Case report

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Salmonella septic shock associated with DIC and thrombocytopenia in a young adult

Mohamed Elsamman Ahmed[1]

Abstract

***Background*: This case report presents the clinical course and management of a 27-year-old male patient admitted with symptoms of fever, fatigue, diarrhea, and mild pallor. The patient exhibited signs of septic shock, including hypotension, tachypnea, and elevated inflammatory markers.**

***Case presentation*: Initial diagnosis revealed sepsis-associated thrombocytopenia and further investigations were conducted to exclude other infectious causes. The patient had a recent travel history to India. Blood culture results confirmed Salmonella infection, with the identified strain being resistant to fluoroquinolones but susceptible to ceftriaxone and meropenem. Imaging studies revealed findings consistent with typhlitis, and the patient exhibited pancytopenia and neutropenia, indicating immune compromise.**

***Management and outcome*: The patient received intravenous fluids and empirical antibiotics after culture collection and closely monitoring laboratory parameters. Norepinephrine administration was initiated to stabilize blood pressure, and intravenous steroids were given to reduce inflammation. Apheresis platelets were given due to low critical platelet count and associated lower gastrointestinal bleeding. Over the course of ten days, the patient showed positive progress, with decreased fever, cessation of diarrhea, and reduced inflammatory markers. Norepinephrine support was gradually tapered, and oral medications were initiated. Regular follow-up appointments were recommended for monitoring and adjustment of the treatment plan.**

***Conclusion*: This case highlights the challenges and multidisciplinary approach involved in managing septic shock associated with disseminated intravascular coagulation and thrombocytopenia, which was complicated by neutropenic enterocolitis (typhlitis). Prompt diagnosis, appropriate antibiotic therapy, supportive care, and close monitoring of laboratory parameters were crucial in optimizing patient outcomes. Further research and clinical studies are warranted to improve understanding and management of this complex condition.**

Key words: Septic shock, thrombocytopenia, disseminated intravascular coagulation (DIC), intravenous fluids, empirical antibiotics, norepinephrine, typhlitis.

[1] Burjeel Farha Hospital, Al Ain, United Arab Emirates

Address for correspondence:
Mohamed Elsamman Ahmed
Burjeel Farha Hospital, Al Ain, United Arab Emirates
MSc Critical Care Medicine, Alexandria University
MRCPUK, London
Email: docmsamman@hotmail.com
ORCID: 0009-0005-7913-075X

Introduction

Septic shock is a condition caused by some types of bacteria, virus, or fungi that enter the bloodstream and causes leakage; this leads to organ damage. Salmonella septic shock is a potentially life-threatening infection caused by bacteria of the Salmonella genus. (1) It is characterized by bacteria in the bloodstream, which triggers an inflammatory response leading to septic shock. Septic shock is a severe condition that can lead to multiple organ failure, including the lungs, kidneys, and liver. The term "sepsis"

has been described as "the systemic inflammatory response syndrome (SIRS) to a microbial infection," with SIRS being at least two of the following: leukocytosis, leukopenia, or neutrophilia, as well as tachycardia (rapid pulse), pyrexia (fever), or hypothermia. (2) The incidence of deaths and mortality rates have decreased over the past few decades; however, the prevalence of septic shock and the severity of the condition have both increased. As a result of this rate and the severity of the disease, more people are surviving more severe illnesses. The decrease in mortality from severe sepsis and septic shock has been partially attributed to advancements in identification and protocol-based early response. The recommended course of treatment for septic shock involves a multidisciplinary approach and typically includes the following components: early recognition and source control, broad-spectrum antibiotics, rapid administration of intravenous fluids, vasopressors, and other supportive care. (3)

In some cases, Salmonella septic shock can also be associated with disseminated intravascular coagulation (DIC) and thrombocytopenia. (4) DIC is a condition in which blood clots form throughout the body's small blood vessels, leading to organ damage and bleeding. (5) The appearance of fibrin-related markers (FRMs) in DIC reflects microvascular modifications. Despite lacking a gold standard and a particular biomarker for diagnosing DIC, the condition may be accurately identified using straightforward scoring systems based on widely accessible common hemostatic measures. DIC may complicate around 35% of sepsis/septic shock cases caused by Gram-negative and Gram-positive bacteria. (6) Thrombocytopenia is a decrease in the number of platelets in the blood, which can lead to abnormal bleeding. (7) Thrombocytopenia is a common onset of sepsis and associates itself with inflammatory responses. (7) The development of thrombocytopenia during sepsis is complex and probably involves many factors. For example, endothelial dysfunction is a major consequence of sepsis and plays a critical role in platelet activation and consumption. (8) This activation, resulting in aggregation, is locally upregulated by cytokine production. However, other cytokine-driven mechanisms are also conceivable, including disseminated intravascular coagulation or platelet destruction. (9)

