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Small Bowel Perforation and Subsequent Pleural Effusion Following Operative Hysteroscopy: A Case Report

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Abstract

A 59-year-old woman developed severe and persistent abdominal pain following a hysteroscopy, which was performed the previous day. Initial differential diagnoses included postoperative infection, ovarian pathology, and uterine perforation. The patient remained haemodynamically stable despite severe abdominal tenderness and signs of peritonism. CT imaging of the abdomen and pelvis demonstrated a pneumoperitoneum with marked small-bowel abnormalities and mesenteric inflammatory change. This finding prompted recognition of small-bowel perforation and the need for emergency surgical intervention. Laparoscopy was converted to laparotomy, where a 7 mm small-bowel perforation was identified and primarily repaired.

The postoperative course was complicated by a significant right-sided pleural effusion. Thoracic ultrasound demonstrated marked septations, raising concern for possible empyema. Pleural fluid analysis was non-diagnostic, with a pH of 7.31 and glucose of 4.6 mmol/L, and microbiological analysis identified no organisms. The effusion was ultimately considered clinically to represent a parapneumonic effusion, although a reactive postoperative effusion related to the recent intra-abdominal pathology remained a consideration. The patient improved clinically with antibiotics and conservative management and was discharged with appropriate respiratory follow-up.

This case highlights the need to consider intra-abdominal visceral injury in patients presenting with severe or persistent abdominal pain and peritonism following operative hysteroscopy, even in the absence of early haemodynamic instability. It also illustrates the diagnostic complexity that can arise when a pleural effusion develops following intra-abdominal pathology, as reactive and infective causes may have overlapping clinical and radiological features.

Categories: Obstetrics/Gynecology, General Surgery
Keywords: bowel perforation, loculated pleural effusion, management of bowel perforation, operative hysteroscopy, uterine perforation

Introduction

Hysteroscopy is a commonly performed procedure used in the diagnosis and treatment of gynaecological intrauterine pathology. It is generally considered safe, with a low overall incidence of complications [1]. The risks are higher with operative hysteroscopy compared to diagnostic hysteroscopy, with one large multicentre study reporting complication rates of 0.95% and 0.13%, respectively [1]. Uterine perforation is the most common complication recognised from hysteroscopic surgery, with a possible risk of injury to surrounding structures such as the bowel [2,3]. Bowel injury is an uncommon but serious complication, often as a consequence of delayed recognition [4]. Bowel injury may result from direct mechanical trauma associated with uterine perforation, allowing hysteroscopic instruments to damage adjacent structures. Thermal injury may also occur during operative procedures involving electrosurgical instruments [5,6]. The incidence of bowel injury has been investigated more extensively in gynaecological laparoscopy than in hysteroscopy. A systematic review of 474,063 gynaecological laparoscopies reported bowel injury in 0.13% of procedures, with delayed diagnosis occurring in 41% of reported injuries [4]. Although these numerical findings cannot be directly linked to hysteroscopy, this evidence highlights the importance of prompt recognition of bowel injury in other gynaecological procedures. It is worth noting that intra-abdominal pathology can also result in reactive or complicated pleural effusions through diaphragmatic inflammation or transdiaphragmatic fluid translocation [7].

This case describes the recognition of a small-bowel perforation as a complication of operative hysteroscopy performed in a 59-year-old female, which later required conversion from the initial laparoscopic procedure to laparotomy for primary repair. The development of a subsequent right-sided pleural effusion further complicated the postoperative course. This highlights the potential for significant gastrointestinal and, unexpectedly, extra-abdominal complications to arise following hysteroscopic surgery in the absence of haemodynamic compromise. Subsequently, it demonstrates the challenges involved in their respective

diagnosis and management.

Case Presentation

A 59-year-old female presented to the emergency department with severe, constant lower abdominal pain, predominantly pelvic and more pronounced on the left side. Her symptoms had developed after the analgesic effect had subsided following a hysteroscopy performed the previous day at another hospital. Following the onset of pain, her symptoms progressively worsened, with the pain radiating to her back and upwards into the chest towards the sternum. This was accompanied by nausea and approximately seven episodes of vomiting. A temperature of 38.1°C was recorded, with associated chills and rigors. Oral intake had not been tolerated since the procedure, with her last meal consumed before the hysteroscopy. There was no history of chest pain, leg swelling or pain, palpitations, urinary symptoms, or any change in bowel habit.

