkable, with an ejection fraction of 54%. A toxicology consultation identified persistent lactic acidosis of unknown etiology. Owing to persistently poor neurological examination results and hemodynamic instability as indicated by hypotension and laboratory test results, a repeat CT scan was obtained. The patient was suspected of having a hypoxic injury with right lower lobe aspiration pneumonia, with a heavy growth of *Escherichia coli* from sputum cultures noted.

On hospital day 8, owing to the consistently low blood pressure with vasopressor support, magnetic resonance imaging (MRI) of the brain was performed. The results showed multiple small infarcts with areas of contrast enhancement with mild mass effects. The differential diagnosis included embolic infarcts or traumatic nonhemorrhagic contusions. Normal flow voids were found in the carotid and basilar arteries. Small bilateral posterolateral hematomas were also evident on imaging.

On the basis of these findings, the neurosurgery service diagnosed the patient as having cerebral emboli. The emboli were determined not to be septic owing to the contrast enhancement within the small areas of the infarct. Heparinization was recommended and initiated. The radiology service recommended that if the patient was managed conservatively, surveillance CT examinations at close intervals should be performed to monitor the complex appearance of the left-sided brain masses and fluid accumulations.

After 5 weeks of inpatient posttrauma care, the patient was discharged home. Computed tomography of the head revealed a lytic process or area of destruction involving the high midline frontal skull. The health care team considered this finding as indicating either a malignant process or an infectious or inflammatory process. Further evaluation with nonemergent contrast-enhanced brain MRI was recommended.

A few days after discharge, the patient presented to the ED of a different hospital because of persistent headaches. He left the ED against medical advice. He followed up with a telephone call to the trauma service, and an appointment was made for 1 week later.

Outcomes

At the follow-up appointment 7 weeks after trauma, an MRI scan of the brain with and without contrast was performed and compared with the MRI scan obtained 1 week after trauma. The results were as follows:

1. A midline rim-enhancing frontal calvaria lesion with diffusion restriction involving the diploic space with extension to overlying scalp and an open wound in the soft tissue suggested a possible intraosseous abscess. There was no associated thrombosis of the superior sagittal sinus or an epidural empyema. No evidence of a cerebral or cerebellar intraparenchymal abscess was found.
2. Previously seen areas of diffusion restriction in the right middle frontal gyrus and right medial occipital lobe were resolved.
3. Interval resolution of mild edema was seen in the left dorsal pons.

Discussion

In this case of trauma due to a motor vehicle accident, the patient underwent a splenectomy soon after arrival at the hospital after persistent bleeding into the peritoneum. As expected, the patient experienced hemorrhagic shock after the initial injury that warranted surgical intervention. Fluid resuscitation was performed in the ED, the operating room, and the ICU. The patient's persistent hypotension required additional investigation and vasopressor support. Microbiological data revealed common causes of hypotension such as atelectasis and pneumonia, but symptoms of sepsis continued despite interventions to treat these 2 causes. Sepsis symptoms can have various origins, including a central venous catheter (CVC), respiratory failure, tissue hypoperfusion, and excessive work of breathing, which can cause muscle breakdown leading to lactic acid buildup.[1,2] After blood cultures came back positive, multiple antibiotics were begun to treat infective shock and persistent hypotension. A CVC was not placed in this patient, which ruled out the diagnosis of central catheter–associated bloodstream infection.

Systemic inflammatory response syndrome (SIRS) is an exaggerated defense response of the body to a harmful stressor such as infection, trauma, surgery, acute inflammation, ischemia, or malignancy.[1] Sepsis is a systemic response to infection. Sepsis is a significant cause of morbidity and mortality in critically ill patients, although it is not caused exclusively by gram-negative bacilli. Systemic inflammatory response syndrome and sepsis share a common inflammatory pathway. The process is a complex interaction of humoral and cellular responses, cytokines, and complement pathways in a balance between proinflammatory and anti-inflammatory responses.[2] Alteration

of coagulation occurs, causing endothelial injury that triggers the coagulation pathway, and with anti-inflammatory mediators inhibited, widespread microvascular thrombosis results.[3]

