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CASE REPORT

Irrigation Fluid Extravasation Presenting as Ventilatory Compromise After Biportal Endoscopic Spine Surgery: A Case Report

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ABSTRACT

During biportal endoscopic spine surgery, irrigation fluid may extravasate and raise intra-abdominal pressure. A 78-year-old woman underwent L4–L5 decompression with left foraminoplasty under general anaesthesia in the prone position; the irrigation pump pressure was set to 30 mmHg, and a total of 42 L of normal saline was recorded. During emergence after supine repositioning, the abdomen was distended and tense, ventilation became difficult, and end-tidal carbon dioxide rose to 50–60 mmHg. Nasogastric and rectal tubes were inserted, and furosemide was administered. Computed tomography showed low-attenuation fluid in perihepatic and perisplenic spaces, retroperitoneum, paracolic gutters, and pelvis. On arrival in the post-anesthesia care unit, 1.3 L was drained from the urinary catheter; urine output remained high on postoperative days 0 and 1. She recovered without percutaneous drainage or laparotomy. This case highlights a reversible early presentation without clinical features of abdominal compartment syndrome and suggests treating unexplained ventilatory difficulty as a warning.

Keywords: Biportal endoscopic spine surgery, irrigation fluid, fluid extravasation, intra-abdominal hypertension, hypercapnia

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INTRODUCTION

Biportal endoscopic spine surgery (BESS) uses separate viewing and working portals, which allow continuous visualisation and two-handed manipulation through small incisions.[1,2] Continuous irrigation is essential, but the posterior spinal working space is not sealed; if inflow exceeds outflow, saline may increase local pressure and track through paraspinal, retroperitoneal or peritoneal tissue planes.[3]

Previous reports of irrigation-related complications have mainly described severe respiratory, neurological or haemodynamic deterioration or established abdominal compartment syndrome (ACS).[4-6] We describe an early, clinically important presentation in which abdominal distension and difficult ventilation became evident during emergence and resolved with conservative, physiology-based management.

CASE REPORT

A 78-year-old woman (161 cm, 67 kg; American Society of Anesthesiologists Physical Status II) with type 2 diabetes mellitus and a 5-month history of buttock and lateral calf radicular pain underwent biportal endoscopic L4–L5 decompression with left foraminoplasty under general anaesthesia in the prone position. Induction of anaesthesia was defined as T0. Surgery lasted from T0+25 to T0+210 min, and anaesthesia ended at T0+255 min. The irrigation pump pressure was set to 30 mmHg, and operative records documented irrigation with 14 bags, each containing 3 L of normal saline (42 L in total).

No major intraoperative haemodynamic instability occurred. During volume-controlled ventilation, peak inspiratory pressure (PIP) increased slightly from 15–18 to 20–23 cm H_2O near the end of surgery. Emergence was unusually prolonged, lasting approximately 45 minutes from the end of surgery to extubation. After repositioning to the supine position, the abdomen was markedly distended and tense; PIP rose to 31–34 cm H_2O, end-tidal carbon dioxide ($EtCO_2$) increased from 35–40 to 50–60 mmHg, and peripheral oxygen saturation (SpO_2) fell to 88–92%. Ventilator settings were adjusted, and manual bag ventilation was performed; nasogastric and rectal tubes were inserted for decompression, and furosemide was administered. With these measures, $EtCO_2$ returned to 40–42 mmHg, and SpO_2 recovered to 98%; the trachea was then extubated, and the patient was transferred to the post-anaesthesia care unit (PACU) (Table 1).

During observation in the PACU from T0+255 to T0+295 min, oxygen administered via a Venturi mask at 6 L/min maintained SpO_2 at 95–99%. At T0+275 min, arterial blood gas analysis showed pH 7.41, $PaCO_2$ 41 mmHg, PaO_2 70 mmHg, and SaO_2 94% on supplemental oxygen, indicating impaired oxygenation; 1.3 L was drained from the urinary catheter on arrival. Abdominal computed tomography (CT) was obtained on postoperative day (POD) 0 shortly after transfer to the intensive care unit (ICU). CT showed low-attenuation fluid in the perihepatic and perisplenic spaces, the retroperitoneum, both paracolic gutters, and the pelvis (Figure 1).

Laboratory findings did not suggest pancreatitis or end-organ injury: serum creatinine, liver enzymes, serum amylase, creatine kinase-MB and troponin I were within their reference ranges, and C-reactive protein was only mildly elevated, consistent with the postoperative state.

She was managed with oxygen therapy, gastrointestinal decompression, fluid-balance monitoring and supportive diuretic therapy.