Septic shock is a life-threatening condition that is a severe response to infection, often caused by bacteria, including Salmonella. During an infection, the body's immune system releases inflammatory molecules called cytokines to fight off the invading pathogens. However, the immune response in sepsis becomes dysregulated, leading to widespread inflammation throughout the body. (10) This excessive immune response can trigger a cascade of events that can damage blood vessels and impair normal blood clotting mechanisms. It can cause small blood clots in the microvasculature, leading to DIC. (10,11) DIC is a condition characterized by excessive clotting, platelet depletion, and clotting factors. (12) A young adult with Salmonella septic shock associated with DIC and thrombocytopenia would require prompt and immediate medical treatment. The first step would be identifying and isolating the Salmonella bacteria and starting antibiotic therapy. (13) The patient would also need supportive care, such as fluids to maintain blood pressure, oxygen to support breathing, and medications to support organ function. In addition, the patient would require monitoring for signs of organ dysfunction and bleeding. Blood products and clotting factor replacement may be necessary to manage DIC and thrombocytopenia. (13-15)

The prognosis for Salmonella septic shock associated with DIC and thrombocytopenia depends on several factors, including the severity of the infection, the promptness of treatment, and the presence of underlying medical conditions. (16) Salmonella septic shock associated with DIC and thrombocytopenia is crucial for optimal patient outcomes. Early recognition and treatment of the infection can prevent the development of sepsis, septic shock, and multiple organ dysfunction syndrome (MODS), which are associated with high morbidity and mortality. (17) Early recognition also allows for early initiation of appropriate antibiotic therapy, which can decrease bacterial load, reduce the risk of complications, and shorten the duration of hospitalization. Delayed or inadequate antibiotic treatment can lead to a more severe disease course and an increased risk of complications such as organ failure, acute respiratory distress syndrome (ARDS), and DIC. (18)

This case report aims to highlight the atrocity of this condition and how it can impact the whole body's functions, eventually leading to organ failure and increased mortality rate if left untreated and unmanaged. This report also highlights the benefits of correct antibacterial and antimicrobial approaches to treat the condition with other closely related management techniques.

Case presentation

This case study details a 27-year-old male patient who was admitted complaining of fever, weariness, diarrhea, and moderate pallor. The patient experienced watery and frequent diarrhea, accompanied by sweating during the fever episodes. No history of

jaundice or significant medical conditions was reported. The patient had recently traveled to India approximately one month prior. Initial examination revealed the patient to be underweight, with a fever above 39 degrees Celsius, mild tachypnea, and a rapid heart rate exceeding 125 beats per minute. Clear chest examination, but generalized abdominal tenderness was observed. The patient was sent for initial laboratory screening, and the results of the tests indicated elevated inflammatory markers and sepsis markers, along with significantly elevated D-dimer levels (>9 ug/ml), International Normalized Ratio (INR) (>1.47), and prolonged activated partial thromboplastin time (aPTT). The initial diagnosis was sepsis-associated thrombocytopenia, and further investigations were planned to exclude malaria, brucellosis, dengue fever, and enteric fever. The management plan included blood and urine culture screenings, malaria screening with each fever spike, lactate and procalcitonin (PCT) testing, intravenous fluids, and empirical antibiotic coverage. Gastroenterology consultation was sought to continue with the planned management approach.

On hospital ICU day 1, the patient persisted with symptoms of fatigue and high-grade fever accompanied by intermittent episodes of watery diarrhea mixed with blood. Liver enzyme levels were abnormal, and the patient exhibited a high positive PCT result, and a low platelet count of approximately 70,000. The previously established treatment plan was continued, focusing on liberal intravenous fluid administration and the initiation of empirical combined antibiotics. Close monitoring and follow-up were scheduled for the blood culture reports. Enterocolitis, sepsis colitis, and neutropenic sepsis were suspected and included in the differential diagnosis.

