Multiple Organ Failure Following Isolated Spleen Laceration: A Rare Case and Management Strategies

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Aim: We aim to analyze and discuss management strategies the metabolic complications encountered in isolated spleen laceration following high-energy trauma and the resulting Multiple Organ Dysfunction Syndrome (MODS).

Case Presentation: A 16-year-old male patient presented to the emergency department following a high-energy traffic accident. Glasgow Coma Scale (GCS) was 15, hemodynamic status was stable, with hematocrit (Hct) at 45.8%, platelet count (PLT) at 302,000/μL, and electrolyte balance was normal. ALT was 27 U/L and AST was 44 U/L. Creatinine (Cr) was 0.9 mg/dL and BUN was 18 mg/dL. Whole-body CT revealed isolated spleen laceration classified as WSES grade 3 and AAST grade 4.

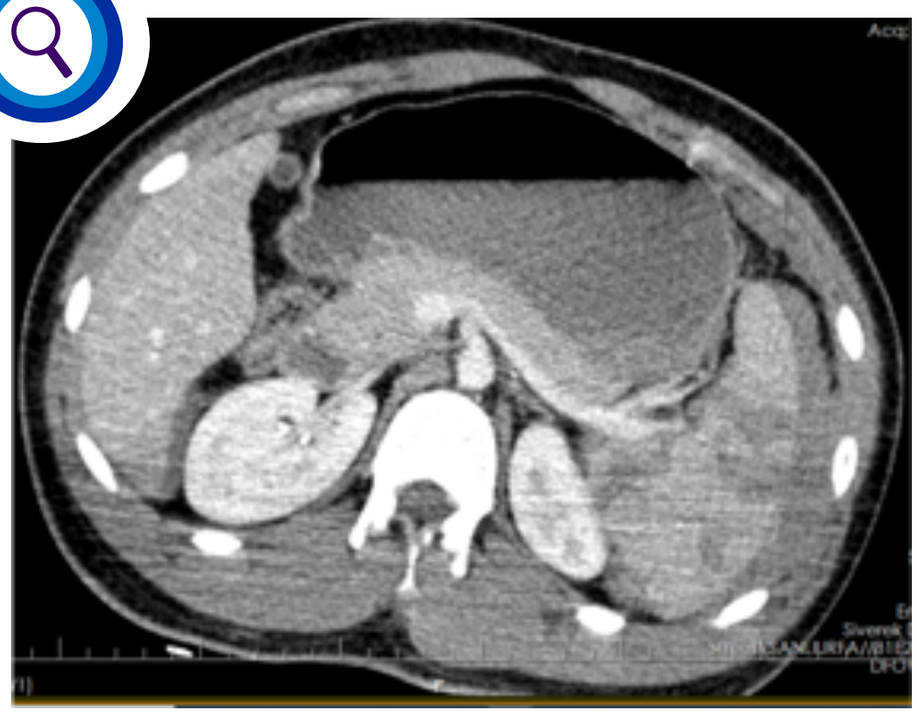


Image 1: *Middle pole laceration and intraperitoneal haemorrhage leakage area behind the splenic vein*