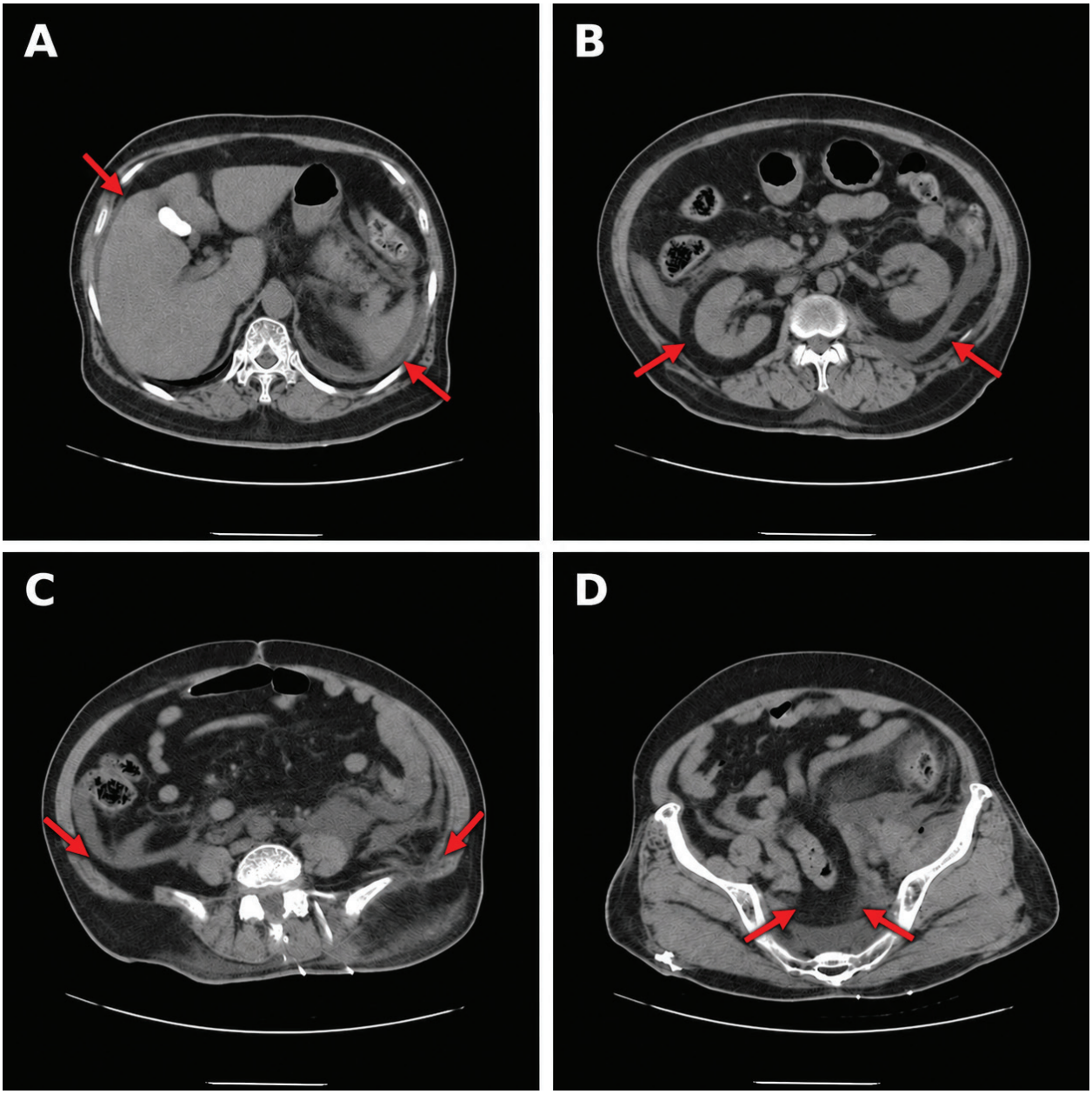


Figure 1: Postoperative axial abdominal computed tomography (CT) images showing low-attenuation fluid in the perihepatic and perisplenic spaces (A), in the retroperitoneum (B), in the paracolic gutters (C), and in the pelvis (D).

Brisk diuresis followed (Table 1), although shift-based recording of fluid intake and output prevented precise dose-response assessment. On POD 1, room-air SpO_2 fell to 87% but improved with nasal oxygen at 2 L/min. Oxygen therapy was tapered, and she was transferred to the ward that night. Subsequent room-air SpO_2 remained 94–97%. Abdominal discomfort resolved without percutaneous drainage or laparotomy.