Prior to the hysteroscopy, the patient was fit and well with no recent infections. She was independent in her activities of daily living, a non-smoker, and reported occasional alcohol consumption. The hysteroscopy had been performed under the two-week-wait cancer pathway. Initially, the patient presented to her general practitioner (GP) due to an episode of post-menopausal bleeding, which had occurred two months previously. A transvaginal ultrasound scan showed 6 mm endometrial thickening, which warranted further investigation. The patient reported that a large endometrial polyp had been identified during the hysteroscopy and that the majority of it had been removed. Additionally, she had been prescribed a seven-day course of rivaroxaban because of a family history of deep vein thrombosis. The patient had commenced rivaroxaban six hours after the hysteroscopy, with her last dose taken that evening before presentation to the emergency department. However, no clinical letter or documentation was provided by the patient to confirm the procedural findings.

Previous gynaecological history included a hysteroscopy approximately 12 years previously, which was uncomplicated and demonstrated endometriosis involving the right ovary and benign cysts affecting both ovaries. There was also a history of nine fibroids, for which the patient had previously undergone uterine fibroid embolisation around the same time. She currently had a large calcified fibroid, reportedly approximately the size of a hockey ball. Menopause occurred at the age of 54, with a preceding history of heavy and painful periods. Obstetric history included three miscarriages of unknown cause and one vaginal delivery at around 40 weeks' gestation, 25 years previously.

Past medical history included hypotension and diverticular disease. Previous surgical history included a laparoscopic Burch procedure, surgery for a prolapsed vaginal wall two years previously, and breast implant insertion approximately 30 years previously. Lastly, no known allergies had been reported or documented.

On initial examination, the patient was tachycardic, with a heart rate of 116 beats per minute, but remained haemodynamically stable with a blood pressure of 134/75 mmHg. She was pyrexial, with a temperature of approximately 38°C, and had no oxygen requirement. Abdominal examination demonstrated severe tenderness in both iliac fossae, with generalised abdominal guarding and signs of peritonism. Mild epigastric tenderness was also present without rebound tenderness. Bowel sounds were present.

Given the recent hysteroscopy, together with the presence of pyrexia and pelvic pain, the patient was initially discussed with the gynaecology team for assessment. Initial differential diagnoses included a gynaecological infection, such as endometritis, and uterine perforation following the recent procedure. Speculum and pelvic examinations were performed. The cervix was closed, with no adnexal masses or tenderness identified, and only a small amount of serous vaginal discharge was noted. Bimanual examination was unremarkable, with no adnexal tenderness or cervical motion tenderness.

Initial management included intravenous fluids and empirical intravenous cefuroxime and metronidazole, in accordance with local guidelines, given the initial suspicion of a gynaecological or intra-abdominal infective source. Adequate analgesia was provided, and the patient was kept nil by mouth while awaiting further investigations.

Initial blood investigations demonstrated a raised white cell count of 14.3 × 10^9/L, a neutrophil count of 13.2 × 10^9/L, and a C-reactive protein (CRP) of 117 mg/L. Haemoglobin was stable at 133 g/L, and venous lactate was 1.3 mmol/L. The coagulation screen demonstrated a raised prothrombin time of 18.5 seconds and a PT ratio of 1.53. Also, the amylase was raised at 98 U/L. The remainder of the blood tests, including renal function and liver function tests, were unremarkable (Table *1*).

Blood parameter	Results	Reference range
Full blood count (FBC)	—	—
Haemoglobin	133 g/L	115–165 g/L
White Blood Cell (WBC)	14.3 × 10^9/L	4–11 × 10^9/L