On hospital ICU day 2, the patient continued to experience recurring fever and abdominal pain, which was now accompanied by severe bloody diarrhea. Upon chest examination, diminished air entry was noted in both lung bases. The decision was made to initiate apheresis platelets and arrange for a contrast-enhanced chest and abdomen computerized tomography (CT) scan to investigate the possibility of abscess, collection, or malignancy. The management plan remained unchanged at this stage. Despite receiving intravenous boluses and aggressive initial management, including corticosteroids, the patient's mean arterial pressure (MAP) dropped and stayed around 55 mmHg, with systolic blood pressure (SBP) hovering around 80 mmHg. Norepinephrine infusion was initiated at 0.01 ug/kg/min and titrated to achieve an MAP target greater than 65 mmHg.

On hospital ICU day 3, the blood culture results revealed the presence of Salmonella infection in the patient's bloodstream. The strain of salmonella identified was found to be resistant to fluoroquinolones but susceptible to ceftriaxone and meropenem, two different classes of antibiotics. This information was crucial in modifying the antibiotic treatment plan, ensuring that the prescribed medications effectively targeted the specific strain of salmonella causing the infection. In addition to the blood culture results, further blood tests revealed pancytopenia, a condition characterized by reduced levels of all blood cell types, and neutropenia, characterized by a low count of neutrophils, a type of white blood cell essential for fighting off infections. These findings indicated that the patient's immune system was compromised, leaving them vulnerable to bacterial infections. The imaging study, specifically a contrast-enhanced CT scan of the abdomen, provided important insights into the patient's condition. It revealed the thickening of the cecum, the initial part of the large intestine, and lymphadenopathy, an abnormal enlargement of lymph nodes. These findings indicated typhlitis, also known as neutropenic enterocolitis, a serious complication in individuals with weakened immune systems, particularly those experiencing neutropenia. The clinical correlation between the CT findings and the patient's symptoms further supported this diagnosis. To manage the patient's condition, the medical team continued the administration of norepinephrine, a medication used to stabilize blood pressure in cases of septic shock. Intravenous steroids were also given to help reduce inflammation and support the patient's immune response. The combination of these interventions aimed to stabilize the patient's condition and provided support during this critical phase of the illness. From hospital ICU day 4 to day 9, the patient exhibited positive progress in their recovery. Fever spikes became less frequent, indicating a reduction in the intensity of the infectious process. The patient also experienced a cessation of diarrhea, which suggested an improvement in gastrointestinal symptoms. Additionally, the levels of inflammatory markers in the patient's blood decreased, indicating a decrease in the overall inflammation associated with the infection.

As the patient's condition improved, the medical team gradually reduced the dose of norepinephrine infusion until it was no longer needed. This demonstrated the patient's stabilization and the restoration of their blood pressure without needing medication support. With the patient's condition becoming more stable, the physician decided to transition the medications from intravenous to oral administration, enabling the patient to continue their treatment in an outpatient setting. The physician recom-

mended transferring the patient to an inpatient facility to ensure ongoing monitoring and appropriate follow-up. This would allow for closer observation, further evaluation, and necessary care to support the patient's recovery. Regular follow-up appointments were advised to track the patient's progress and made any adjustments to the treatment plan as needed.

Findings

The patient underwent treatment and management for ten days in the hospital, and his complete blood count (CBC) and biomarkers for potential septic shock associated with thrombocytopenia were regularly checked. Laboratory tests for CBC, C-reactive protein (CRP), PCT, lactate dehydrogenase (LDH), alanine transaminase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALK-P), total bilirubin (BILIR-T), INR, aPTT, and D-dimer were done on the patient for better treatment of the condition.

Throughout the patient's hospitalization in the intensive care unit (ICU), the results of their CBC were done for better follow-up of the case. The recorded values for hemoglobin (Hb), white blood cells (WBCs), and platelets (PLAT) over several days are given in **Table 1**.

These CBC results provided information about the patient's blood cell counts, including red blood cells, white blood cells, and platelets. These were important indicators of the patient's overall health and immune response. The recorded values showed fluctuations in these blood cell parameters over time, which may reflect the patient's response to treatment, the progression of their underlying condition, and the efficacy of interventions to address their health concerns.

