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**| RESEARCH ARTICLE**

# A Bleeding Conundrum: Intraventricular Hemorrhage and Gross Hematuria Secondary to Pneumosepsis

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**| ABSTRACT**

We report the case of a 67-year-old Saudi man with multiple comorbidities, including type 2 diabetes mellitus, hypertension, ischemic heart disease, and chronic kidney disease, who presented with progressive shortness of breath, altered mental status, and fever. His symptoms began five days prior with productive cough, pleuritic chest discomfort, and generalized body aches, initially managed at home. On presentation, he was hypotensive, tachycardic, hypoxemic, and disoriented, with signs of respiratory distress and new ecchymoses. Laboratory evaluation revealed leukocytosis, elevated inflammatory markers, acute kidney injury, and coagulopathy. Chest radiography demonstrated new bilateral multifocal pneumonic patches compared with prior baseline imaging. Blood cultures grew Streptococcus pneumoniae, confirming pneumococcal sepsis. During intensive care unit admission, he developed gross hematuria, progressive thrombocytopenia, prolonged coagulation times, and evidence of disseminated intravascular coagulation. Acute neurological deterioration prompted urgent CT imaging, which revealed extensive intraventricular hemorrhage with early hydrocephalus. Despite aggressive supportive care, including hemodynamic stabilization, transfusions, renal replacement therapy, and tailored antibiotics, the patient developed refractory shock and multiorgan failure. After multidisciplinary discussion and family consultation, care was transitioned to comfort measures, and he subsequently died. This case illustrates the fulminant and often fatal nature of pneumococcal sepsis complicated by disseminated intravascular coagulation and intracranial hemorrhage, emphasizing the need for early recognition, close monitoring, and multidisciplinary management in high-risk patients.

**| KEYWORDS**

Disseminated Intravascular Coagulation, Pneumonia, Sepsis, Pneumosepsis, Bleeding, Brain Hemorrhage, Intraventricular Hemorrhage, Gross Hematuria, Platelets, Fibrinogen, PT, PTT, Coagulation profile

**| ARTICLE INFORMATION**

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**Introduction**

Sepsis remains a leading cause of morbidity and mortality worldwide despite advances in antimicrobial therapy and critical care management. It is defined as life threatening organ dysfunction caused by a dysregulated host response to infection [1]. Pneumonia is among the most common infectious sources of sepsis in both community and hospital settings and is frequently associated with rapid clinical deterioration, especially in older adults and patients with comorbid disease [5,8]. Pneumosepsis represents a severe disease spectrum in which pulmonary infection triggers a systemic inflammatory response that may progress to multiorgan failure. Community acquired pneumonia accounts for a significant proportion of sepsis cases admitted to intensive care units. Mortality rates increase substantially once respiratory infection is complicated by septic shock or coagulation abnormalities [5,8]. Current guidelines emphasize early recognition, prompt antimicrobial therapy, hemodynamic support, and careful monitoring for complications such as acute respiratory distress syndrome, acute kidney injury, and coagulopathy [6]. Despite adherence to evidence based management, unpredictable progression may still occur in some patients. Disseminated intravascular coagulation is a serious and well recognized complication of sepsis. It is characterized by widespread activation of coagulation pathways leading to microvascular thrombosis, consumption of clotting factors, and an increased risk of bleeding [2,3]. Sepsis associated DIC reflects the interaction between inflammation, endothelial injury, platelet activation, and impaired anticoagulant mechanisms [2]. The lungs are often the primary source of infection in patients who develop DIC, highlighting the close link between pulmonary inflammation and systemic coagulation disturbances [3,7]. The reported incidence of DIC in sepsis varies depending on diagnostic criteria but has been described in up to one third of patients with severe sepsis and septic shock [2,7]. Its presence is consistently associated with worse outcomes, including higher mortality, prolonged intensive care stay, and increased risk of bleeding complications [3]. Early recognition is therefore essential, although diagnosis remains challenging due to overlapping laboratory abnormalities seen in severe infection. Bleeding manifestations in DIC range from mild mucosal bleeding to life threatening hemorrhage. Gross hematuria is a recognized but less commonly emphasized presentation and may reflect severe consumption of coagulation factors and platelet dysfunction [7]. Hemorrhagic complications may involve multiple organ systems, including the central nervous system. Intracranial hemorrhage in the setting of sepsis associated DIC is rare but devastating and is associated with high mortality and poor neurologic outcomes [2,3]. Intraventricular hemorrhage in adults is particularly uncommon outside of trauma or vascular malformations, making its occurrence in septic DIC clinically significant. The pathophysiology underlying central nervous system bleeding in sepsis is complex. Endothelial dysfunction, disruption of the blood brain barrier, cytokine mediated injury, and consumption coagulopathy all contribute to vascular fragility [3]. Hypotension and impaired cerebral autoregulation during septic shock may further increase the risk of intracranial bleeding. These mechanisms highlight how systemic infection can lead to focal catastrophic complications even in the absence of primary neurologic disease. Management of sepsis associated DIC remains largely supportive and focused on treatment of the underlying infection [2,4]. Current recommendations emphasize early source control, appropriate antimicrobial therapy, and careful use of blood products based on bleeding risk rather than laboratory values alone [7]. Anticoagulant therapy remains controversial and is generally reserved for selected cases under specialist guidance [4]. Corticosteroids may be considered in refractory septic shock but do not directly treat DIC and must be used cautiously in patients with active bleeding [6]. Despite growing understanding of the mechanisms involved, sepsis related DIC continues to pose significant diagnostic and therapeutic challenges. Clinical presentations can evolve rapidly, and unusual bleeding patterns may delay recognition of the underlying coagulation disorder. Case reports remain valuable in highlighting rare complications and reinforcing the need for vigilance in patients with severe infection. This case describes a patient with pneumosepsis complicated by disseminated intravascular coagulation, intraventricular hemorrhage, and gross hematuria. The combination of severe pulmonary infection, systemic coagulopathy, and catastrophic bleeding illustrates the unpredictable nature of sepsis and underscores the importance of early recognition and multidisciplinary management. Reporting such cases contributes to improved awareness of rare but fatal complications and supports ongoing efforts to refine diagnostic and therapeutic strategies in sepsis associated coagulopathy.