Platelets	245 × 10^9/L	150–450 × 10^9/L
Red Blood Cell (RBC)	4.42 × 10^{12}/L	3.5–5.5 × 10^{12}/L
Haematocrit	0.412 ratio	0.37–0.47 ratio
Mean Cell Volume (MCV)	93.2 fL	75–105 fL
Mean Corpuscular Haemoglobin (MCH)	30.1 pg	26–35 pg
Mean Corpuscular Haemoglobin Concentration (MCHC)	323 g/L	290–350 g/L
Red Cell Distribution Width (RDW)	13.60%	11–15%
Mean Platelet Volume (MPV)	10.6 fL	8.2–11.9 fL
Neutrophils (absolute)	13.2 × 10^9/L	2–7.5 × 10^9/L
Lymphocytes (absolute)	1.2 × 10^9/L	1–4 × 10^9/L
Eosinophils (absolute)	0.0 × 10^9/L	0.0–0.4 × 10^9/L
Monocytes (absolute)	0.3 × 10^9/L	0.2–0.8 × 10^9/L
Basophils (absolute)	0.0 × 10^9/L	0.0–0.1 × 10^9/L
Urea and electrolytes	—	—
Sodium	138 mmol/L	133–146 mmol/L
Potassium	3.8 mmol/L	3.5–5.3 mmol/L
Urea	4.3 mmol/L	2.5–7.8 mmol/L
Creatinine	58 µmol/L	49–90 µmol/L
Estimated glomerular filtration rate (eGFR)	>90 mL/min/1.73 m^2	90–120 mL/min/1.73 m^2
C-reactive protein (CRP)	117 mg/L	0–5 mg/L
Liver function tests	—	—
Total bilirubin	18 µmol/L	0–20 µmol/L
Alkaline phosphatase (ALP)	68 U/L	49–146 U/L
Alanine transaminase (ALT)	12 U/L	0–55 U/L
Albumin	36 g/L	35–50 g/L
Amylase	98 U/L	35–50 U/L
Coagulation screen	—	—
Prothrombin time	18.5 s	10.5–14.7 s
Prothrombin Time ratio (PT ratio)	1.53	0.87–1.21
Activated Partial Thromboplastin Time (APTT)	29.4 s	26.4–35.9 s
Activated Partial Thromboplastin Time ratio (APTT ratio)	0.95	0.85–1.15
Venous blood gas (VBG)	—	—
pH	7.424	7.31–7.41
Partial pressure of carbon dioxide (pCO_2)	5.61 kPa	5.3–6.7 kPa
Partial pressure of oxygen (pO_2)	4.04 kPa	4.5–6.0 kPa
Sodium	139 mmol/L	133–146 mmol/L
Potassium	3.9 mmol/L	3.5–5.0 mmol/L
Chloride	104 mmol/L	98–106 mmol/L
Ionised calcium	1.19 mmol/L	1.15–1.33 mmol/L

Glucose	5.7 mmol/L	3.5–5.5 mmol/L
Lactate	1.3 mmol/L	0.5–2.2 mmol/L
Total haemoglobin	138 g/L	115–165 g/L
O_2 saturation	57.30%	—
OxyHb	56.20%	—
CarboxyHb	1.40%	0.5–1.5%
MetHb	0.50%	0–1.5%
DeoxyHb	41.90%	—
Base excess (actual, calculated)	2.8 mmol/L	−3 to +3 mmol/L
Standard bicarbonate (calculated)	25.9 mmol/L	22–29 mmol/L
Anion gap (calculated)	7.8 mmol/L	8–16 mmol/L
Hematocrit (HCT) (calculated)	42.20%	35–50%
Fraction of Inspired Oxygen (FiO_2)	21.00%	—

TABLE 1: Initial laboratory investigations, including venous blood gas analysis prior to laparotomy

A contrast-enhanced CT scan of the abdomen and pelvis demonstrated free air and fluid within the peritoneal cavity, consistent with gastrointestinal perforation. There was also a small-to-moderate right-sided hydropneumothorax with underlying lung collapse. The small bowel was markedly oedematous, with significant surrounding mesenteric inflammatory change and haziness. There was a calcified uterine leiomyoma, with no gas seen within the endometrial cavity. These laboratory and imaging findings were considered consistent with a small-bowel perforation, with the hydropneumothorax being a consequence of the bowel injury (Figure *1*).