Several laboratory tests were conducted throughout the patient's hospitalization in the ICU to assess their inflammatory and biochemical markers. The results of CRP, PCT, and LDH tests are as follows in **Table 2**.

These laboratory test results provided valuable information regarding the patient's inflammatory response and cellular damage. Elevated levels of CRP and PCT indicated an ongoing inflammatory process, while LDH levels reflected cellular damage or breakdown. The fluctuations observed in these markers over time may suggest the progression of the patient's condition and response to treatment. These test results were crucial in monitoring the patient's clinical course and guiding the healthcare team in making informed decisions regarding further management and intervention.

During the course of their stay in the ICU, the patient had their liver profile evaluated. The recorded values for ALT, AST, ALK-P, and BILIR-T on different ICU days are provided in **Table 3**.

These hepatic profile results provided insights into the patient's liver function. ALT and AST are enzymes typically found within liver cells, and elevated levels may indicate liver damage or injury. ALK-P is an enzyme associated with the biliary system, and its elevation can indicate liver or biliary tract dysfunction. BILIR-T represents the total bilirubin level, which can increase in cases of liver disease or impaired bilirubin metabolism. Monitoring these markers assisted in evaluating the patient's liver function and guiding the management and treatment plan.

Lastly, the patient was assessed for his coagulation profile. The recorded values for INR, aPTT, and D-dimer on different ICU days are given in **Table 4**.

These coagulation profile results provided information about the patient's blood clotting ability and fibrinolysis (breakdown of blood clots). The INR measures how long it takes for the blood to clot compared to a reference range. A higher INR value indicates a delay in clotting. aPTT measures the time it takes for blood to clot in response to specific activators, and prolonged aPTT may indicate a deficiency or dysfunction in certain clotting factors. D-dimer is a marker of fibrinolysis and is elevated when there is ongoing clot formation and breakdown. These values helped assess the patient's coagulation status, guide appropriate treatment, and monitor their response to therapy.

Results

During the patient's ICU stay, a comprehensive assessment of their laboratory results was conducted to evaluate various aspects of their health. The patient's CBC revealed fluctuations in hemoglobin, white blood cell count, and platelet levels over time. In terms of inflammatory markers, CRP and PCT showed initially elevated values (3.72 and 2.61, respectively), that gradually decreased, indicating a response to treatment. Hepatic profile analysis indicated fluctuating levels of liver enzymes (ALT, AST), ALK-P, and BILIR-T, suggesting potential liver dysfunction. The coagulation profile displayed varying INR, aPTT, and D-dimer levels, which reflected the patient's clotting ability and fibrinolysis. These findings collectively contributed to a comprehensive understanding of the patient's health status, guided clinical decision-making, and helped monitor their response to therapy. The first antibiotic course, which included Augmentin™ and ciprofloxacin, was unsuccessful in managing the septic process. There was no clinical improvement reported

even after the announcement of culture results and the subsequent treatment of ceftriaxone suited to the culture findings. The antibiotic was changed to meropenem, which resulted in a significant improvement in the patient's health. The patient was treated for thrombocytopenia using high-dose steroids and apheresis platelet transfusion. The case involved organ dysfunctions in the hepatic and homeostatic systems. A rare complication, neutropenic enterocolitis (typhlitis), was identified, adding complexity to the clinical presentation.

CT abdomen and pelvis with contrast

Fatty stranding with mesenteric lymphadenopathy was seen around the free fluid in the right abdomen and the pelvis appendix looked elongated and at the borderline but could not be rolled out. Right mild pleural effusion caecum could indicate inflammatory changes for clinical correlation (**Figure 1A and B**).

Ultrasound abdomen

Right pleural effusion noted. A thickened cecal wall and fatty stranding with mesenteric lymphadenopathy in the right lower abdomen could indicate inflammatory changes. However, the abdominal ultrasound was otherwise unremarkable. Clinical data correlation and follow-up were recommended for further assessment.

