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CASE REPORT

When "Adequate" MAP Is Not Enough: Underresuscitation as a Driver of Multiple Organ Dysfunction Syndrome in Polytrauma

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ABSTRACT

Background: Trauma remains the leading preventable cause of morbidity and mortality in adults, and the development of secondary sepsis significantly increases mortality risk. Reliance on static hemodynamic endpoints, such as mean arterial pressure (MAP), without dynamic perfusion markers or structured reassessment, may conceal ongoing shock and allow silent progression toward multiple organ dysfunction syndrome (MODS). This case report illustrates how underresuscitation, sustained despite an apparently adequate MAP, drove progressive organ failure in a polytrauma patient who deteriorated after initial treatment.
Case: Male, 68 years old, with a history of coronary heart disease and stroke, had shock due to kidney trauma, tension pneumothorax, hemothorax, multiple rib fractures, complete left radius fracture, and right frontal laceration following a traffic accident while driving a car. Initial emergency resuscitation included fluid loading, water seal drainage placement, and central vasoactive infusions (dobutamine/norepinephrine) before transfer to the high care unit. After 31 hours of treatment, he developed multiple organ dysfunction syndrome (MODS), requiring intensive care unit admission, mechanical ventilation, and continuous renal replacement therapy (CRRT). Despite transient improvement during CRRT, he deteriorated post-termination and died on day five due to unrecognized secondary septic shock.
Discussion: Despite an apparently adequate MAP, the patient's escalating vasopressor and inotrope requirement signaled unresolved shock that went unrecognized, allowing sequential hemorrhagic, obstructive, and septic shock states to progress undetected into MODS. Early mechanical ventilation plays a crucial role in polytrauma resuscitation to reduce oxygen demand and prevent respiratory complications like ARDS. Insufficient identification of shock etiology and lack of follow-up in this patient led to suboptimal resuscitation up to MODS and death.
Conclusion: Static hemodynamic targets such as MAP can mask ongoing tissue hypoperfusion. Perfusion guided, structured reassessment not isolated blood pressure targets together with early airway management and prompt sepsis bundle implementation, is essential to prevent underresuscitation from silently driving MODS in complex trauma patients.

Keywords: Multiple organ dysfunction syndrome, mechanical ventilation, polytrauma, shock, resuscitation, underresuscitation.

INTRODUCTION

Trauma remains one of the leading causes of preventable morbidity and mortality among adults. According to the 2021 World Health Organization (WHO) report, approximately 4.4 million people die annually from trauma, accounting for nearly 8% of all global deaths.[1] Although only a subset of trauma patients develop hemodynamic instability, these individuals have a substantially increased risk of clinical deterioration and mortality, particularly those with multiple injuries or polytrauma.[2] Therefore, polytrauma patients require rapid and accurate initial assessment to identify and manage life-threatening conditions, especially injuries affecting the airway and respiratory function.[2,3] Thoracic trauma involving pulmonary parenchymal injury, such as pulmonary contusion or hemothorax, increases risk of Acute Respiratory Distress Syndrome (ARDS) and may induce production of proinflammatory cytokines secondary to tissue injury.[4,5] Inadequate initial resuscitation in critically ill trauma patients may contribute to progressive organ dysfunction, including Multiple Organ Dysfunction Syndrome (MODS), and increases the risk of mortality.[3] This case report describes a patient with polytrauma who experienced deterioration following initial resuscitation. An analysis of each stage of resuscitation provides important clinical lessons for optimizing the management of similar cases in the future.

CASE

A 68-year-old male was admitted to the emergency department with complaints of shortness of breath and pain in the chest and left flank following a traffic accident one hour prior to admission. The patient was driving a car when he collided with the vehicle in front of him and was subsequently hit from behind by another vehicle. His chest hit the steering wheel and dashboard during the collision. There was no history of loss of consciousness or projectile vomiting.

The patient had a history of coronary artery disease, that had undergone placement of three coronary stents, and stroke. His regular medications included isosorbide dinitrate (ISDN) 5 mg once daily, acetylsalicylic acid 80 mg twice daily, bisoprolol 2.5 mg once daily, clopidogrel 75 mg once daily, and simvastatin 20 mg once daily.

On initial examination, the patient was alert and fully conscious. His vital signs were as follows: blood pressure 86/48 mmHg, heart rate 99 beats/minute, respiratory rate 33 breaths/minute, and oxygen saturation 97–98% with oxygen supplementation via nasal cannula at 4 L/min. Physical examination revealed left chest wall crepitus, right chest and left flank contusions, swelling of the left wrist, and an open wound on the right temporal region.

