Sequential applications of Cytosorb and Plasma Exchange Therapy in septic shock: a case report

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Introduction

Severe septic shock is often associated with altered coagulation, frequently leading to disseminated intravascular coagulation, and with multiorgan failure. Several extracorporeal blood purification techniques have been suggested to manage the sepsis-induced organ dysfunction[1,2].

Here we present a patient with severe septic shock, who developed refractory coagulopathy and was treated with sequential extracorporeal techniques: CytoSorb with the aim of reducing the inflammatory cytokines storm[1] and Therapeutic plasma exchange (TPE) to reduce circulating harmful mediators and to increase depleted circulating factors during a severe plasma-refractory coagulopathy and sepsis-related liver disfunction[2].

Case Presentation

A 62 - year - old woman with a history of breast cancer undergoing neoadjuvant chemotherapy, presented to Papa Giovanni XXIII Hospital with fever, cough and back pain. In the emergency department (ED) tachypnoea, hypoxaemia, tachycardia, haemodynamic instability, lactic acidosis and oliguria were observed. Laboratory tests showed a severe neutropenia, coagulopathy and acute renal failure. The SAPS II score was 36. A chest CT scan ruled out pulmonary embolism and acute aortic dissection, but showed right lobar pneumonia, leading to the diagnosis of septic shock from pulmonary origin. Blood samples were taken for cultural investigations, then broad spectrum empirical antibiotic therapy with piperacillin-tazobactam started. In addiction, organ support manoeuvres including orotracheal intubation, mechanical ventilation, high volume crystalloid resuscitation and vasoactive support with norepinephrine up to 0,4 mcg/kg/min, were performed .

Then, patient was admitted in ICU, where hydrocortisone and epinephrine up to 0,1 mcg/kg/min were added according to PiCCO monitoring and echocardiographic evaluation. Empiric antibiotic therapy was further escalated with meropenem and vancomycin due to clinical severity.

On day-1 severe hypoxemic respiratory failure, hyperdynamic circulatory state with low vascular systemic resistances (despite high infused dose of norepinephrine [up to 0,4 mcg/kg/min] and epinephrine [up to 0,1 mcg/kg/min]), metabolic acidosis (pH 7,2) mainly lactic (12,3 mmol/L), anuria and increase of inflammatory markers: white blood cell count (500 /mm3), CRP (20 mg/l), PCT (76 ng/ml), IL-6 (22 ng/ml) and PSP (> 600) occurred. Therefore, treatment with CVVHDF plus Cytosorb filter was started for 48 hours. After two Cytosorb cycles, improvement in metabolic acidosis, circulatory shock (Figure 1, Panel b) and reduction in inflammatory markers (Figure 1, Panel a) were observed. On day-3, due to a new severe plasma-refractory coagulopathy and sepsis-related liver dysfunction, two cycles of Therapeutic Plasma Exchange (TPE) were performed. This resulted in a rapid recovery of hepatic (Figure 1, Panel d) and coagulation (Figure 1, Panel c) parameters[2]. On day-6, a worsening in inflammatory markers and vasoactive demand was observed. Therefore, two more cycles of Cytosorb cycles were administered with subsequent reduction in inflammatory markers and vasoactive drugs requirements. Pseudomonas aeruginosa was identified in the respiratory culture; however, antibiotic therapy was not de -escalated due to the severity of patient's condition.