Discussion

Sepsis is a challenging health issue associated with acute organ dysfunction, a significant risk of death, and an imbalanced host reaction to an infection. Any infecting organism, such as Salmonella, Shigella, or Escherichia coli, can potentially cause sepsis. As a result, there are many different ways that the syndrome might manifest itself, and these variations greatly depend on the places and food you might intake. (19) Sepsis can develop in a community through various methods, either food, infected people, air-borne bacteria, or as a result of a stay in a hospital/medical facility. Approximately 80% of sepsis cases treated in hospitals begin in the community. Sepsis is most frequently caused by infections in the lung (64% of cases), abdomen (20%), and bloodstream (15%). (19) This condition's exact incidence and prevalence are difficult to determine due to its rarity and the lack of standardized diagnostic criteria. However, studies have reported that Salmonella-associated sepsis and septic shock incidence vary widely depending on the population studied, ranging from 0.4 to 4.7 cases per 100,000 person-years. (20)

Regarding the specific association of Salmonella septic shock with DIC and thrombocytopenia, the incidence and prevalence are even more difficult to estimate. One study conducted in South Korea found that overt DIC was present in 17.6% of patients with septic shock and was associated with a higher risk of mortality. (21) Another study conducted in Japan found that out of 1013 patients, the mortality rate of patients with severe sepsis was 21.5%, and the prevalence of DIC was 50.9%. (22) Septic shock and thrombocytopenia can be associated with the complex pathophysiological processes that occur during severe infections. Thrombocytopenia, or low platelet count, is a common feature of DIC and septic shock. (23) The inflammatory response and the formation of microclots in the blood vessels can consume and deplete platelets, resulting in thrombocytopenia. (24)

Additionally, the interaction between activated platelets and the damaged blood vessels can further contribute to the development of DIC. (25) Thrombocytopenia in septic shock and DIC can have significant clinical implications. It can lead to impaired blood clotting, increased bleeding tendencies, and organ dysfunction. (26) The combination of septic shock, DIC, and thrombocytopenia requires immediate medical intervention, including treatment of the underlying infection, supportive care, and, often, interventions to manage coagulation abnormalities. (27)

This case report represents a case of a 27-year-old young adult who came in for admission with a complaint of fever, fatigue, and diarrhea. His diarrhea was watery and contained traces of blood. The patient was sent for initial screening, and the results indicated high sepsis biomarkers, indicating a severe bacterial infection in the body. The patient had a recent travel history to India and exhibited elevated inflammatory markers, sepsis markers, and thrombocytopenia. Initial diagnosis suggested sepsis-associated thrombocytopenia, and further investigations that included CRP, PCT, LDH, ALP, ASP, ALK-T, and more were performed to exclude other possible infections. The patient's condition worsened, leading to the suspicion of typhlitis, a complication associated with neutropenia. The patient received appropriate antibiotic treatment based on blood culture results, and supportive measures were employed to stabilize his condition. Throughout the hospital stay, the patient's laboratory markers and clinical progress were closely monitored, leading to adjustments in treatment as necessary. The case highlights the importance of meticulous diagnosis, multidisciplinary management, and tailored interventions in addressing sepsis-associated thrombocytopenia and its complications.

Septic shock associated with DIC and thrombocytopenia can be managed and treated verily. Some management interventions include early recognition of the disease as it prevents it from worsening the case and reduces the chances of mortality. (28) Initiating appropriate antibiotics and timely administration of broad-spectrum antibiotics is crucial in treating the underlying infection and preventing its progression. (29) In fluid resuscitation, intravenous fluids are administered to restore adequate blood volume and optimize organ perfusion. (30) Vasopressor support may be required in persistent hypotension despite fluid resuscitation, such as norepinephrine, to maintain adequate blood pressure and tissue perfusion. (30,31) Source control, identification, and management of the primary source of infection, such as drainage of abscesses or removal of infected devices, play a critical role in controlling the infection and preventing further complications. (32) Supportive care of patients with septic shock and DIC often requires ICU monitoring and support, including respiratory support, renal replacement therapy if necessary, and close hemodynamic monitoring. (33) Blood component therapy and transfusion of blood products, such as platelets, fresh frozen plasma, and cryoprecipitate, may be considered in patients with severe thrombocytopenia and coagulation abnormalities. (34) Anticoagulation management, using anticoagulants in the context of DIC, is a complex decision requiring an individualized assessment based on the patient's clinical condition, bleeding risk, and coagulation profile. (35) Treatment of underlying conditions, identifying and addressing the underlying cause of sepsis, such as controlling the infection source or managing any predisposing factors, are essential for optimal management. (36) Close monitoring and laboratory tests, regular monitoring of vital signs, laboratory parameters (including complete blood count, coagulation profile, and inflammatory markers), and organ function are necessary to assess the response to treatment and guide further management decisions. (37) A collaborative approach, given the complexity of septic shock associated with DIC and thrombocytopenia, a multidisciplinary approach involving intensivists, infectious disease specialists, hematologists, and other relevant specialists are crucial for comprehensive patient care. (38)