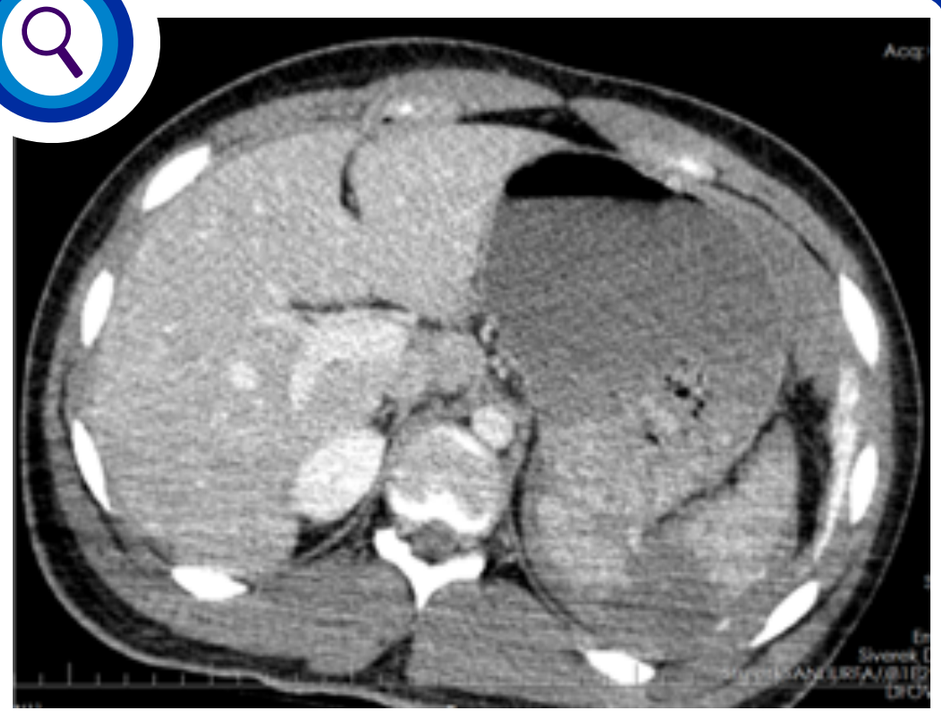
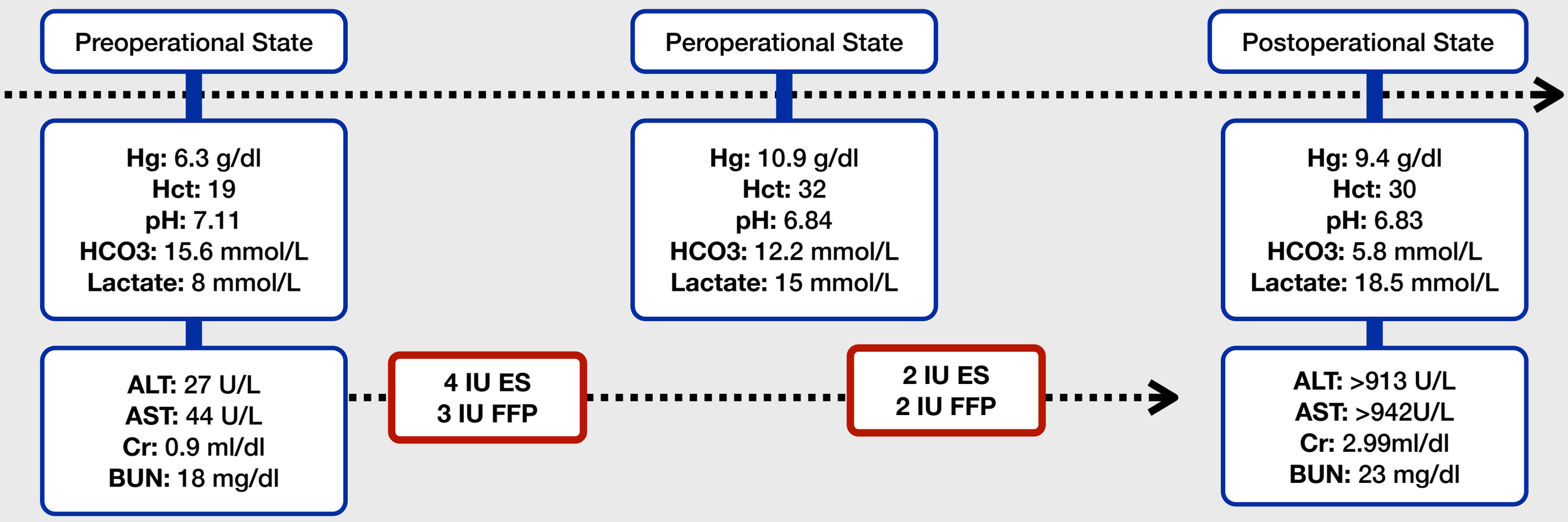


Image 2: *Upper pole laceration and subcapsullar hematoma*



- ☑ A proper metabolic state couldn't be achieved after one day of surgery.
- ☑ High liver function tests remained over the maximum level of laboratory limits.
- ☑ Urine output decreased and creatinine continued to elevate. Acidosis couldn't be corrected.
- ☑ Intracranial pressure elevation symptoms, high pulse pressure, moderate bradycardia continued and his consciousness was never improved.
- ☑ The patient was considered as Multi Organ Dysfunction Syndrome (MODS).
- ☑ The patient was considered to dialysis but a sudden cardiac activity was stoped.
- ☑ The patient did' not respond to resuscitation in ICU and his death was declared.

Acute Kidney Injury, Fulminant Liver Failure Without Hepatic Parenchymal Disruption and MODS

- ☑ It is rarely seen high elevation of AST and ALT levels in early times of trauma that leading to fulminant liver failure(FLF) without any hepatic parenchymal disruption.
- ☑ The reason could be a ischemia/reperfusion injury of the liver parenchyma and acceleration/deceleration of the liver causes parenchymal cell swelling also contributed with high catecholamine release attributed cell injury.
- ☑ Fulminant liver failure attributed high lactate levels therefor with the renal impairment metabolic lactic acidosis had been worsened.
- ☑ High sympathetic activity and catecholamine release may causes severe cardiac distribution.
- ☑ Mortality might be seen according to myocardial destruction attributing low cardiac output, low BP, seve arrhythmias.
- ☑ Ischemia/Reperfusion injury and high sympathetic activity also take role in hepatic failure and AKI.
- ☑ Despite adequate fluid resuscitation and cardiac inotropic support there may occur an unbreakable vicious circle.

CONCLUSION

- ☑ Early hemodialysis for AKI and plasmapheresis could be considered to impair inflamation and remove excessive cytokines, catecholamines from circulatory system.
- ☑ It is shown that AKI unresponsive to fluid resuscitation in SIRS could be good managed with early hemodialysis and decrease mortality.
- ☑ In order to reduce the metabolic load on the liver and kidney, it is necessary to be as fast as possible in the interventional issues and to avoid intensive blood transfusion.