CASE REPORT

Trauma-induced capillary leak syndrome after penetrating chest injury: Manifestation of massive ascites and pulmonary secretions aggravated by transfusion

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ABSTRACT

Trauma with prolonged shock can cause systemic capillary leak syndrome regardless of the site of injury and a transfusion can aggravate it. The systemic capillary leak induces both an abdominal compartment syndrome and pulmonary edema, and a transfusion can aggravate these sequelae within hours. In our case, 21-year-old man with a penetrating injury in his left thorax experienced delay in rescue and definitive surgery. To manage life-threatening shock, massive blood transfusion and crystalloids had been infused. Cardiopulmonary cerebral resuscitations were performed 2 times during the surgery. Massive amount of pulmonary secretions emitted from his airways with severe hypoxia along with development of massive ascites causing abdominal compartment syndrome, while the surgery was underway. After temporary abdominal closure, he was moved to the intensive care unit and underwent venovenous extracorporeal membranous oxygenation. He recovered without any notable complications. It is important to prevent and correct the shock rapidly by appropriate rescue, controlling the source and infusing less amount of crystalloid and transfusion.

Keywords: Abdominal compartment syndrome; capillary leak syndrome; injuries; pulmonary edema; stab.

INTRODUCTION

Systemic capillary leak syndrome (CLS) occurs after trauma, and it is called trauma-induced CLS (TICS). TICS can be caused by not only multiple severe trauma but also localized trauma.[1] The high mortality rate of TICS is thought to be related to non-specific clinical aspects and its rapid progression to multiple organ dysfunction.[2,3] During this process, abdominal compartment syndrome (ACS) and pulmonary edema can progress with fatal results, especially in case of pulmonary edema, because transfusion related acute lung injury (TRALI) which is a rare complication of transfusion can aggravate it with pulmonary capillary leak.[4–6]

We report a rare case with a penetrating injury limited to the left chest in whom severe pulmonary edema and ACS occurred simultaneously due to prolonged shock caused by a delay in rescue.

CASE REPORT

Emergency medical service received a call from a 21-year-old man who had been stabbed three times in the left anterior chest (Fig. 1). It took 3 h to search and transfer him to the first hospital. The initial blood pressure (BP) at the first emergency department (ED) was 80/40 mmHg, heart rate (HR) was 160/min, respiratory rate (RR) was 30/min, and peripheral arterial saturation (SaO_2) was 85%. Arterial pH was 7.225, base excess was –11.3 mmol/L, and lactate was 12.5 mmol/L. A tension pneumothorax on the left side

Cite this article as: Youn SH, Shin YC, Yoon J, Baek S, Kim Y. Trauma-induced capillary leak syndrome after penetrating chest injury: Manifestation of massive ascites and pulmonary secretions aggravated by transfusion. Ulus Travma Acil Cerrahi Derg 2022;28:863-866.

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Ulus Travma Acil Cerrahi Derg 2022;28(6):863-866 DOI: 10.14744/tjtes.2020.46026 Submitted: 19.06.2020 Accepted: 30.08.2020

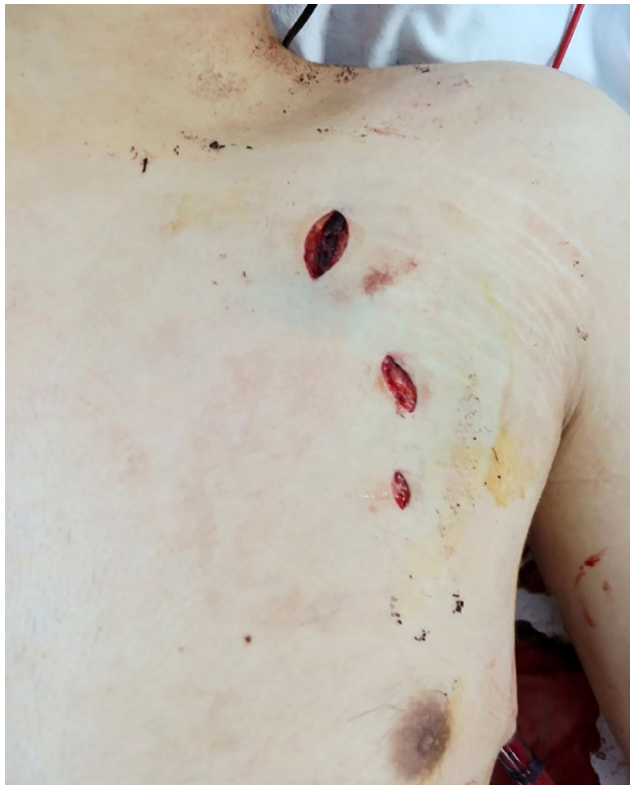

Figure 1. Three stab wounds on the left anterior chest.

