Abnormal serum liver chemistry related to pneumoperitoneum after endoscopic submucosal dissection

A 61-year-old man had been followed after endoscopic submucosal dissection (ESD) for early gastric cancer. In his youth, the patient had undergone total colectomy because of Crohn’s disease. Surveillance esophagogastroduodenoscopy revealed synchronous early gastric cancer, so a second ESD was performed. Although gastric perforation occurred during the procedure, it was closed completely using endoclips (Fig. 1 and 2). Antibiotic therapy was started with fasting. The following day, 12 h after the second ESD, laboratory data included aspartate aminotransferase 225 U/L (normal range 12–32 U/L), alanine aminotransferase 201 U/L (2–38 U/L), lactate dehydrogenase 526 U/L (119–229 U/L), creatine phosphokinase 512 U/L (40–218 U/L), C-reactive protein 3.47 mg/dl (<0.3 mg/dl), and white blood cell count 27.75 × 10⁹/L (3.4–9.4 × 10⁹/L). As electrocardiography and troponin test ruled out ischemic heart disease, an effect of pneumoperitoneum was strongly suspected. An abdominal CT scan showed severe pneumoperitoneum with compression of the inferior vena cava (Fig. 3), and therefore abdominal decompression with a 14-gauge puncture needle was performed. Immediately after the puncture, the abdominal fullness and back pain improved. Four days later, almost all of the laboratory parameters recovered to within the normal ranges, and the patient made an uneventful recovery during the 14 months of follow-up after the procedure.

Gastric perforation is a major complication of endoscopic treatment, but most perforations are small and can be managed endoscopically using endoclips [1, 2]. After endoscopic closure, if severe abdominal fullness persists, needle puncture is performed for decompression of the pneumoperitoneum in order to prevent negative systemic effects. It has been reported that prolonged pneumoperitoneum increases main arterial blood pressure and systemic vascular resistance but decreases stroke volume and cardiac output during laparoscopic surgery with carbon dioxide insufflation [3, 4]. In our patient, abdominal CT scan revealed compression of the inferior vena cava, suggesting high intra-abdominal pressure. In this condition, we believe that the increased main arterial blood pressure and systemic vascular resistance caused congestion of the liver, resulting in elevation of liver enzyme levels [5].

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3 Endoscopic closure was performed using an endoclip.

References

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