The Early Diagnosis And Treatment Of Acute Hemorrhagic Shock After Laparoscopic Cholecystectomy
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INTRODUCTION
In the last decade, laparoscopic procedures became popular in surgery. Especially cholecystectomy is widely performed today. Besides some advantages, laparoscopic cholecystectomy has also some disadvantages and complications. In order to avoid these complications they must be well recognized and diagnosed (1).

The most dangerous complication of laparoscopy is big vessel damage and bleeding. The incidence is less than 0.1% but most of the deaths related to laparoscopic cholecystectomy are from bleeding due to vessel damage. Early diagnosis and treatment is essential in reducing mortality so that postoperative period care is very important.

We report a case of a 69 year old man who underwent laparoscopic cholecystectomy.

CASE REPORT
A 69 year old man, in ASA I class, underwent laparoscopic cholecystectomy. He was extubated without any problem and admitted to the recovery room. 20 minutes later, peripheral O$_2$ saturation reduced to 88% and nasal O$_2$ support was started. After his consciousness returned, his agitation increased, became tachipneic and peripheric cyanosis was observed. Although the analgesic dose was increased because of his pain, agitation did not disappear. O$_2$ saturation persisted at about 88-89%. Ventilatory insufficiency was determined by physical examination and the chest radiogram demonstrated right diagram elevation.

Arterial blood gases were as follows: pH : 7.22, pO$_2$: 60.3mmHg, pCO$_2$: 52 mmHg, HCO$_3$: 20.9mmol/L

A central venous catheter was inserted because of the progressive decrease of arterial blood pressure (50/30 mmHg) and increase in pulse rate to 146 beats/min. After verifying a volume deficit with CVP, fluid resuscitation was started after determining hemoglobin and hematocrit levels. The parasynthesis was hemorrhagic and a laparotomy was performed immediately. After four units blood transfusion and appropriate surgery the patient was admitted to intensive care unit.

DISCUSSION
In 1911, Jacobeus first introduced laparoscopy term using peritoneal endoscopy. After the successful reports of laparoscopic cholecystectomy from France and USA in 1987 and 1988 it was widely used. The selective and rational use of laparoscopy is cost effective and leads a decrease in hospitalization duration. It also prevents unnecessary laparotomy and is useful in accurate diagnosis.

Parallel with this development some complications due to laparoscopic surgery were described. Anaesthesiologists must be aware of them and know their appropriate approaches.

Intraoperative CO$_2$ insufflations may causes pain, pulmonary hemodynamic and endocrine problems. Also, acute hemorrhage, bowel and bladder perforation, subcutaneous
Emphysema, pneumothorax and pneumomediastinum can be seen. In the postoperative period, pulmonary failure, delayed bleeding, wound infection, pain, nausea and vomiting may develop.

In spite of the prevention like nasogastric intubation and bladder decompensation during laparoscopy, serious even fatal vascular complications may occur.

In laparoscopic cholecystectomy, due to cistic or hepatic arterial damage bleeding can be seen. Expansion related to pneumoperitoneum may lead vascular rupture and bleeding.

In the postoperative period, hidden bleeding due to vascular damage causes a decrease in hematocrit values, hematoma formation and extreme pain. Uncontrolled bleeding occurs in 1.9% of laparoscopic cholecystectomy procedures.

Transfusion requirement or reexploration rate after hemorrhage was reported to be 0.5%. Diagnosis of big vessel damage can be established by the blood coming out from trocar or Veres needle. In some cases, injury can be restricted in the retroperitoneal area and like in our case shock may appear in the postoperative period. Hemorrhage, hypovolemia, pain, abdominal distension and diaphagmatic elevation results in shock. Shock is characterized by tissue perfusion reduction and insufficient oxygen supply. Clinically, the mental status changes and peripheral pulse debility and oliguria develops. In our case, mental changes and decrease in peripheral $O_2$ saturation were the first findings.

Reduced blood flow is compensated by increased $O_2$ extraction. If $O_2$ extraction can not be increased sufficiently V2O2 becomes dependent to blood flow. In such cases adequate $O_2$ transfer to tissues can be obtained if cardiac output is high enough. In massive hemorrhage cardiac output decreases. Decreased ventricular load and QT depression causes vasoconstriction and a increase in central vascular resistance. In pulmonary thromboembolism, because of PAP and PVR increase, right ventricle output decreases. In cardiogenic shock the primarily problem is pump failure. When peripheral vasoconstriction response develops in order to maintain coronary arterial pressure and MAP, CVP increases.

In our case, electrocardiogram, ETCO$_2$ and $paO_2$ were normal and there was no pulmonary edema and precordial murmur. Therefore, we eliminated pulmonary embolism.

Rapid hemodynamic changes, high and weak pulse, low blood pressure, reduced CVP directed us to hemorrhagic volume loss. We related the delayed hemodynamic changes to the insufflation’s pressure effect to the damaged vessels.

The anesthesiologist must be aware of the hemorrhagic complications in laparoscopic procedures. In the postoperative period mental changes, pulse rate, arterial tension, urinary output and skin color changes must be closely followed.

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