Chest radiography showed multiple left rib fractures and a complete fracture of the left radius. Point-of-care ultrasound (POCUS) showed no significant abnormalities. Non-contrast computed tomography (CT) of the head and abdomen revealed a Grade I intrarenal hematoma without evidence of intracranial hemorrhage or cerebral edema. Initial laboratory investigations showed hemoglobin 13.8 g/dL, leukocyte count 51,300/µL, random blood glucose 322 mg/dL, SGOT/SGPT 71/56 U/L, urea/creatinine 56.3/1.35 mg/dL, and D-dimer >20,000 ng/mL.

The patient was diagnosed with suspected hemorrhagic shock secondary to renal trauma, along with suspected pulmonary contusion, multiple left rib fractures, a complete fracture of the left radius, and a right frontal scalp laceration. Initial management included a 1,000 mL crystalloid fluid bolus during the first hour, continuous infusions of dobutamine (3 mcg/kg/min, titrated according to clinical response) and norepinephrine (0.05 mcg/kg/min, titrated), as well as analgesia with a 50 mg intravenous tramadol bolus followed by a continuous infusion of 200 mg over 24 hours.

After five hours of observation in the Emergency Department, the patient developed worsening dyspnea. He remained alert and fully conscious, with a respiratory rate of 38 breaths/minute, oxygen saturation of 95–96% while receiving oxygen via a non-rebreather mask (NRM) at 15 L/minute, heart rate of 140 beats/minute, and blood pressure of 79/50 mmHg with dobutamine (4 mcg/kg/min) and norepinephrine

(0.2 mcg/kg/min) support. Point-of-care ultrasound (POCUS) showed absent lung sliding on the left side. Repeat chest radiography revealed a left pneumothorax, pulmonary contusion, multiple left rib fractures, and extensive subcutaneous emphysema involving the bilateral cervical region and left chest wall.

The patient was diagnosed with a left tension pneumothorax. Needle thoracostomy was performed using a 16-gauge intravenous catheter connected to a mini water-sealed drainage (WSD) system. Following the procedure, the patient's dyspnea was improved. He remained alert and fully conscious, with a respiratory rate of 20–22 breaths/minute, oxygen saturation of 100% with oxygen supplementation via a non-rebreather mask (NRM) at 15 L/minute, heart rate of 88 beats/minute, and blood pressure of 100/70 mmHg with dobutamine (4 mcg/kg/minute) and norepinephrine (0.2 mcg/kg/minute) support.

A central venous catheter was inserted into the right internal jugular vein, and definitive chest drainage was planned. A WSD tube was inserted into the left thoracic cavity 14 hours after admission to the Emergency Department. Subsequent chest computed tomography (CT) revealed a left hematopneumothorax and right pleural effusion, with right hemothorax considered as a differential diagnosis. At 27 hours after admission to the Emergency Department, the patient was transferred to the High Care Unit (HCU) for further monitoring. At that time, he complained of pain at the WSD insertion site but remained alert and fully conscious. His respiratory rate was 22–30 breaths/minute, oxygen saturation was 98–99% while receiving oxygen via a non-rebreather mask (NRM) at 15 L/minute, and heart rate was 100 beats/minute. His blood pressure was 87/59 mmHg with dobutamine (8 mcg/kg/minute) and norepinephrine (0.9 mcg/kg/minute) support—a marked escalation from the initial dose (0.05 mcg/kg/minute) despite a nominally acceptable MAP, a signal of unresolved shock that was not further investigated at this stage.

Four hours later, the patient's condition deteriorated, with a decreased level of consciousness (GCS E3M4V1), respiratory rate of 35 breaths/minute, oxygen saturation of 80% despite NRM oxygen therapy at 15 L/minute, heart rate of 115 beats/minute, and blood pressure of 63/38 mmHg with dobutamine (15 mcg/kg/minute) and norepinephrine (0.9 mcg/kg/minute) support. Laboratory evaluation revealed elevated inflammatory markers, including CRP of 23.98 mg/dL and procalcitonin >32 ng/mL. Coagulation parameters showed elevated D-dimer levels of 19,500 ng/mL and fibrinogen of 469 mg/dL. Renal function had deteriorated, with urea of 112.9 mg/dL and creatinine of 3.85 mg/dL.

The patient was diagnosed with multiple organ dysfunction syndrome (MODS) associated with mixed shock, including obstructive shock, hemorrhagic shock, and suspected septic shock. He was immediately intubated and transferred to the intensive care unit (ICU) within one hour.