was diagnosed. Therefore, a closed thoracostomy was performed with 500 mL of blood drainage for the next 3 h. The patient was transferred 6 and a ½ h after the injury and arrived at our hospital with 67/31 mmHg of BP, 143/min of HR, 24/min of RR, 98% of SaO_2, and 8 points on the Glasgow Coma Scale. Fluid resuscitation and orotracheal intubation were performed in the ED. On squeezing, his dry chest tube, 2000 mL of blood gushed out, leading to a diagnosis of hypovolemic shock and massive hemothorax. Thoracotomy was started 7 and a ½ h after the injury with the right lateral positioning, followed by pulmonary wedge resection of the left upper lobe. Shortly after the start of the surgery, SaO_2 decreased and was maintained around 80% with fraction of inspired oxygen (FiO_2) of 1.0 during the operation. About 15 min after the start of the surgery, a large amount of serosanguinous fluid gushed out through the endotracheal tube. On performing endotracheal suction, secretions were constantly sucked out and on pausing the suction, the ventilator circuit was obstructed every 10 s due to the secretions, seriously interfering with proper ventilation. Around the end of the surgery, a cardiac arrest occurred and cardiopulmonary cerebral resuscitation (CPCR) was performed for 5 min.

After the thoracotomy, a tense abdomen was detected when the position of the patient was changed to supine. Therefore, immediate laparotomy was performed revealing moderate bowel edema, severe retroperitoneal edema, and a large amount of clear fluid collection in the abdominal cavity. Open abdomen procedure was performed to release the intra-abdominal pressure and then temporary abdomen closure was

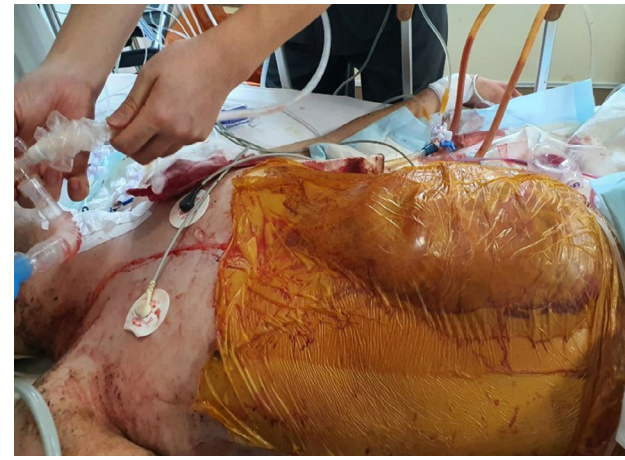

Figure 2. Temporarily closed abdomen. The image shows bowel edema and constantly overflowing ascites and pulmonary secretion.

done (Fig. 2). During the laparotomy, SaO_2 was maintained around 50% with 1.0 of FiO_2 and another cardiac arrest occurred. During the total operation time of 1 h and 45 min, total CPCR time was 15 min.

From the time of the injury to before the surgery, about 4300 mL of crystalloid, 11 units (about 2500 mL) of packed red blood cell (pRBC) and 3 units (about 450 mL) of fresh frozen plasma (FFP) were infused. In addition, about 15,000 mL of crystalloid, 4500 mL of hydroxyethyl starch, 1550 mL of pRBC, and 680 mL of FFP were infused during the surgery.

Laboratory examination results immediately after transfer to the intensive care unit (ICU) demonstrated 6.901 of arterial pH, 63.1% of SaO_2, −17.8 mmol/L of base excess, protein <1.0 g/dL, albumin <1.0 g/dL, and 14.0 mmol/L of lactate. Chest X-ray revealed diffuse pulmonary haziness especially on the right side (Fig. 3). Venovenous extracorporeal membranous oxygenation (ECMO) was started 30 min after transfer to the ICU. Abdominal closure was performed in the ICU on the next day. The patient continued to improve and ECMO was removed on the 3rd post-operative day, and he was transferred to the general ward on the 8th day. He was discharged from the hospital 27 days after surgery. No sequelae were observed at discharge including neurologic problems.

