DIC-Induced Septic Shock in A Patient with Multiple Comorbidities: A Case Report

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This case reports an 89-year-old male who presented with sepsis, disseminated intravascular coagulopathy, acute kidney injury, and upper GI bleeding to the emergency department. He was admitted to the ICU, where he was managed for septic shock, diabetic ketoacidosis, and coagulopathy then sent home on medications with an improved status.

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Informed Consent

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

Abstract

This case reports an 89-year-old male who presented with sepsis, disseminated intravascular coagulopathy, acute kidney injury, and upper GI bleeding to the emergency department. He was admitted to the ICU, where he was managed for septic shock, diabetic ketoacidosis, and coagulopathy, then sent home on medications with an improved status.

Keywords: Disseminated Intravascular Coagulopathy; septic shock; multiple comorbidities; case report

Key Clinical Message

In order to avoid missing the significant impacts of DIC and sepsis on vital organs, we implore physicians to have a high index of suspicion of DIC and, more critically, to begin therapy as soon as possible.

Background

Sepsis is systematic inflammatory response syndrome (SIRS) as a response to active infection in the host[1]. Exaggerated response in sepsis can result into multi-organ failure (MOF) syndrome that includes acute respiratory distress syndrome (ARDS) and acute kidney injury (AKI), shock and death [2]. when sepsis causes persisted hypotension despite adequate fluid resuscitation along with hypoperfusion or organic dysfunction this called septic shock [1]. One common complication of sepsis is Disseminated Intravascular coagulopathy (DIC), defines as acquired consumption of coagulation factor due to hemorrhage and thrombus formation with suppressed fibrinolysis. The key modulator of sepsis related DIC is thrombin, it connects inflammation and coagulations by stimulating the release of proinflammatory cytokines and chemokines from the immune and endothelial cells resulting in endothelial injury which exacerbates inflammation [3].

In intensive Care Unit (ICU), sepsis is a common cause of morbidity and mortality as mortality rates range from 40% in severe sepsis to 60% in septic shock. sepsis also responsible for 20% of AKI cases in ICU. Moreover, AKI occurs in 35-65% of ICU patients and increases their mortality rates three-to-five-folds when compared to patients without AKI[4]. Furthermore, sepsis was identified as risk factor for stress ulcer related gastrointestinal bleeding. For acute coronary syndrome (ACS) patients, sepsis can increase the gastrointestinal bleeding risk, and both sepsis and bleeding can reinforce each other. Thus, using of CRUSADE bleeding risk score for ACS is recommended as an acknowledged and common scoring system, and proton pump inhibitors (PPIs) are the preferred drugs to prevent gastrointestinal bleeding induced by sepsis in ACS patients [5].

The effect of diabetes mellites on infection especially among elderlies is well known, since diabetic patient have higher frequency for upper respiratory tract infection, urinary tract, skin, fungal and hospital acquired infection. Even of its known effect of impairing immunity and vasculature, the relation between diabetic patient and sepsis were not established, one cohort study done on non-ICU patients revealed that no difference in primary outcomes of septic patients with type 2 Diabetes mellitus (T2D) when compared to non-diabetics [6]. We present a case of acute DIC, sepsis, Upper GI bleeding and AKI in patient known to have diabetes and ACS.

Case Presentation

An 89-year-old male that presented to the emergency room (ER) with decreased oral intake and general weakness associated with nausea and decreased urine output for 1 day. At the day of presentation, he was hypotensive, tachycardic, the oxygen saturation was 98% and afebrile, he reported one episode of coffee ground vomiting with no abdominal or chest pain. The patient past medical history was significant for Diabetes Mellites, Hypertension, Ischemic heart disease, Seizure disorders, Old Cerebral Vascular Accident (CVA), Deep Venous Thrombosis (DVT) in his left arm. The patient also had a history of sepsis and retroperitoneal hematoma 3 months prior to this ER visit. his previous surgical history includes Bilateral hip and left knee replacement, right sided inguinal hernia repair and three stents for ischemic heart disease (IHD).