Upon arrival in the intensive care unit (ICU), the patient had a decreased level of consciousness (GCS E1M1Vt). Mechanical ventilation was initiated using pressure support synchronized intermittent mandatory ventilation (PSIMV) mode with an inspiratory pressure of 15 mmHg, pressure support of 12 mmHg, respiratory rate of 12 breaths/minute, positive end-expiratory pressure (PEEP) of 5 mmHg, and FiO_2 of 100%. The patient's respiratory rate was 17 breaths/minute, with an oxygen saturation of 97%. Oscillation was observed in the left thoracic WSD system. Heart rate was 100 beats/minute, and mean arterial pressure (MAP) ranged from 56–89 mmHg with dobutamine (20 mcg/kg/min), norepinephrine (2 mcg/kg/min), and vasopressin (0.04 mcg/kg/min) support. Initial ICU laboratory evaluation revealed hemoglobin of 8.4 g/dL, leukocyte count of 21,600/µL, albumin level of 2.1 g/dL, D-dimer >20,000 ng/mL, and lactate level of 7.9 mmol/L—evidence that tissue hypoperfusion had already been ongoing for the 31 hours preceding ICU admission, undetected because lactate had not been measured earlier. Renal function tests demonstrated severe acute kidney injury, with urea of 130.9 mg/dL and creatinine of 4.87 mg/dL. Liver function tests indicated acute hepatic dysfunction, with SGOT of 186 U/L and SGPT of 144 U/L. The patient then received

Table 1. Vital Sign and Laboratory Trend in Emergency Department and High Care Unit

	Primary survey	2 hours	5 hours (Pneumo-thorax)	7 hours (Post mini WSD)	27 hours (Before transferred to HCU)	31 hours (HCU)
MAP (mmHg)	60	74 (dobutamine 4 mcg/kg/min)	59 (NE 0,2 mcg/kg/min, dobutamine 4 mcg/kg/min)	80 (NE 0,2 mcg/kg/min, dobutamine 4 mcg/kg/min)	70 (NE 0,8 mcg/kg/min, dobutamine 8 mcg/kg/min)	47 (NE 0,9 mcg/kg/min, dobutamine 15 mcg/kg/menit
HR (beats/min)	99	90	140	88	100	115
GCS	E4M6V5	E4M6V5	E4M6V5	E4M6V5	E4M6V5	E3M4V1
RR (breaths/min)	30	22-24	38	20-22	20-30	35
SpO2 (%)	97-98 on NC 3 L/m	97-98 on NC 3 L/m	95-96 on NRM 15 L/m	95-96 on NRM 15 L/m	98-99 on NRM 15 L/m	80 on NRM 15 L/m
Hb (g/dL)		13.8			11.4	10.5
Leucocyte (/uL)		51.300			25.800	23.700
Platelet (/uL)		375.000			227.000	222.000
Procalcitonin (ng/mL)						>32
CRP (mg/dL)						23.98
Urea (mg/dL)		56.3				112.9
Creatinine (mg/dL)		1.35				3.85
SGOT (U/L)		71				
SGPT (U/L		56				
D-Dimer (ng/mL)		>20.000				19.500
PT (times)		0.9				1.1
aPTT (times)		0.8				0.8

MAP : mean arterial pressure; HR : heart rate : GCS : Glasgow coma scale; RR : respiratory rate; SpO2 :peripheral oxygen saturation; NE : norepinephrine; NRM : non-rebreathing mask; NC : nasal cannula ; WSD : water sealed drainage; Hb : hemoglobin; CRP: C-reactive protein; PT: prothrombin time; aPTT: activated partial thrombin time

packed red blood cell (PRBC) transfusion with a target hemoglobin level above 10 g/dL. Based on surgeon evaluation, an additional WSD tube was inserted into the right thoracic cavity due to multiple right-sided rib fractures associated with delayed-onset pneumothorax. On the first day of ICU care, the patient's level of consciousness remained impaired (GCS E1M1Vt). Mechanical ventilation was continued with a respiratory rate of 15 breaths/minute and oxygen saturation of 97% using pressure support synchronized intermittent mandatory ventilation (PSIMV) mode (inspiratory pressure 15 mmHg, pressure support 12 mmHg, respiratory rate 12 breaths/minute, and PEEP 5 mmHg). Hemodynamic status remained unstable, with a heart rate ranging from 110–120 beats/minute and mean arterial pressure (MAP) ranging from 63–95 mmHg with dobutamine (20 mcg/kg/min), norepinephrine (2 mcg/kg/min), and vasopressin (0.04 mcg/kg/min) support. Laboratory evaluation after