DISCUSSION

In 1960, Clarkson et al.[7] reported CLS as a rare condition and its fatality. Subsequently, several studies were published on the relationship between trauma, shock, and vascular leakage, and their mechanisms.[8] The 131th Annual Meeting of the American Surgical Association announced that 21% of severe trauma patients admitted to the ICU can have TICS.[9]

The pathophysiology of TICS is considered to include alterations in vascular permeability, hydrostatic pressure, and osmotic pressure. These develop due to inflammation, aggressive crystalloid administration, catabolism, direct tissue

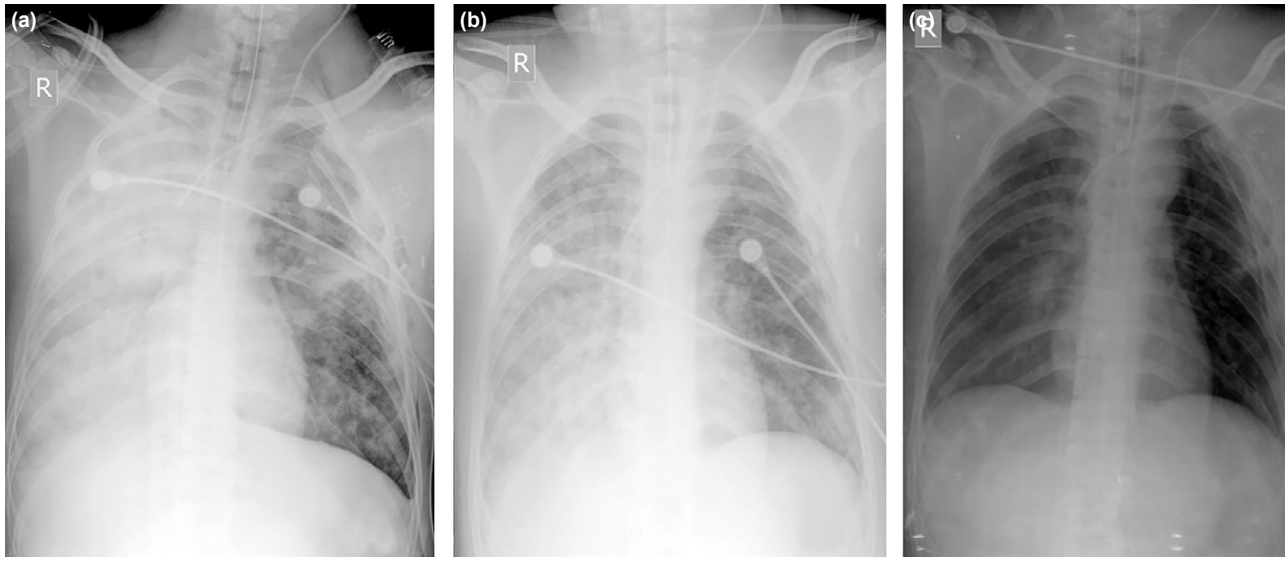

Figure 3. Serial X-ray findings showing gradual improvement after the operation. **(a)** Operation day, **(b)** Post-operative day 1, and **(c)** Post-operative day 2.

injury, and postcapillary hypertension.[10] Vascular leak causes edema in vital organs including the brain, heart, liver, lung, and kidney. In the respiratory system, vascular leak induces alveolar edema that can cause V-Q mismatch and life-threatening hypoxia. Similarly, in the abdomen, it aggravates retroperitoneal edema and ascites that raise intra-abdominal pressure which is the major cause of death in ACS.[1,8,11] In our case, the intravenously infused crystalloid volume (approximately 4500 mL before surgery) was not enough to cause fatal pulmonary edema and ACS. Studies have shown that the administration of more than 5000 mL of crystalloid within 24 h is one of the independent risk factors for secondary ACS, and more than 7000 mL of crystalloid increases the risk of fatal pulmonary edema.[12–15] Therefore, prolonged shock until definitive surgery due to delayed rescue and transfer could be an additional cause of TICS.

Nevertheless, the massive pulmonary edema could also be explained by the amount of pre-operative transfusions including 11 units of pRBCs and 3 units of FFPs since the risk of TRALI reportedly increases with the number of units of blood transfused regardless of its components. Moreover, a report has indicated that multiple transfusions (pRBCs >10 units) within 24 h is also one of the independent risk factors for secondary ACS and transfusion needs to be considered as another factor which might cause TRALI and secondary ACS.[12]

The term "TRALI" was first used by the Mayo Clinic in 1985 in a report of a case series by Drs. Popovsky and Moore.[16] It is clinically defined as a new acute lung injury (ALI) with the onset of symptoms or signs within 6 h of a transfusion. TRALI is usually temporary, and supportive care mostly leads to a recovery of the SaO_2 to the level before the transfusion within 48–96 h.[17] The related clinical aspects were reported by Teodori et al.[4] in a case report of TRALI with massive pulmonary secretions similar to the respiratory manifestations in our case.