On physical exam the patient was conscious, alert, oriented to time and place, but he looked pale, the chest was clear with good bilateral air entry. On abdominal examination he had soft lax abdomen without

tenderness. he had a weak bilateral radial pulse. no lower limb edema. His glucose level and arterial blood gas (ABG) was consistent with Diabetic ketoacidosis (DKA) that was managed by IV fluids and endocrine consultation in the ER. The patient glucose level reached 20 mg/dl so the insulin was stopped, and he was started on 100cc /HR glucose saline His work up results included elevated D-Dimer, Fibrinogen degradation products, international normalized ratio (INR) and leukocytosis (lab results are summarized in table 1). In addition to hyperkalemia and elevated creatinine level. Soft tissue Ultrasound revealed anterior right sided chest hematoma\sout, so hematology department were consulted regarding Disseminated intravascular coagulation (DIC), and he was diagnosed with Acute kidney injury (AKI). his chest x- ray were normal and electrocardiogram (ECG) showed sinus tachycardia.

The decision was taken to admit the patient as a case of DIC, sepsis, upper gastrointestinal bleeding (UGIB), DKA, and AKI to the intensive care unit (ICU). The patient got packed red blood cells (RBCs), NOVO 7 (Factor VIIa, recombinant) two ampules twice daily, and tranexamic acid 1 gram three times IV after it was determined that he had DIC on the first day after being admitted to the ICU. The hematoma in the patient was treated conservatively with cold compressors and hemoglobin and hemophilia correction. The ICU's ABG result revealed compensatory metabolic acidosis and negative ketone levels in both the blood and urine.After the administration of the IV fluids the patient potassium reading dropped to 4.8, his creatinine level also started to drop, and the patient passed one litre of urine collected by Foleys catheter.

The urine culture showed mixed bacteriuria, so the patient started receiving Tienam® (IMIPENEM and CILASTATIN SODIUM) 250mg *3 IV. In order to prevent second UGI bleeding episode, the patient remained nothing by mouth (NPO) and started Proton Pump Inhibitor (PPI).On the second day of ICU admission, his haemoglobin elevated from 7.4 in ER to 8.1. he had normal ABG's reading, urea and creatinine readings were decreasing, and he stayed on PPI's. On the third day, the patient had hypokalaemia and hypocalcaemia (K: 3 mmol/l), (corrected Ca: 8.1), this was corrected by infusion of 40 milliequivalent (Meq) Potassium chloride (KCL) and 2mg of Magnesium sulphate (MgSO4) for hypokalaemia and vitamin D for hypocalcaemia. On the fourth day, He became tachypnic and reported chills. His blood pressure (BP) was 170/100, COVID-19 swabs and blood cultures were sent, and he was started on Perfalgan® (acetaminophen). His ABGs revealed PH of 7.47, Pco2 30 and Hco3 of 21. The patient was given DVT prophylaxis by intermittent pneumatic compression and his PPI dose was changed to twice a day. He was still off anticoagulants.

The patient's general condition and ABG improved on the fifth and sixth days, but there were no other noteworthy changes, therefore it was decided to transfer him to the floor. The patient's heart rate was 140, his blood pressure was 90/60, and his temperature was 36.5 on the seventh day at 4 pm. The patient's only complaint was weakness all around. The patient was started on normal saline 500ml, 2mg MgSO4 over 2 hours, and another 2mg MgSO4 over 24 hours after the ECG showed sinus tachycardia and labs showed K+ reading of 3.25 and Mg of 1.27. The patient's electrolytes and blood pressure improve until 8 o'clock after 50 Meq of KCL.when he develops hypotension again, so he was given another 500 ml of normal saline, septic workup was sent. Hemoglobin was 8.9 and cardiac enzymes were negative.