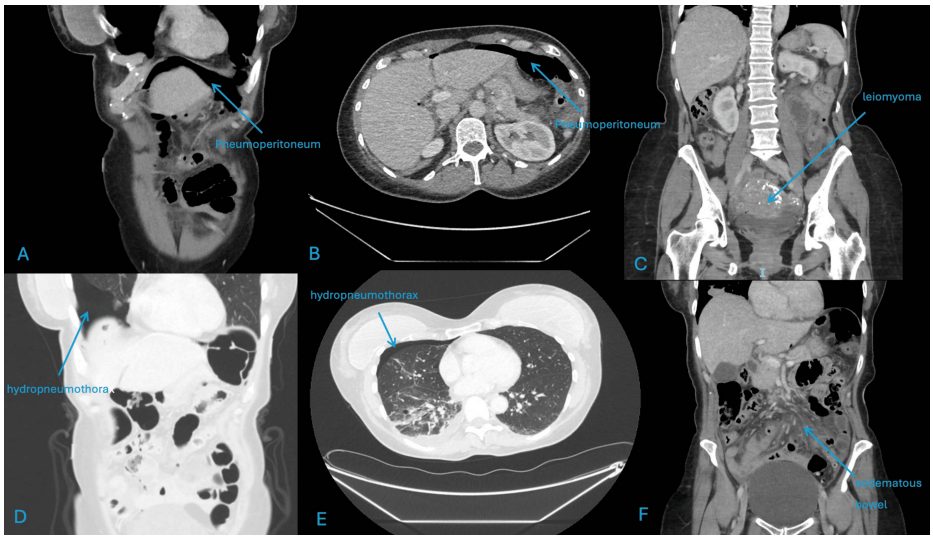


FIGURE 1: Findings on admission CT scan

A and B: demonstrate pneumoperitoneum, free air within the peritoneal cavity(coronal and axial, respectively). C: demonstrates a calcified uterine leiomyoma and no gas seen within the endometrial cavity (coronal). D and E: lung window demonstrating small-to-moderate right-sided hydropneumothorax with underlying lung collapse (coronal and axial, respectively). F: demonstrates markedly oedematous bowel with significant surrounding mesenteric inflammatory change and haziness (coronal).

The patient was urgently referred to the general surgical team and proceeded to theatre for emergency operative management. As she had commenced rivaroxaban the previous day, an urgent discussion was held

with the haematology team regarding peri-operative anticoagulation management. As the last dose of rivaroxaban had been taken the evening following the hysteroscopy, the haematology team advised that no reversal agent was required. Instead, a single dose of vitamin K and tranexamic acid was administered empirically given the raised prothrombin time rather than rivaroxaban reversal, with fresh frozen plasma available for intra-operative administration if required to minimise the risk of bleeding. Antibiotic therapy was changed to intravenous amoxicillin, metronidazole, and gentamicin, as per local guidance for an intra-abdominal source of infection.

At laparoscopy, blood was identified within the pelvis, right paracolic gutter, and subhepatic region. These findings were escalated to and discussed with the general surgery consultant, and a decision was made to proceed to midline laparotomy for definitive management. There were no free bowel contents or gross faecal contamination within the peritoneal cavity. The entire length of the small bowel was systematically examined, identifying a 7 mm perforation approximately 2.5 metres distal to the duodenojejunal flexure. Localised small-bowel contents were contained within the adjacent bowel loops and mesentery at the site of perforation. Flakes of fibrinous exudate adherent to the small-bowel loops and mesentery were also noted. It is important to note that there was no documented intrauterine perforation within the operative findings.

As the perforation was small and contained, with healthy surrounding bowel, it was closed using interrupted 3-0 Vicryl sutures, ensuring satisfactory apposition without significant luminal narrowing. The bowel was then re-examined for additional injuries, and the peritoneal cavity was thoroughly irrigated with warm saline. A Robinson drain was subsequently inserted.

Postoperatively, the patient was initially kept nil by mouth for three days due to an inability to tolerate oral intake. Intravenous proton pump inhibitor therapy and antiemetics were commenced. She received multidisciplinary team input, including review by the pain team, physiotherapists, and dietitians, to optimise her postoperative recovery. The pain team recommended morphine patient-controlled analgesia (PCA), which was used for five days. Subsequently, the pain improved significantly and was managed with oral paracetamol and codeine.