Through a multidisciplinary approach, the targets of this case were to treat septic shock, find the underlying cause, deal with related problems, including thrombocytopenia, and improve the patient's clinical condition. The case report emphasized the significance of early detection, quick action, and continuous observation to enhance patient outcomes in severe sepsis and septic shock.

Strengths

The Salmonella septic shock associated with DIC and thrombocytopenia case presented a comprehensive clinical scenario, diagnostic workup, treatment approach, and clinical progression. It emphasized broad-spectrum antibiotics, fluid resuscitation, vasopressor support, and other supportive measures. It also emphasized the need for multidisciplinary care and the involvement of various medical specialties. The case provided insight into the potential outcomes and the importance of timely and appropriate management.

Limitations

This case of Salmonella septic shock associated with DIC and thrombocytopenia provided valuable insights, but several limitations were to be considered. The case was presented as a single clinical scenario, which might not capture the full spectrum of variations in presentation, management, and outcomes that could occur. Generalizability: The findings and outcomes presented might not be applied to all patients with Salmonella septic shock associated with DIC and thrombocytopenia. The case did not provide statistical data or cite specific references to support the incidence, prevalence, or outcomes of Salmonella septic shock associated with DIC and thrombocytopenia.

Conclusion

The presented case highlights the complexity and severity of sepsis-associated thrombocytopenia. The patient's symptoms, travel history, and laboratory findings pointed towards an infectious etiology, eventually identified as a Salmonella infection. Neutropenia and pancytopenia indicated an impaired immune response, further complicating the patient's condition. Timely diagnostic investigations, appropriate antibiotic adjustments based on resistance patterns, and aggressive management strategies were essential in addressing the patient's evolving clinical picture. This case underscores the importance of close monitoring, interdisciplinary collaboration, and tailored interventions to manage sepsis-associated thrombocytopenia and its complications effectively.

*The author used the CARE checklist when writing the report. (39)

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Ethical consent
Written informed consent was obtained from the patient for their anonymized information to be published in this article.

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Table 1. CBC findings

ICU day	Hb (g/dl)	WBCs ($x10^9$/l)	PLAT ($x10^3$/μl)
1	11	3.17	61
3	10.2	2.2	35
4	10.5	2.3	48
5	10.1	6.98	65
6	9.2	6.11	77
7	9.7	5.26	100

Legend: CBC=complete blood count; ICU=intensive care unit; Hb=hemoglobin; WBC=white blood cells; PLAT=platelets.

Table 2. Inflammatory and sepsis markers

ICU day	CRP (mg/dl)	PCT (μg/l)	LDH (U/l)
1	37.2	2.61	
2	57.4	12.4	
3	64	8.5	>1000
4			1406
5		9.39	876
6	15.4	4.05	682
7	3.9	1.24	
8		0.17	

Legend: ICU=intensive care unit; CRP=C-reactive protein; PCT=procalcitonin; LDH=lactate dehydrogenase.

Table 3. Hepatic profile of the patient

ICU day	ALT (U/l)	AST (U/l)	ALK-P (U/l)	BILIR-T (µmol/l)
1	316	696	105	18.9
2	590	1646	153	30
3	477	891	160	20
4	438	666	193	17.5
5	345	335	190	
6	302	291		
7	171	73	160	9.7