## Case Presentation

### Patient's history and Physical Examination

This case involves a sixty seven year old Saudi man who presented to the emergency department with progressive shortness of breath and altered mental status. He had been in his usual state of health until five days prior to presentation, when he developed fever, productive cough, and generalized body aches. The cough was associated with yellow sputum and was accompanied by pleuritic chest discomfort on the right side. He initially attributed his symptoms to a common respiratory infection and managed them at home with oral paracetamol and increased fluid intake. Over the following two days, he noted worsening fatigue and reduced appetite, spending most of the day resting. His family reported that he became less interactive and appeared more lethargic than usual. On the day of presentation, his shortness of breath worsened significantly. He became breathless while walking short distances inside the house and required frequent pauses to catch his breath. His family noticed that he was speaking less and appeared confused at times, answering questions slowly and inaccurately. He also complained of reduced urine output since the previous day and dark colored urine. Several hours before arrival, he passed visibly bloody urine, which alarmed the family and prompted them to bring him to the hospital. He denied any dysuria, flank pain, or prior episodes of hematuria. There was no history of recent trauma, urinary catheterization, or anticoagulant use. His past medical history was significant for type 2 diabetes mellitus for more than fifteen years, hypertension, ischemic heart disease with prior percutaneous coronary intervention, and stage 3 chronic kidney disease. He also had a history of dyslipidemia and obesity. He was compliant with his medications, which included metformin, basal insulin, amlodipine, bisoprolol, atorvastatin, and aspirin. He was not on warfarin or direct oral anticoagulants. He had no known bleeding disorders. He had been hospitalized once in the past for community acquired pneumonia several years earlier but had no history of recurrent infections. He was a former smoker with a twenty pack year history and had quit more than ten years ago. He did not consume alcohol. He lived with his family and was functionally independent prior to this illness. There was no recent travel, no exposure to sick contacts, and no known contact with tuberculosis. He had not received antibiotics before presentation. His vaccination history was incomplete, and he was unsure about prior pneumococcal vaccination. There was no family history of bleeding disorders, stroke, or chronic lung disease. On arrival to the emergency department, he appeared ill and fatigued. He was drowsy but arousable to voice and able to answer simple questions. His temperature was 38.6°C, heart rate 112 beats per minute, blood pressure 94 over 58 millimeters of mercury, respiratory rate 26 breaths per minute, and oxygen saturation 89 percent on room air. He was placed on supplemental oxygen via nasal cannula with improvement in saturation. He was using accessory muscles of respiration and spoke in short sentences. Cardiovascular examination revealed tachycardia with a regular rhythm and no audible murmurs. Peripheral pulses were palpable but weak. Capillary refill was delayed. There was no peripheral edema. Respiratory examination showed reduced air entry over the right lower lung zone with coarse crackles. The left lung fields had preserved air entry without wheezing. There was no chest wall tenderness. The abdomen was soft and mildly distended, with no focal tenderness. The bladder was not palpable. Examination of the external genitalia was unremarkable, with no visible source of bleeding. Neurological examination showed no focal deficits. He was disoriented to time but recognized family members. Motor strength was symmetric, and there were no signs of meningeal irritation. Skin examination revealed cool extremities and scattered ecchymoses over the forearms, which the family stated were new. There was no petechial rash. Overall, the clinical picture was consistent with severe infection complicated by circulatory compromise and evolving systemic involvement, prompting urgent resuscitation and further evaluation.