Table 2. Vital Sign and Laboratory Trend in ICU

	H0	H1 (CRRT I)	H2 (CRRT I-II)	H3 (CRRT II)	H4	H5
MAP (mmHg)	56-89 (on NE 2 mcg/kg/m, dobutamine 20 mcg/kg/m, vasopressin 0,04 mcg/kg/m)	63-95 (on NE 2 mcg/kg/m, dobutamine 20 mcg/kg/m, vasopressin 0,04 mcg/kg/m)	93-106 (on NE 2 mcg/kg/m, dobutamine 20 mcg/kg/m, vasopressin 0,04 mcg/kg/m)	85-100 (on NE 1 mcg/kg/m, dobutamine 20 mcg/kg/m, vasopressin 0,04 mcg/kg/m)	63-75 (on NE 2 mcg/kg/m, vasopressin 0,04 mcg/kg/m)	Asystole
HR (beats/min)	100-120	110-120	100-110	90-110	100-110	
GCS	E1M1Vett	E1M2Vett	E1M2Vett	E2M4Vett	E1M3Vett	
SpO2	97% on PSIMV	97% on PSIMV	99-100% on PSIMV	98-99% on PSIMV	96-97% on CMV-PC	50-65%
Hb (g/dL)	8.4	10.4	10.4			
Leucocyte (/uL)	21.600	15.700	10.300			
Platelet(/uL)	172.000	81.000	40.000			
Procalcitonin (ng/mL)	>32		>32			
CRP			35		41.69	
(mg/dL)	21.11					
Lactate (mmol/L)	7.9	6.0	5.1			
Albumin (g/dL)	2.1	2.65	2.75			
Urea (mg/dL)	130	113	97		209.9	
Creatinine (mg/dL)	4.87	3.9	2.5		4.6	
SGOT (U/L)	186					
SGPT (U/L)	144					
D-Dimer	>20.000*	>20.000	>20.000	14.468	15.804	
PT (times)	1.1	1.2	1.4	1.1		
aPTT (times)	0.8	0.8	1.1	0.8		
Balance (UO) mL (mL/kg/hours)	1.697 (0)	3.354 (0.1) on furosemide 2 mg/hours	3.490 (1.4) on furosemide 2 mg/hours	1.372 (1,2) on furosemide 2 mg/hours	4.200 (1,2) on furosemide 2 mg/hours	

MAP: mean arterial pressure; HR: heart rate; GCS: Glasgow coma scale; SpO2: peripheral oxygen saturation; UO: urine output; NE: norephinephrine; PSIMV: pressure support synchronised intermittent mandatory ventilation; CMV-PC: continuous mandatory ventilation pressure control; Hb: hemoglobin; CRP: C-reactive protein; PT: prothrombin time; aPTT: activated partial thrombin time.

transfusion showed a hemoglobin level of 10.4 g/dL following administration of two units of packed red blood cells (PRBCs) and a platelet count of 81.000/µL. Serum albumin increased to 2.65 g/dL following administration of 20% human albumin. Coagulation studies revealed a state of severe hypercoagulability, with D-dimer >20,000 ng/mL, though PT and aPTT were not

Table 3. Resuscitation Strategy Evaluation

Resuscitation Strategy	Application
Initial assessment and resuscitation	
Airway maintenance	Intubation was not performed earlier.
Respiration and ventilation	Mechanical ventilation with a lung-protective strategy was not performed. Water-seal drainage (WSD) tubes were inserted into both sides of the chest.
Circulation	Two peripheral intravenous access lines were inserted Central access was inserted Needle thoracentesis was performed MAP was maintained >60 mmHg, but the increased vasopressors and inotropes doses was not evaluated Lactate test was performed only in the ICU
Disability and neurological status	A head CT scan was performed.
External exposure control	Open wound care was performed. Prevention of hypothermia was performed.
Damage control resuscitation	
Hemorrhage control	WSDs were inserted in both sides of the thorax.
Identification of coagulopathy	Coagulation factors were evaluated.
Massive transfusion protocol	There was no indication to initiate the massive transfusion protocol.
Sepsis management	
ICU care	ICU admission occurred one hour after sepsis was diagnosed. However, the diagnosis itself was delayed.
Management of the source of infection	Antibiotics were administered once sepsis was diagnosed.
Vasopressor administration	Vasopressors were administered starting in the emergency department