Table 1: Perioperative timeline

Variable	Event
T0	Induction of general anaesthesia.
T0+25 to T0+210 min	Prone L4–L5 decompression with left foraminoplasty; irrigation pump pressure set to 30 mmHg; documented irrigation, 42 L.
T0+210 to T0+255 min	Emergence after repositioning to the supine position: abdominal distension and tension, PIP 31–34 cm H_2O, $EtCO_2$ 50–60 mmHg, SpO_2 88–92%; ventilator adjustment and manual bag ventilation; nasogastric and rectal tubes inserted for decompression; furosemide 30 mg; $EtCO_2$ returned to 40–42 mmHg and SpO_2 recovered to 98%; trachea extubated.
T0+255 to T0+295 min	PACU: oxygen via a Venturi mask at 6 L/min; SpO_2 95–99%; arterial blood gas analysis showing pH 7.41, $PaCO_2$ 41 mmHg, PaO_2 70 mmHg and SaO_2 94% on supplemental oxygen; 1.3 L drained from the urinary catheter on arrival.
POD 0, after ICU transfer	CT: low-attenuation fluid in the perihepatic and perisplenic spaces, retroperitoneum, both paracolic gutters and pelvis.
POD 0 evening	Furosemide 10 mg; cumulative urine output, 4.33 L for POD 0.
POD 1	Furosemide 20 mg; urine output, 4.55 L; transient room-air SpO_2 87%; transfer from ICU to ward.
Later	Room-air SpO_2 94–97%; no percutaneous drainage or laparotomy.

PIP, peak inspiratory pressure; POD, postoperative day; SaO_2, arterial oxygen saturation; SpO_2, peripheral oxygen saturation; T0, induction of anaesthesia. Furosemide cumulative dose: 60 mg (30 mg at emergence, 10 mg on POD 0, 20 mg on POD 1).

DISCUSSION

Because intra-abdominal pressure (IAP) was not measured, this case should be framed as suspected early or transient intra-abdominal hypertension (IAH) caused by irrigation fluid extravasation rather than as confirmed ACS.[7,8] Unlike a previously reported case requiring invasive decompression[5], our patient had ventilatory difficulty and impaired oxygenation without shock, oliguria, renal dysfunction, pancreatitis, or progressive symptoms.

Irrigation during BESS requires balanced inflow and outflow. When outflow is impaired, or surgery is prolonged, saline may track through paraspinal and retroperitoneal planes and distribute into intraperitoneal compartments.[3] Irrigation fluid extravasation during hip arthroscopy has produced fluid in both the retroperitoneum and the peritoneal cavity on CT,[9] consistent with the supramesocolic (perihepatic and perisplenic), paracolic and pelvic distribution observed in our patient. Prone positioning can mask abdominal distension; after repositioning to the supine position, accumulated fluid may reduce thoracoabdominal compliance, elevate the diaphragm, increase PIP, raise $EtCO_2$ or increase the need for supplemental oxygen. The mild late increase in PIP in this case may therefore have been an early warning sign.

Alternative diagnoses were less likely. Normal amylase and widespread low-attenuation fluid favoured irrigation fluid extravasation over pancreatitis, whereas venous congestion typically causes tissue oedema rather than discrete abdominopelvic collections. The absence of haemodynamic instability, renal disturbance and persistent abdominal pain argued against major bleeding, urinary tract injury or bowel obstruction.

Management should be stepwise: communicate with the surgeon, reduce or stop irrigation, confirm outflow and reassess once supine. Stable patients may be managed with oxygen, gastrointestinal decompression and fluid-balance monitoring. Correcting a positive fluid balance with diuretics is a recognized medical measure to lower IAP,[7] and in our patient furosemide was used cautiously once she was hemodynamically stable to mobilize reabsorbed fluid; its specific role after irrigation fluid extravasation is not established, and it should be reserved for patients without hypovolaemia. Persistent respiratory compromise, hypotension, oliguria or abdominal tension should prompt IAP measurement and consideration of image-guided percutaneous drainage; laparotomy is reserved for refractory deterioration.[7,8]

Limitations include the absence of IAP measurement, retrospective reconstruction of irrigation volume and urine output, CT obtained after the acute event rather than at maximal distension, and the inferential nature of the proposed mechanism. Nevertheless, this case broadens the spectrum of irrigation-related complications to include an early presentation that was reversible with conservative management.

CONCLUSION

After BESS, sudden abdominal distension with difficult ventilation during emergence should raise suspicion of irrigation fluid extravasation and possible evolving IAH. Early recognition and physiology-based stepwise management may prevent progression to ACS while avoiding unnecessary invasive treatment in stable patients.

AUTHORS' CONTRIBUTIONS

Concept and design: HGP, WJ, YHC

Literature review: HGP

Compilation of manuscript: HGP

Manuscript editing & proofreading: WJ, YHC

DECLARATION OF CONFLICTS OF INTEREST

The authors declare no potential conflicts of interest relevant to this article.

DECLARATION OF PATIENT'S CONSENT

Written informed consent was obtained from the patient and her family for publication of this case.

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