### Diagnostic Workup

Following initial assessment in the emergency department, the patient demonstrated rapid clinical deterioration. Despite supplemental oxygen, he remained tachypneic with increasing oxygen requirements and persistent hypotension. His mental status fluctuated, with periods of increased drowsiness and reduced responsiveness. Given the severity of his presentation and concern for sepsis with evolving organ dysfunction, he was transferred to the intensive care unit for close monitoring and further evaluation. Baseline laboratory investigations on admission showed marked leukocytosis with neutrophil predominance. Inflammatory markers were significantly elevated. Renal function tests revealed a rise in serum creatinine above his known baseline, consistent with acute kidney injury on chronic kidney disease. Liver enzymes were mildly elevated, with a disproportionate rise in total bilirubin. Serum lactate was elevated on admission and remained persistently high on repeat measurements, indicating ongoing tissue hypoperfusion. Arterial blood gas analysis demonstrated hypoxemia with respiratory alkalosis and a concurrent metabolic acidosis, in keeping with severe sepsis and respiratory compromise. Chest imaging was reviewed early in the course. A prior baseline chest radiograph from routine outpatient care showed clear lung fields with no focal opacities. In contrast, a chest radiograph obtained on arrival to the emergency department demonstrated bilateral, multifocal patchy air space opacities involving both lung fields, consistent with extensive pneumonic changes. These comparative findings are shown together in Figure 1, highlighting the acute nature of the pulmonary process.

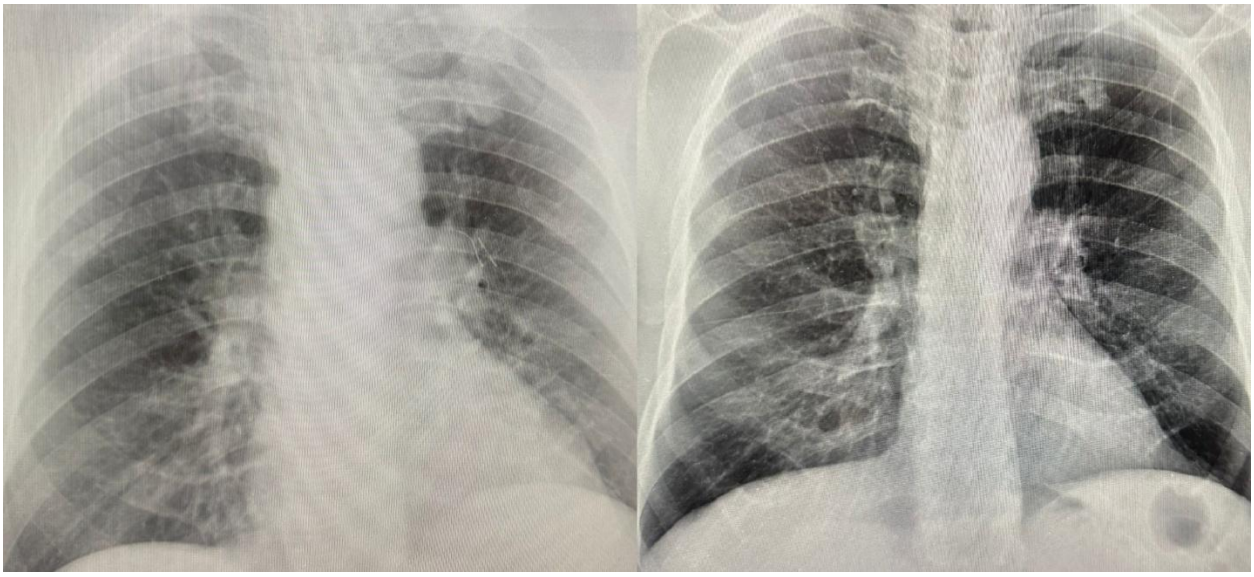

**Figure 1:** comparison between baseline\normal chest X-ray on the right and new chest X-ray on the left demonstrating multiple pneumonic patches bilaterally.