WSD : water sealed drainage; MAP : mean arterial pressure; ICU : intensive care unit; CT : computed tomography

prolonged. Renal function assessment indicated acute kidney injury (AKI), with urea at 113.8 mg/dL and creatinine at 3.9 mg/dL. Lactic acid levels had decreased to 6.2 mmol/L. The procalcitonin level was markedly elevated at >32 ng/dL, accompanied by a leukocyte count of 15,700/uL, indicating septic shock. The Sequential Organ Failure Assessment (SOFA) score was 16. A decision was made to initiate CRRT using the continuous veno-venous hemodialysis (CVVHD) method, with settings of 150 mL effluent, 1000 mL dialysate, 1000 mL replacement fluid, and 50 mL post-filter removal, utilizing heparin (200 IU) as an anticoagulant. Three hours after the initiation of CVVH, the patient showed signs of improved urine output—rising to 0.3 mL/kg/hour from an initial state of anuria. On the first day post-CRRT, circulatory parameters showed a heart rate of 100–110 beats per minute and a mean arterial pressure of 93–106 mmHg, with dobutamine (20 mcg/kg/min), norepinephrine (2 mcg/kg/min), and vasopressin (0.04 mcg/kg/min) support. The patient's CRP level was 35 mg/dL. Renal function showed improvement with a urea level of 97 mg/dL and creatinine of 2.5 mg/dL. Lactate levels improved to 5.1 mmol/L. The patient remained in a state of severe hypercoagulability, characterized by a D-dimer level >20,000 ng/mL, a PT duration 1.4 times the control value, and a platelet count of 40,000/µL. On the second day of ICU care, the patient's cumulative fluid balance was 3,490 mL, with a urine output of 1.4 mL/kg/hour with furosemide infusion at 2 mg/hour.

On the third day of ICU care, the patient showed improved consciousness, presenting as somnolent (E2M4Vett) while receiving propofol sedation at 50 mg/hour. Circulatory parameters

showed a heart rate ranging from 90–110 beats per minute and a mean arterial pressure of 85–100 mmHg with dobutamine (20 mcg/kg/min), norepinephrine (1 mcg/kg/min), and vasopressin (0.04 mcg/kg/min) support. CVVHD was continued for a second day. Following CVVHD, laboratory results showed the patient's D-dimer level had decreased to 14,468 ng/mL and the PT ratio was 1.1. Blood gas analysis showed a pH of 7.45, pCO2 of 50.5 mmHg, HCO3 of 35.6 mmol/L, and arterial oxygen saturation of 99%. The patient's cumulative fluid balance was positive 1,372 mL, with a urine output of 1.2 mL/kg/hour with furosemide infusion at 2 mg/hour.

On the fourth day of ICU care, CVVHD was discontinued due to national health insurance coverage restrictions. The patient's general condition deteriorated again to E1M3Vett. Circulatory parameters showed a heart rate of 100–110 beats per minute and a MAP of 63–75 mmHg with norepinephrine (2 mcg/kg/min) and vasopressin (0.04 mcg/kg/min) support. Laboratory results showed D-dimer levels rising again to 15,804 ng/mL, and renal function worsening, with urea at 209.6 mg/dL and creatinine at 4.56 mg/dL. The patient's CRP level rised again to 41.69 mg/dL. Blood gas analysis showed a pH of 7.28, elevated pCO2 of 73.0 mmHg, HCO3- of 35.0 mmol/L, and arterial oxygen saturation of 92%, indicating uncompensated respiratory acidosis. The patient's cumulative fluid balance was positive 4,200 mL, with a urine output of 1.2 mL/kg/hour over with furosemide infusion at 2 mg/hour.

On the fifth day of ICU care, the patient developed oxygen desaturation followed by cardiac arrest. Cardiopulmonary resuscitation was performed for five cycles; however, return of spontaneous circulation was not achieved, and the the patient died.

DISCUSSION

The strategy for managing trauma cases consists of an initial assessment (primary survey) and resuscitation. Resuscitation is performed simultaneously, while the patient's condition is periodically reassessed to evaluate the effectiveness of the resuscitation. According to Advanced Trauma Life Support (ATLS) guidelines, the initial assessment follows the ABCDE sequence: airway maintenance with cervical spine restriction (airway), respiration and ventilation (breathing), circulation with hemorrhage control (circulation), disability and neurological status (disability), and exposure control (exposure).[3,6]