Systemic inflammatory response syndrome is identified by the presence of 2 of the following criteria: temperature greater than 38 °C or less than 36 °C, heart rate greater than 90/min, respiratory rate greater than 20/min or Pco_2 less than 32 mm Hg, or a leukocyte count greater than 12 000/μL or less than 4000/μL. A new clinical risk stratification score has been demonstrated to predict adverse outcomes. The quick Sequential (Sepsis-related) Organ Failure Assessment score uses blood pressure, respiratory rate, and the Glasgow Coma Scale to predict death and ICU transfer of patients with suspected sepsis. A quick Sequential (Sepsis-related) Organ Failure Assessment score before ICU admission is more accurate than SIRS in predicting mortality but is less sensitive than SIRS in predicting clinical deterioration.[4]

The Surviving Sepsis Campaign, which was developed in 2002 by the Society of Critical Care Medicine along with the European Society of Intensive Care Medicine and the International Sepsis Forum, has evolved in its recommendations to maximize patient survival of sepsis.[1,5] The Campaign recommends optimal fluid replacement and selection, dose titration, and escalation of vasopressor therapy to achieve an adequate MAP and identify and control the primary source of the sepsis.[1] The health care team supported the patient with fluid resuscitation, vasopressor therapy, and antibiotics for the initial 3 days. Systemic inflammatory response syndrome requires more advanced markers than traditional C-reactive protein (CRP), erythrocyte sedimentation rate, and white blood cell count. Procalcitonin, lactate, and CRP are the most frequently used biomarkers in sepsis.[6,7] The usefulness of procalcitonin was first discussed in 1997, and bacterial, fungal, or parasitic infections are associated with increased procalcitonin serum levels.[3,7] In the case discussed here, CRP and procalcitonin levels were not measured. However, these additional markers of critical illness can help predict the severity of illness and mortality.[7] Sepsis in patients with a life-threatening disorder is associated with a poor prognosis, whereas the prognosis in a previously healthy person is good.[8] The overall mortality rate for gram-negative bacteremia is 33% in North America.[9] When septic shock occurs, the mortality increases to 50% to 60%.[6] In polytrauma patients, septic complications are the predominant cause of late death, with overall mortality of 45%. In addition to CRP and procalcitonin, lactate clearance in persistent hypoperfusion helps differentiate patients on a septic course and the evolution of SIRS.[10]

Sepsis symptoms can have various origins, including a CVC, respiratory failure, tissue hypoperfusion, and excessive work of breathing, which can cause muscle breakdown leading to lactic acid buildup.

Although the use of appropriate synergistic antibiotics to counter the infecting organism is helpful, the overwhelming influence of host factors in predicting the outcome suggests that prevention and early treatment are the best means of reducing mortality.[11] On day 6, the health care team decided to perform repeat imaging of the patient to determine sources of infection. The finding of nontraumatic cerebral septic emboli (CSE) on head CT (vs CSE as a direct result of trauma to the head) explained the continued shock symptoms requiring vasopressors and respiratory support despite aggressive treatment with antimicrobial agents.

Generally, a septic embolus obstructs a blood vessel as a result of an infected thrombus from a distal infection.[12] In a multiple-trauma patient such as in this case, this cause of sepsis should be suspected whether or not infection is present. Septic emboli are an underreported complication, typically because of 2 insults that occur. First, there is an early embolic or ischemic insult due to vascular occlusion. Second, the infection leads to inflammation and abscess formation. Treatment consists of long-term antibiotic administration and, if possible, source control.[12] The sequential CT scan on day 6 identified hypoxic-ischemic injury with generalized cerebral edema, as noted in the CT report.