Over the first twenty four hours of intensive care admission, the patient's respiratory status continued to worsen, with escalating oxygen requirements. During this period, he developed overt bleeding manifestations. Nursing staff noted progressive discoloration of urine in the urinary catheter bag, which evolved into frank gross hematuria. An image documenting the gross hematuria is shown in Figure 2.

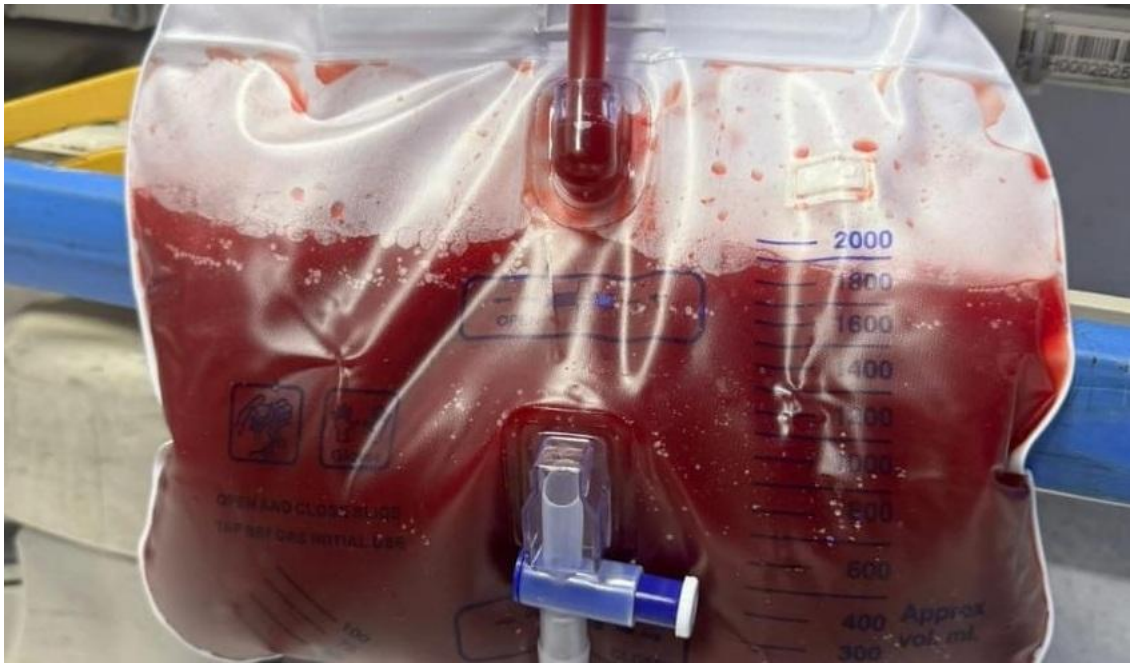


**Figure 2**: urine bag demonstrating rapid-onset gross hematuria.

There was no evidence of catheter related trauma, obstruction, or prior urological disease. Bleeding was also observed from venipuncture sites and around peripheral intravenous cannulae. Serial laboratory testing revealed a progressive fall in hemoglobin levels without evidence of external blood loss. Platelet counts declined rapidly over a short period. Coagulation studies showed marked prolongation of prothrombin time and activated partial thromboplastin time, with rising international

normalized ratio values. Fibrinogen levels were reduced, and D dimer levels were markedly elevated. These abnormalities progressed on repeat testing and were consistent with disseminated intravascular coagulation. Peripheral blood smear confirmed thrombocytopenia without features of microangiopathic hemolysis. Renal function continued to deteriorate in parallel with the coagulopathy. Serum creatinine and urea levels rose further, and urine output decreased despite confirmed catheter patency. Urinalysis demonstrated numerous red blood cells without casts. There was no pyuria or bacteriuria on microscopy. Liver function tests showed worsening hyperbilirubinemia with mild transaminitis, consistent with sepsis related hepatic dysfunction. On the second day of intensive care admission, the patient developed acute neurological deterioration. He became abruptly more obtunded, with a noticeable decline in Glasgow Coma Scale score. He was noted to have new onset right sided weakness and anisocoria on examination. There was no history of trauma or seizure activity. In view of the known coagulopathy and new focal neurological signs, urgent neuroimaging was obtained. Computed tomography of the brain revealed extensive intraventricular hemorrhage with layering of blood within both lateral ventricles. There was early ventricular dilatation suggestive of evolving hydrocephalus, without significant midline shift at the time of imaging. These findings are demonstrated in Figures 3.