Intubation was initially withheld as the patient maintained adequate oxygen saturation and remained fully conscious. Rather than initiating intubation and sedation, the patient was transferred to the High Care Unit (HCU) for close monitoring. Intubation could have been performed while the patient was still in the Emergency Department. According to ATLS guidelines, intubation is indicated for airway protection (in cases of altered consciousness risking the loss of airway patency (specifically a GCS score <9), severe maxillary fractures, laryngeal or tracheal injury, or persistent airway obstruction due to neck hematoma or inhalation injury) and to facilitate ventilation (in cases of apnea or respiratory distress—such as tachypnea >30 breaths/minute, hypoxia, or hypercarbia).[3] Even when a patient is fully conscious and oxygen saturation targets are preserved, mechanical ventilation can be a strategy to reduce the patient's oxygen demand. Furthermore, patients with multiple trauma experience severe pain resulting from their injuries. Pain increases the risk of agitation and exerts negative immunomodulatory effects.[7] Inadequately managed pain raises myocardial oxygen demand and can lead to cardiac arrhythmias and myocardial ischemia. Additionally, agitation increases the risk of vascular access dislodgement, ventilator dyssynchrony, and even spontaneous extubation. Effective pain management using multimodal analgesia—combining simple analgesics and opioids with sedation such as propofol or alpha-2 agonists—has been shown to reduce morbidity in severe trauma patients.[7,] A study demonstrated that earlier intubation of critically ill trauma patients was associated with a statistically significant reduction in mortality.9 A study also found that early intubation was superior in shortening ICU length of stay and correcting acid-base disturbances in patients with thoracic trauma.[10]

An initial assessment of respiratory function revealed increased work of breathing resulting from rib fractures accompanied by pulmonary contusion. Systematic reviews indicate that ARDS develops in 17% of patients with isolated pulmonary contusion and in 78% of those with pulmonary contusion associated with polytrauma. ARDS develops in approximately 82% of patients when more than 20% of the lung parenchyma is compromised. This is attributed to the release of proinflammatory cytokines following parenchymal damage, which precipitates acute lung injury.[11] The primary therapeutic goal for such patients is the maintenance of optimal PEEP. Although non-invasive ventilation may be considered, intubation remains the preferred intervention. A lung-protective ventilation strategy—utilizing low tidal volumes (4–6 mL/kg of ideal body weight)—is expected to reduce the risk of alveolar distension and structural damage.[12]

Based on the assessment of the circulatory component, the patient presented in a state of shock. Shock is a life-threatening syndrome characterized by inadequate tissue perfusion, leading to cellular hypoxia and organ dysfunction. Management of shock involves reducing oxygen consumption and increasing oxygen delivery to the tissues.[13] Two peripheral intravenous access lines were inserted upon the patient's arrival. Central venous access was subsequently secured to facilitate the administration of vasoactive agents and to assess fluid adequacy.

The need to activate the massive transfusion protocol (MTP) can be assessed using the Assessment of Blood Consumption (ABC) score, which comprises the presence of a penetrating injury, a positive Focused Assessment with Sonography for Trauma (FAST) result, systolic blood pressure ≤90 mmHg, and a heart rate ≥120 beats per minute. MTP may be initiated if the ABC score is ≥2, or in the presence of persistent hemodynamic instability or active bleeding requiring intervention.[6,14] In this patient, there was insufficient evidence to warrant MTP activation.

The etiology evaluation of the shock was conducted using physical examination, POCUS, laboratory tests, and radiological imaging (CT scans of the thorax, abdomen, and head). In the early stage, the cause of the shock could not be identified. The patient's condition subsequently deteriorated, developing into obstructive shock caused by a left tension pneumothorax and a right hemothorax. After the patient was stable enough for transfer to the High Care Unit (HCU), a further deterioration occurred due to septic shock.

Mean arterial pressure (MAP) and lactate levels can be used to assess tissue perfusion in patients with shock. In this case, the patient's MAP had been supported since initial admission to the emergency department using high-dose vasopressors and inotropes. MAP is a key indicator of the driving force behind venous return and cardiac output. Therefore, an increase in MAP significantly enhances tissue blood flow and improves tissue perfusion. Certain tissues, such as the brain and kidneys, have the ability to autoregulate blood flow. A MAP below the threshold of approximately 60–65 mmHg is associated with reduced organ perfusion.[15] In this situation, the patient experienced recurrent clinical deterioration with MAP values falling below 60 mmHg. Although a MAP >65 mmHg was maintained prior to the patient's discharge from the emergency department (Table 1), the escalating doses of vasopressors and inotropes indicated a persistent imbalance in oxygen delivery—this discordance between a numerically acceptable MAP and rising catecholamine requirement is itself the clinical signature of underresuscitation. However, a comprehensive evaluation to identify the underlying cause of this condition was not performed.

The rise in lactate levels during shock is caused by tissue hypoxia, accelerated glycolysis due to a hyperadrenergic state, medications (epinephrine, beta-2 agonists), or liver failure manifesting as anaerobic metabolism resulting from oxygen delivery falling below a critical threshold. Lactate measurement serves as an objective marker for assessing the response to resuscitation. Although guidelines suggest that lactate is not a direct surrogate for tissue hypoperfusion, the parameter is sufficiently sensitive to assess end-organ hypoperfusion.[16,17] In this case, the elevated lactate levels observed

while the patient was in the ICU (table 2) indicate persistent tissue hypoperfusion.