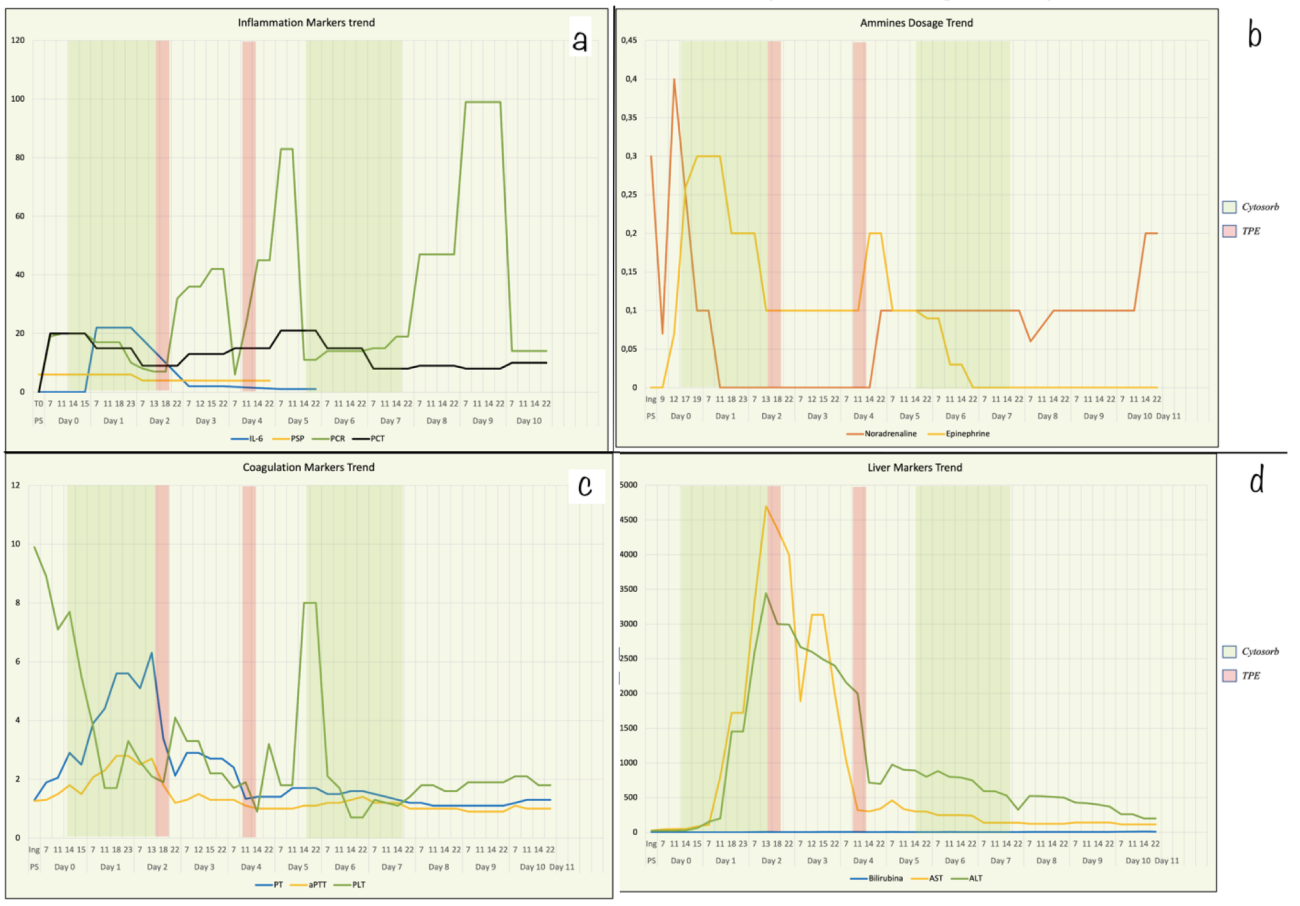

Figure 1: *inflammatory markers(a), vasoactive drugs (b), coagulation (c) and liver markers (d) during ICU stay*

As a consequence of DIC, the patient developed necrosis of the extremities that would have required amputation of some fingers and toes. As a result of respiratory failure, the patient was colonized with Pseudomonas aeruginosa and, on day 18, the patient developed a bilateral hypertensive pneumothorax requiring the placement of two chest drains and resulting in a bronchopleural fistula with abundant air leakage. As a consequence of liver failure, the patient developed chronic liver failure with spontaneous coagulopathy and piastrinopenia.

On day 21, the patient died during a new episode of septic shock due to a new VAP episode.

Conclusion

To our knowledge, this is the first case of sequential use of Cytosorb and TPE in sepsis-induced multi- organ failure during a severe septic shock.

Since the first cycle of Cyotosorb cycle a reduction in inflammatory biomarkers and vasoactive drugs has been observed, demonstrating its potential benefit in interrupting the cytokines storm and reducing the inflammation-related organs injury. TPE was also helpful in correcting sepsis-related liver dysfunction.

Bibliography

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