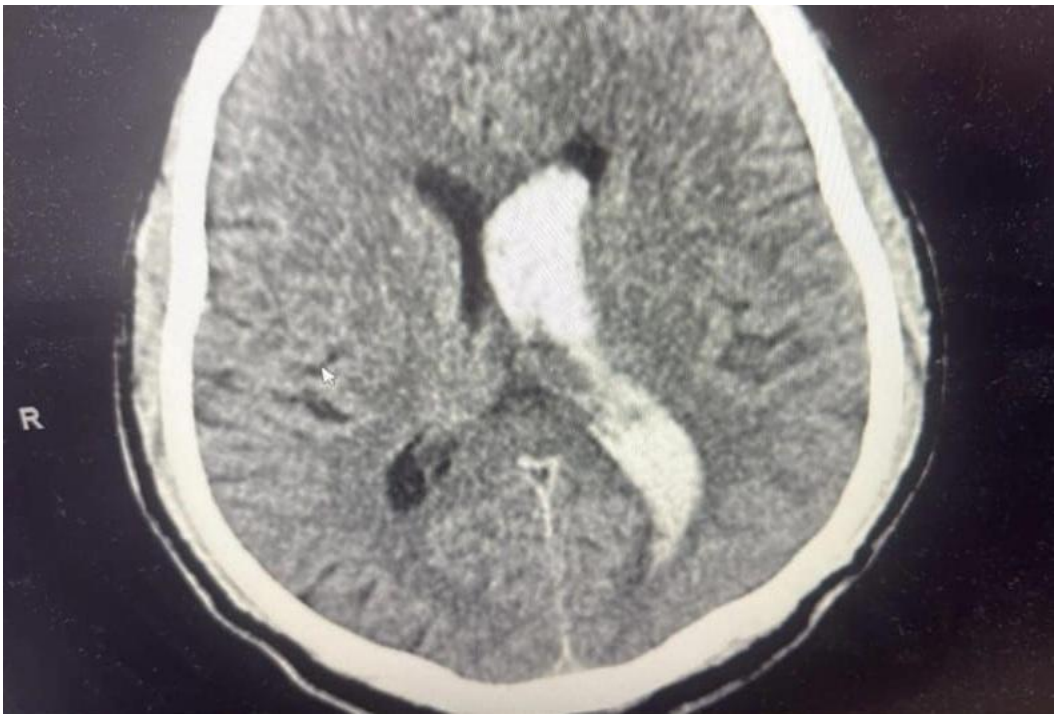


**Figure 3:** brain CT revealing spontaneous intraventricular hemorrhage.

No underlying vascular malformation or mass lesion was identified. The imaging was consistent with spontaneous intracranial hemorrhage occurring in the setting of severe coagulopathy. Blood cultures drawn on admission subsequently grew Streptococcus pneumoniae, sensitive to standard beta lactam antibiotics, confirming pneumococcal sepsis as the underlying source of infection. Repeat laboratory testing following the neurological event showed further deterioration in coagulation parameters, with critically low platelet counts and worsening prolongation of clotting times. Inflammatory markers remained markedly elevated. Overall, the patient's hospital course was characterized by rapidly progressive pneumosepsis complicated by disseminated intravascular coagulation, severe bleeding manifestations including gross hematuria, and catastrophic intracranial hemorrhage. The close temporal relationship between pulmonary infection, laboratory evidence of coagulopathy, and subsequent bleeding highlights the aggressive nature of sepsis associated disseminated intravascular coagulation in this case.