Sepsis is a life-threatening condition characterized by organ dysfunction resulting from the body's inability to manage an infection. It can be a cause of death in trauma patients, with a mortality rate reaching 30%. A diagnosis of sepsis is established based on clinical assessment indicating organ dysfunction, accompanied by evidence of infection. Screening methods that can be used to diagnose sepsis include the Systemic Inflammatory Response Syndrome (SIRS) criteria, quick Sequential Organ Failure Score (qSOFA), Sequential Organ Failure Assessment (SOFA), National Early Warning Score (NEWS), and Modified Early Warning Score (MEWS). Given the variations in sensitivity and specificity among these screening methods, relying on a single method to diagnose sepsis is not recommended.[15,18]

In this case, the patient was admitted to the ICU already in a state of septic shock and MODS, presenting with central nervous system involvement (reduced consciousness), renal involvement (anuria), hematological abnormalities (thrombocytopenia and hypercoagulability), hepatic dysfunction (hypoalbuminemia and elevated transaminase enzymes), cardiovascular instability, and respiratory involvement (ARDS). According to the Surviving Sepsis Guidelines,[15] specific recommendations must be implemented within a short timeframe. First, patients should be admitted to the ICU within six hours of being diagnosed with sepsis or septic shock. In this case, ICU admission occurred one hour after the septic shock was diagnosed. However, the increasing need for vasopressors and inotropes suggests that sepsis may have developed earlier and undetected.

The second guideline concerns the immediate management of the source of infection. In this case, elevated CRP and high procalcitonin levels were observed in the High Care Unit (HCU). Although CRP is not a specific parameter for infection, procalcitonin is a reliable biomarker for detecting bacterial infection.[19] In patients diagnosed with or suspected of having septic shock, antibiotics must be administered within one hour of diagnosis. Bacterial cultures should be obtained prior to antibiotic administration.[15] In this case, intravenous ceftriaxone (2 grams) was administered in the HCU. Subsequently, intravenous ampicillin-sulbactam (3 gram every 6 hours) and intravenous amikacin (1 gram every 24 hours) were administered following the diagnosis of sepsis.

Trauma-induced injury has been shown to increase plasma antithrombin III levels. Antithrombin III is a glycoprotein that targets factor Xa and factor II (thrombin), acting as an inhibitor of the coagulation pathway. A deficiency in this inhibitory factor can lead to a hypercoagulable state. Furthermore, suppression of the fibrinolytic system characterized by elevated levels of plasminogen activator inhibitor-1 can exacerbate this hypercoagulable condition. Hypercoagulability is also frequently observed in patients with septic shock and contributes to organ failure by impairing microcirculation.[20,21] In this case, the phenomenon of hypercoagulability may arise as a result of shock that remained unresolved from the initial stage of patient management. This can be a factor that exacerbates the patient's condition, potentially leading to MODS.

Taken together, this case traces a single causal chain rather than a series of isolated complications. Withheld elective intubation and reliance on MAP as the primary resuscitation endpoint allowed a persistent oxygen-delivery deficit to go unrecognized despite escalating vasopressor and inotrope support. This unresolved, silent hypoperfusion permitted three sequential and undiagnosed shock states hemorrhagic, obstructive, and septic to progress unchecked, culminating in MODS and death. The lesson is not that resuscitation was withheld, but that the endpoints used to judge its adequacy were insufficient to detect its failure.

CONCLUSION

Several resuscitation strategies for polytrauma cases were not optimally implemented in this patient (Table 3), and the deficit was masked by an apparently adequate MAP rather than by an absence of treatment.

This underresuscitation and unresolved shock can lead extensive organ failure and even death. Early mechanical ventilation is required as a resuscitation strategy for polytrauma patients to reduce oxygen demand, and the underlying cause of shock must be identified through dynamic, perfusion-guided reassessment rather than isolated blood pressure targets to enable timely, definitive management.