The patient got better on the eighth and ninth days, and his sinus tachycardia went away. He was then moved to the floor. When the patient was examined by the haematology department, he was stable and had no current complaints. The patient was in good health and had no complaints on the tenth day. His labs 9.1 Haemoglobin (Hb), 4.39 WBC, and 280 platelets. C-reactive protein (CRP) and creatinine:62 was moving downward. The patient was healthy, vitally stable, and complaining less on the eleventh and final day. Since the patient's lab tests and x-rays were free, it was decided to discharge the patient after one week with a clinic appointment. The patient discharge medications were Carbamazepine 200mg tablets twice a day, Levofloxacin 750mg once a day for 7 days, Omeprazole 40mg twice a day and Lanreotide 120mg Injection every 4 weeks.

Discussion

This case represents a septic shock that was complicated by DIC. This case was diagnosed with DIC using the International Society on Thrombosis and Hemostasis (ISTH) diagnostic criteria for sepsis induced DIC, which

depends on four laboratory results. (Shown in table 2.). Since DIC affects small vessels, it commonly presents with organ dysfunction, this explains why this patient presented with AKI [7]. on the other hand Septic ketoacidosis was a newly described pathological condition characterized by elevations in b-hydroxybutyrate and acetoacetate, it can appear in non-diabetic patients as well [8]. It is also known to causes tubular damage and mortality. Both DIC and Ketoacidosis can be used to explain end organ damage in this patient [5].

A scoring system called CRUSADE (Can Rapid Risk Stratification of Unstable Angina Patients Suppress Adverse Outcomes with Early Implementation of the ACC/AHA Guidelines) used to evaluate the risk of bleeding in-patient with ACS. This system uses personal characteristic of patient (gender, diabetes and peripheral vascular disease), admission vitals and evaluation of (heart rate, systolic blood pressure, and signs of congestive heart failure) and labs values (hematocrit and calculated creatinine clearance). It does not define sepsis as risk factor for bleeding. However, two case reports for patients presented with both ACS and sepsis had developed gastrointestinal bleeding suggested that, sepsis is an important risk factor of gastrointestinal bleeding. Like our patient who has a history of ACS and presented with sepsis complicated by upper GI bleeding [5].

Conclusion

Sepsis is a significant cause of mortality and morbidity as well as a significant financial burden on the healthcare system. The current definition makes it simple to identify individuals who are affected, but because so many different types of patients are covered, it may have made it more difficult to create efficient treatments and diagnose diseases more accurately. Greater disease description based on biological pathways is beneficial for prognostic and therapeutic purposes, as other medical specialties, such as oncology, have discovered. A complicated biological cascade that includes sepsis and its consequences can be categorized according to certain clinical traits. The clinical diversity in these occurrences shows that the best ways to describe patient status, forecast clinical course, and direct treatment are through the use of definitions based on clinical end points in conjunction with assessments of illness severity.

Declarations

Ethics approval and consent to participate

The article describes a case report. Therefore, no additional permission from our Ethics Committee was required.

Consent for publication

The consent for publication was obtained from the patient.

Availability of data and material

All data generated or analyzed during this study are included in this published article.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

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Table 1. Laboratory values in Emergency department

Lab
RBS
Na
K
Urea
Creatinine
Cl
Calcium
Phosphorus
Magnesium
D- dimer
FDP
CBC

RBS: random blood sugar; Na: natrium; K: kalium; Cl: Chlorine; FDP: Fibrin degradation products; CBC: Complete blood

Table.2 Sepsis- Induced Coagulopathy and International society on thrombosis and Haemostasis Overt-DIC Scoring System[5].

	Platelet	SIC	Overt DIC
Platelet count (×10ˆ9)	2	< 100	<50
	1	>/= 100, <150	>/= 50, >100
FDP or D-dimer	3	-	Strong increase
	2	-	Moderate increase
	1	-	-
Prothrombin time–INR	2	>1.4	>/=6 s
	1	>1.2, </=1.4	>/= 3, <6 s
	1	-	<1
Fibrinogen (g/l) -Total SOFA score	>/= 2	2	-
	1	1	-

SIC: sepsis-induced coagulopathy; DIC: Disseminated intravascular coagulation; FDP: Fibrin degradation products; INR: international normalised ratio; Total SOFA score: sequential organ failure assessment score.