**Management course**

Management focused on rapid stabilization, treatment of severe sepsis, and control of life threatening complications related to disseminated intravascular coagulation and intracranial hemorrhage. Upon transfer to the intensive care unit, the patient was managed in a negative pressure isolation room with continuous cardiac monitoring, invasive blood pressure monitoring, and strict input and output charting. High flow supplemental oxygen was initiated due to worsening hypoxemia, with frequent reassessment of respiratory effort and gas exchange. Given the diagnosis of pneumosepsis, broad spectrum intravenous antibiotics were started immediately after blood cultures were obtained and later tailored once culture results confirmed Streptococcus pneumoniae sensitive to beta lactam therapy. Aggressive fluid resuscitation was undertaken early in accordance with sepsis management guidelines, using balanced crystalloids to restore perfusion while carefully monitoring for fluid overload

in the setting of worsening lung involvement. Despite adequate volume resuscitation, the patient remained hypotensive and required initiation of vasopressor support to maintain adequate mean arterial pressure. Lactate levels were monitored serially to assess response to therapy, although they remained persistently elevated, reflecting ongoing circulatory failure. As laboratory evidence of disseminated intravascular coagulation became apparent and bleeding manifestations progressed, management shifted to close coordination with hematology and critical care teams. Platelet transfusions were administered in response to severe thrombocytopenia and active bleeding. Fresh frozen plasma and cryoprecipitate were given to address prolonged clotting times and low fibrinogen levels, guided by serial coagulation profiles and clinical bleeding rather than laboratory thresholds alone. Red blood cell transfusions were provided as hemoglobin levels declined in the context of ongoing hematuria and intracranial bleeding. All antiplatelet agents were discontinued. Renal function continued to worsen, with declining urine output despite adequate resuscitation. Nephrology was consulted early, and renal replacement therapy was initiated when metabolic acidosis and fluid overload became difficult to control with conservative measures. Gross hematuria persisted throughout this period, requiring frequent catheter care and monitoring for obstruction. Following the sudden neurological deterioration, neurosurgery and neurology teams were urgently involved. The intracranial hemorrhage was managed conservatively due to the extensive intraventricular involvement, severe coagulopathy, and poor overall physiological reserve. Serial neurological examinations and repeat imaging were used to monitor for progression. Measures were taken to reduce secondary brain injury, including head elevation, careful control of blood pressure, and avoidance of factors that could worsen intracranial pressure. Surgical intervention was deemed not feasible given the diffuse nature of the hemorrhage and the uncontrolled coagulation disorder. Despite maximal supportive care, the patient's condition continued to deteriorate. He developed refractory shock with escalating vasopressor requirements and worsening oxygenation consistent with severe sepsis related lung injury. Laboratory parameters showed persistent coagulopathy, rising inflammatory markers, and progressive multiorgan failure involving the respiratory, renal, hepatic, and central nervous systems. Family meetings were held regularly to provide updates on the patient's condition and prognosis. Given the extent of organ failure, catastrophic intracranial hemorrhage, and lack of meaningful neurological recovery, the prognosis was discussed as extremely poor. After multidisciplinary discussion and in accordance with the family's wishes, goals of care were revisited. A decision was made to transition to comfort focused care once it became clear that further escalation would not alter the outcome. The patient passed away in the intensive care unit several days after admission due to complications of severe pneumococcal sepsis, disseminated intravascular coagulation, and intracranial hemorrhage. This case highlights the aggressive and often fatal course that pneumosepsis can take when complicated by profound coagulopathy and central nervous system bleeding, even with timely, guideline based critical care management.