Our case report describes a patient who was stabbed three times in an area limited to the left chest, which might not have been life-threatening if he had received prompt definitive treatment. However, he experienced shock due to delayed rescue and transfer. He had obstructive shock from tension pneumothorax until the first closed thoracostomy. Sustained hypovolemic shock produced a condition that easily progressed to TICS and lack of management of hemorrhage also required multiple transfusions, one of the risk factors of TRALI. The patient had massive secretions coming out constantly through his airway and into the abdominal cavity as well. The massive pulmonary secretions further damaged his uninjured lung which was in a dependent position during the surgery and worsened his hypoxia which together with shock induced the cardiac arrest. At the same time, massive ascites increased the intra-abdominal pressure and damaged the related organs.

As the duration of the shock lengthened, the following TICS mechanisms were possibly involved. First, sustained catabolism induced hypoalbuminemia that altered the oncotic pressure. Second, infusion of crystalloids for correcting shock promoted changes in hydrostatic and oncotic pressure. Third, a cytokine surge caused by severe inflammation increased vascular permeability. In addition, problems in the pulmonary microcirculation developed due to multiple transfusions.[6,10] The immediate open abdomen strategy for ACS which was performed in the operating room and rapid post-operative ECMO for respiratory failure supported the life of the patient for 72 h, and he was eventually able to recover.

Studies have reported mortality of 38~67% in secondary ACS, in-hospital mortality of 7.4~17% in acute pulmonary edema, and 5~10% in TRALI.[14,18,19]

Conclusion

Since progressive shock is one of the major causes of cytokine release, it is important to correct the shock rapidly by controlling the source and infusing less amount of crystalloid. This can also give the chance to reduce transfusions. Prompt rescue and appropriate field triage may reduce the associated morbidity and mortality by preventing shock.

Informed Consent: This case was approved by the Institutional Review Board of National Medical Center (NMC-2006-002), which waived the need for informed consent from the patient.

Peer-review: Internally peer-reviewed.

Authorship Contributions: Concept: S.H.Y., Y.C.S., J.Y., S.B., Y.K.; Design: S.H.Y., S.B.; Supervision: S.H.Y., Y.K.; Literature search: S.H.Y.; Writing: S.H.Y., S.B.; Critical revision: Y.K.

Conflict of Interest: None declared.

Financial Disclosure: The authors declared that this study has received no financial support.

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OLGU SUNUMU - *ÖZ*

Penetran göğüs yaralanması sonrası travmaya bağlı kapiller kaçış sendromu: Transfüzyonla şiddetlenen masif asit ve pulmoner sekresyon

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Uzamış şokun eşlik ettiği travma, yaralanma bölgesinden bağımsız olarak sistemik kapiller kaçış sendromuna neden olabilir ve herhangi bir transfüzyon bu durumu ağırlaştırabilir. Sistemik kapiller kaçış hem abdominal kompartman sendromunu hem de pulmoner ödemi indükler ve herhangi bir transfüzyon, bu sekelleri saatler içinde ağırlaştırabilir. Bizim olgumuz olan, sol göğüs kafesinde penetran yaralanması olan 21 yaşındaki erkek hasta, gecikmiş kurtarma cerrahisi ve kesin cerrahi geçirdi. Hayatı tehdit eden şok durumunu yönetmek üzere, yoğun kan transfüzyonu ve kristaloid infüzyonu yapıldı. Ameliyat sırasında iki kez kardiyopulmoner serebral resüsitasyon uygulandı. Ameliyat boyunca, abdominal kompartman sendromuna neden olan masif asit gelişimi ile birlikte, şiddetli hipoksinin eşlik ettiği hava yollarından masif miktarda pulmoner sekresyon çıkarıldı. Abdomen geçici olarak kapatıldıktan sonra hasta yoğun bakım ünitesine alındı ve veno-venöz ekstrakorporeal membran oksijenasyonu uygulandı. Hasta, önemli bir komplikasyon kalmadan iyileşti. Uygun kurtarma prosedürü, kaynağın kontrol edilmesi ve daha az miktarda kristalloid ve transfüzyon infüzyonu ile şokun hızla önlenmesi ve düzeltilmesi önemlidir.

Anahtar sözcükler: Abdominal kompartman sendromu; bıçaklanma; kapiller kaçak sendromu; pulmoner ödem; yaralanmalar.

Ulus Travma Acil Cerrahi Derg 2022;28(6):863-866 doi: 10.14744/tjtes.2020.46026