REFERENCES

1. World Health Organization. Preventing injuries and violence: an overview. Geneva: World Health Organization; 2022.
2. Cole E, Gillespie S, Vulliamy P, Brohi K, Akkad H, Apostolidou K, et al. Multiple organ dysfunction after trauma. Br J Surg. 2020;107(4):402-12. doi:10.1002/bjs.11361.
3. American College of Surgeons Committee on Trauma. Advanced Trauma Life Support (ATLS®): Student Course Manual. 10th ed. Chicago (IL): American College of Surgeons; 2018.
4. Ganter MT, Roux J, Miyazawa B, Howard M, Frank JA, Su G, et al. Interleukin-1β causes acute lung injury via αvβ5 and αvβ6 integrin-dependent mechanisms. Circ Res. 2008;102(7):804-12. doi:10.1161/CIRCRESAHA.107.161067.
5. Goodman RB, Pugin J, Lee JS, Matthay MA. Cytokine-mediated inflammation in acute lung injury. Cytokine Growth Factor Rev. 2003;14(6):523-35. doi:10.1016/S1359-6101(03)00059-5.
6. Galbraith CM, Wagener BM, Chalkias A, Siddiqui S, Douin DJ. Massive trauma and resuscitation strategies. Anesthesiol Clin. 2023;41(2):283-301. doi:10.1016/j.anclin.2023.02.003.
7. Joseph A. Sedation of the trauma patient in the intensive care unit. J Emerg Crit Care Med. 2018;2:8. doi:10.21037/jeccm.2018.01.08.
8. Dattatri R, Jain VK, Iyengar KP, Vaishya R, Garg R. Anaesthetic considerations in polytrauma patients. J Clin Orthop Trauma. 2021;12(1):50-57. doi:10.1016/j.jcot.2020.09.019.
9. Xixi NA, Kremmydas P, Xourgia E, Giannopoulou V, Sarri K, Siempos II. Association between timing of intubation and clinical outcomes of critically ill patients: a meta-analysis. J Crit Care. 2022;71:154062. doi:10.1016/j.jcrc.2022.154062.
10. Nasr-Esfahani M, Boroumand AB, Kolahdouzan M. Early intubation vs. supportive care outcomes in patients with severe chest trauma: a randomized trial study. Arch Acad Emerg Med. 2019;7(1):e35. doi:10.22037/aaem.v7i1.390.
11. Relja B, Horstmann JP. Mechanical ventilation of the patient following traumatic injury. In: Trauma Anaesthesia. 1st ed. Cham: Springer; 2018. p. 85-110. doi:10.1007/978-3-319-60213-7_6.
12. Rendeki S, Molnár TF. Pulmonary contusion. J Thorac Dis. 2019;11(Suppl 2):S141-51. doi:10.21037/jtd.2018.11.53.
13. Killu K, Sarani B, editors. Fundamental critical care support. 6th ed. Mount Prospect (IL): Society of Critical Care Medicine; 2017.
14. Nunez TC, Voskresensky IV, Dossett LA, Shinall R, Dutton WD, Cotton BA. Early prediction of massive transfusion in trauma: simple as ABC (assessment of blood consumption)? J Trauma. 2009;66(2):346-52. doi:10.1097/TA.0b013e3181961c35.
15. Evans L, Rhodes A, Alhazzani W, Antonelli M, Coopersmith CM, French C, et al. Surviving Sepsis Campaign: international guidelines for management of sepsis and septic shock 2021. Intensive Care Med. 2021;47(11):1181-247. doi:10.1007/s00134-021-06506-y.
16. Evans L, Rhodes A, Alhazzani W, Antonelli M, Coopersmith CM, French C, et al. Surviving sepsis campaign: international guidelines for management of sepsis and septic shock 2021. Crit Care Med. 2021;49(11):e1063-e1143. doi:10.1097/CCM.0000000000005337.
17. Dunn JOC, Mythen MG, Grocott MP. Physiology of oxygen transport. BJA Educ. 2016;16(10):341-8. doi:10.1093/bjaed/mkw012.
18. Mas-Celis F, Olea-López J, Parroquin-Maldonado JA. Sepsis in trauma: a deadly complication. Arch Med Res. 2021;52(8):808-16. doi:10.1016/j.arcmed.2021.06.004.

19. Liang H, Liu Z, Xiao Y, Yan J, Fan X, Jin H. Prediction of sepsis in trauma patients. Burns Trauma. 2014;2(3):106-12. doi:10.4103/2321-3868.135611.
20. Riha GM, Kunio NR, Van PY, Kremenevskiy I, Anderson R, Hamilton GJ, et al. Uncontrolled hemorrhagic shock results in a hypercoagulable state modulated by initial fluid resuscitation regimens. J Trauma Acute Care Surg. 2013;75(1):129-34. doi:10.1097/TA.0b013e31829231e1.
21. Kim SM, Kim SI, Yu G, Kim JS, Hong SI, Kim WY. Hypercoagulability in septic shock patients with thrombocytopenia. J Intensive Care Med. 2022;37(6):721-7. doi:10.1177/08850666211018135.

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