**Discussion**

This case illustrates a severe and rapidly progressive form of pneumosepsis complicated by disseminated intravascular coagulation and catastrophic intracranial bleeding. It highlights several important clinical lessons related to early recognition of sepsis, the evolution of sepsis associated coagulopathy, and the prognostic implications of bleeding complications in critically ill patients. Sepsis is currently defined as life threatening organ dysfunction caused by a dysregulated host response to infection [1]. Pneumonia remains one of the most common sources of sepsis worldwide and is frequently associated with progression to septic shock and multiorgan failure, particularly in older adults with multiple comorbidities [5,8]. In this patient, advanced age, diabetes mellitus, chronic kidney disease, and cardiovascular disease likely contributed to impaired immune response and reduced physiologic reserve, predisposing him to rapid deterioration once infection developed [8]. Streptococcus pneumoniae remains a leading cause of community acquired pneumonia and pneumosepsis, despite widespread vaccination programs [5]. Pneumococcal sepsis is well recognized for its ability to trigger an intense inflammatory response, leading to endothelial dysfunction, capillary leak, and activation of coagulation pathways [2]. This case reinforces the need for early suspicion of severe disease in patients presenting with respiratory infection and subtle signs of systemic involvement, such as confusion, hypotension, and reduced urine output. Disseminated intravascular coagulation is a common and serious complication of sepsis, particularly in patients with septic shock [2,7]. It represents a complex interaction between inflammation and coagulation, characterized by widespread thrombin generation, consumption of clotting factors, platelet activation, and impairment of endogenous anticoagulant pathways [3]. Studies suggest that up to thirty to forty percent of patients with severe sepsis develop laboratory evidence of DIC, and its presence is associated with significantly increased mortality [2,3]. In this patient, the rapid decline in platelet count, prolongation of clotting times, reduced fibrinogen, and markedly elevated D dimer levels were classic features of sepsis associated DIC [7]. An important clinical cue highlighted by this case is the appearance of bleeding manifestations as an early sign of advanced coagulopathy. While DIC often begins with a predominantly prothrombotic phase, progression to consumption coagulopathy can result in spontaneous bleeding [2]. Gross hematuria, as seen in this patient, is a recognized but underreported manifestation and should prompt urgent evaluation for systemic coagulopathy rather than being attributed solely to urological causes [7]. Bleeding from venipuncture sites and rapid hemoglobin decline further supported the diagnosis and underscored the severity of the condition. The development of intracranial hemorrhage represents one of the most devastating complications of sepsis associated DIC. Central nervous system bleeding is uncommon but carries extremely poor prognosis [3]. Intraventricular hemorrhage in adults is particularly rare outside of trauma or structural brain lesions, making

its occurrence in this case notable. The pathophysiology involves a combination of severe coagulopathy, endothelial injury, cytokine mediated disruption of the blood brain barrier, and impaired cerebral autoregulation during septic shock [3]. These mechanisms increase vascular fragility and susceptibility to spontaneous hemorrhage even in the absence of hypertension or trauma. Current sepsis management guidelines emphasize early antimicrobial therapy, aggressive hemodynamic support, and close monitoring for organ dysfunction [1,6]. However, there is no definitive therapy that reverses sepsis associated DIC, and management remains largely supportive and focused on treating the underlying infection [2,4]. Blood product transfusion is recommended in the presence of active bleeding or high bleeding risk, rather than based solely on laboratory abnormalities [7]. This case demonstrates that even with timely and guideline based care, outcomes can remain poor once severe coagulopathy and intracranial bleeding develop.

Another important aspect highlighted by this case is the limitation of invasive interventions in the setting of uncontrolled coagulopathy. Neurosurgical options for intraventricular hemorrhage, such as external ventricular drainage, may be contraindicated when bleeding risk is prohibitive and overall prognosis is poor. Early involvement of multidisciplinary teams and clear communication with family members regarding prognosis are essential components of care in such scenarios. From a prognostic perspective, sepsis complicated by DIC and central nervous system hemorrhage carries a very high mortality rate [2,3]. Advanced age, preexisting comorbidities, persistent shock, and multiorgan failure further worsen outcomes [8]. This case underscores the importance of early recognition of sepsis and vigilant monitoring for evolving coagulopathy, particularly in high risk patients with pneumococcal infection. In conclusion, this case highlights the aggressive nature of pneumococcal sepsis and its potential to progress rapidly to disseminated intravascular coagulation and fatal bleeding complications. It reinforces key clinical lessons regarding early identification of sepsis, recognition of bleeding as a marker of advanced DIC, and the need for timely multidisciplinary management. Reporting such cases contributes to clinician awareness of rare but catastrophic complications and emphasizes the ongoing challenges in managing severe sepsis despite adherence to current guidelines.

## Conclusion

This case highlights the critical need for early recognition of sepsis, especially in older patients with pneumonia and comorbidities. Subtle signs like confusion, reduced urine output, and rising oxygen needs should prompt urgent evaluation. Bleeding manifestations such as gross hematuria in sepsis signal evolving disseminated intravascular coagulation and require prompt assessment. Intracranial hemorrhage, though rare, is a devastating complication that demands immediate neuroimaging when neurological changes occur. Despite guideline-based management, outcomes remain poor once multiorgan failure and severe coagulopathy develop. This case underscores the importance of vigilance, timely escalation, multidisciplinary care, and early family communication. Recognizing bleeding as a marker of advanced disease and responding swiftly are essential to improving patient outcomes in severe pneumosepsis.

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