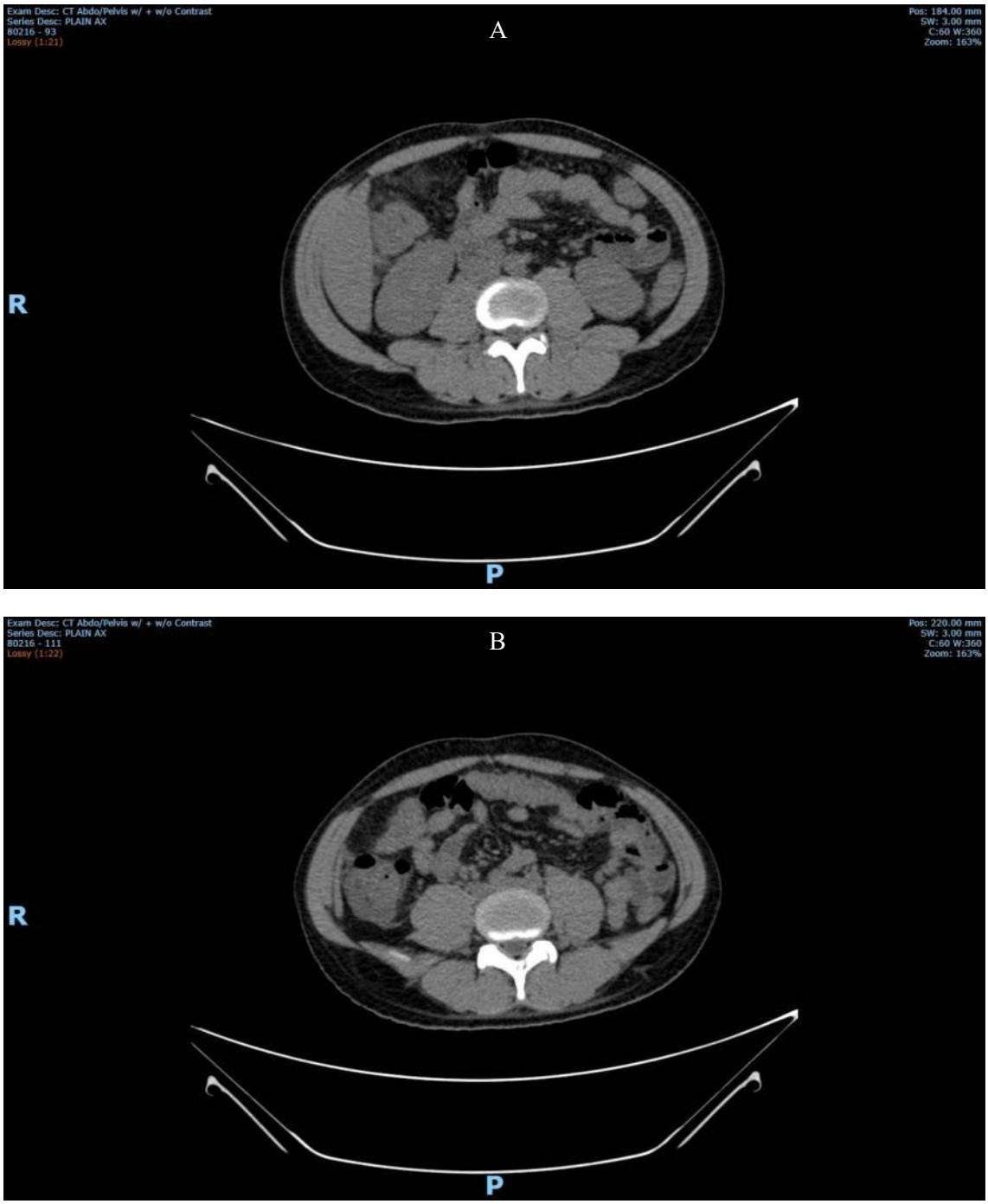
Legend: ICU=intensive care unit; ALT=alanine transaminase; AST=aspartate aminotransferase; ALK-P=alkaline phosphatase; BILIR-T=total bilirubin.

Table 4. Coagulation profile of the patient

ICU day	INR	aPTT (seconds)	D-dimer (µg/ml)
1	1.3		>9.0
3	1.47	41.3	
4	1.24	43.5	7.38
6	1.22	41.5	8.4
7	1.18	33.4	
8			7.81

Legend: ICU=intensive care unit; INR=International Normalized Ratio; aPTT=activated partial thromboplastin time.

Figure 1. Computerized tomography scan of abdomen/pelvis with and without contrast



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