Case reports and research on CSE in infective endocarditis (IE) show the origin to be dislodgment of cardiac vegetation followed by vessel occlusion, which results in infarct or ischemia.[13] When the patient continues to show hypotension and possible sepsis without a source, IE should be considered. Cerebral artery occlusion from infarct or transient ischemic attacks accounts for 40% to 50% of central nervous system complications in IE. More than 40% of the occlusions affect the middle cerebral artery.[14] More common causes of CSE have been associated with IE.[14] Infective endocarditis has been associated

with an ST-elevation myocardial infarction and is thought to result from a coronary embolism that leads to an obstruction or coronary artery compression due to abscess formation. Even with advanced techniques, in emboli related to IE, outcomes have not improved.[15] Although coronary artery septic embolism with resulting myocardial infarction is rare, occurring in only 1% of patients diagnosed with IE, the complication with consequent intracranial hemorrhage can result in death even with aggressive interventions.[16] Another possible source of sepsis in patients who are monitored is the CVC placed for continuous blood pressure monitoring.[16]

The primary risk factor in neurological complications associated with CSE is the absence of timely and appropriate antibiotics.[12] Early antibiotic therapy and better critical care interventions are needed to avoid complications from septic emboli, which can appear in the lungs and brain as well as solid organs such as the spleen and extremities. Clinical signs and symptoms can vary widely, with early symptoms including vascular occlusion of the cerebrum or bowel or myocardial infarction. Late complications include mycotic aneurysms and abscesses.[17] Multiple trauma with risk for CSE is not well documented in the literature, but similar mechanisms of CSE in other conditions can be applied to these patients.

Pertinent laboratory test results and invasive monitoring, such as use of a pulmonary artery catheter, were not used to manage this case. The Surviving Sepsis Campaign recommends against the routine use of a pulmonary artery catheter.[18] However, owing to the challenges of maintaining an adequate MAP with vasopressors, the patient may have benefited from its use.[19] Laboratory tests such as for CRP, erythrocyte sedimentation rate, and procalcitonin, which serve as predictors of mortality, were not performed. In conjunction with standard critical care measures, these tools can help identify what may be hindering a patient's recovery.

Trauma nursing requires specialized evidence-based knowledge and skill. An integrative review by Walter and Curtis[20] analyzed the trauma nurse's varied responsibilities. After reviewing 1000 articles, the authors noted a lack of consistency in qualification requirements, orientation, and impact of the specialist trauma nurse. Also challenging is the care of trauma patients in the ICU. Continuing education requirements were not addressed in the review and are critical to the care of trauma patients in the dynamic environment of the ICU.

This case offers an excellent opportunity to review what was done well and what to do differently when a patient does not improve with interventions. Critical care nurses routinely coordinate, deliver, and monitor patient care. They are essential to the flow of information from the patient to the overall health care team. A study that identified variation in nurse documentation and communication among 8 different medical-surgical nursing units in the United States found that nurses rarely engaged in interdisciplinary communication.[21] These issues can compound serious and undetectable clinical errors. The researchers concluded that nursing units should establish a standardized format in communicating the patient's plan of care to members of the health care team. Hands-off communication and medical record review are essential elements of the nurse's role. In addition, the study authors recommended using team meetings to discuss the patient's plan. The interdisciplinary team approach focuses on the multifaceted aspects of patient care. The critical care nurses who cared for the patient in the present case were instrumental in identifying important issues such as the airway complication and hemodynamic instability.

Many different types of clinicians, ranging from nurses to physical therapists, should have specialized knowledge of trauma and should receive continuing education in the specialty to achieve optimal patient outcomes. Trauma nurse leaders are becoming essential in trauma centers. The presence of these professionals has been shown to decrease length of stay in the hospital and the ICU.[22] In a recent study by Polovitch et al,[23] trauma nurse leaders were found to reduce the activation time of response to a traumatic injury patient's arrival and care. Orchestrated time-sensitive interventions by a well-functioning interprofessional team helps identify instability and correct any situations that might lead to a patient's early death.

Nursing units should establish a standardized format in communicating the patient's plan of care to members of the health care team.

Conclusion

When a patient has persistent hypotension with no identifiable cause, the health care team, including critical care nurses, should look for evidence of other factors such as septic emboli, which can present atypically, as

witnessed in this case presentation. Nurses should review the entire clinical picture of the patient with the team and participate in interdisciplinary care. CCN

Financial Disclosures
None reported.

See also

To learn more about sepsis, read "Strategies for the Management of Sepsis" by Gilbert et al in *AACN Advanced Critical Care,* 2019;30(1):5-11. https://doi.org/10.4037/aacnacc2019526. Available at **www.aacnacconline.org**.

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