On postoperative day four, she was allowed sips of water, which were well tolerated. Therefore, her diet was subsequently advanced to thin fluids. As her oral intake improved, her CRP continued to downtrend. However, she subsequently developed a postoperative ileus on day five, requiring insertion of a nasogastric (NG) tube for free drainage, and she returned to being kept nil by mouth. The ileus persisted for four days, during which total parenteral nutrition (TPN) was required. She continued to receive regular dietetic input for nutritional support. Throughout this period, she remained afebrile and haemodynamically stable, with gradual clinical improvement. Bowel function subsequently returned. By postoperative day nine, she was able to pass flatus and open her bowels, and she was able to tolerate oral intake. The NG tube was removed, and TPN was discontinued on postoperative day nine.

On postoperative day ten, she became generally unwell and developed a new fever. Blood tests demonstrated a rise in inflammatory markers, with CRP increasing from 69 to 86 mg/L and white cell count increasing from 11.5 to 12.4 × 10^9/L. CT of the abdomen and pelvis was performed to assess for a potential postoperative intra-abdominal collection. The CT demonstrated an increase in the right-sided pleural effusion initially identified on admission with compressive atelectasis. The abdominal drain was appropriately positioned, with no free fluid or collection identified. There was no significant residual pneumoperitoneum (Figure *2*). The relationship between the initial hydropneumothorax and the subsequent pleural collection was not definitively established.

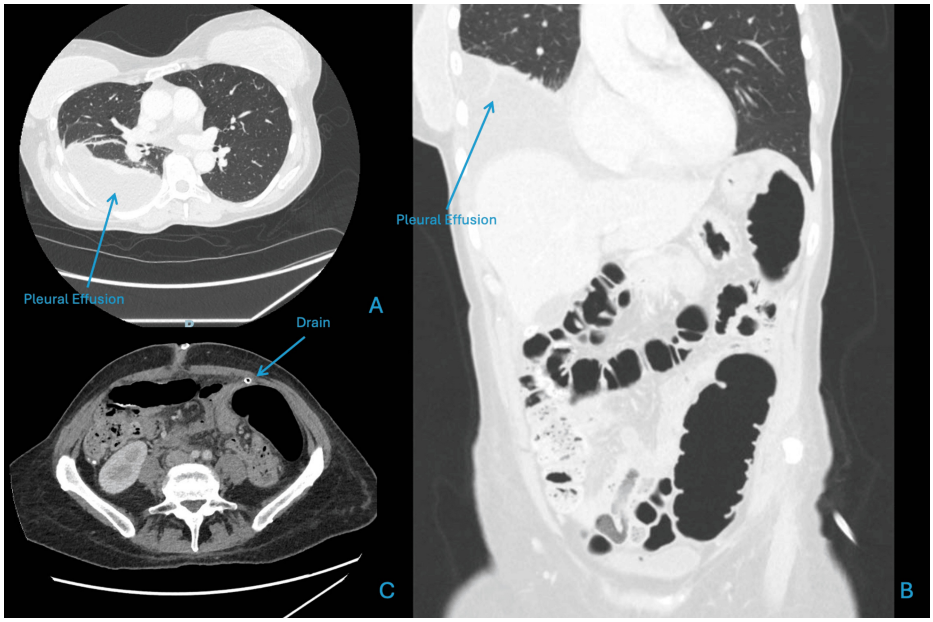


FIGURE 2: Repeat CT scan findings

A and B: lung window demonstrating right-sided pleural effusion with compressive atelectasis (axial and coronal, respectively). C: demonstrates an appropriately positioned abdominal drain with no free fluid or collection identified (axial).

The patient was reviewed by the respiratory team the same day, and bedside thoracic ultrasound demonstrated an echogenic pleural fluid collection measuring approximately four rib spaces, with significant septations. The initial impression was of a pleural effusion, possibly representing transdiaphragmatic movement of fluid from the abdomen. However, given the presence of septations, there was concern for a possible empyema. The respiratory team recommended insertion of a chest drain to facilitate drainage of the collection and obtain pleural fluid for microbiological and biochemical analysis.

Later that evening, she developed significant temperature spikes and a new oxygen requirement. A septic screen was performed, including repeat blood tests, blood cultures, and chest radiography. The chest X-ray demonstrated the previously established pleural effusion with no additional pathology (Figure *3*). It was also noted that her amoxicillin, metronidazole, and gentamicin, which had been administered for 10 days, had been discontinued from the drug chart earlier that day and were subsequently restarted that evening. The blood cultures were later reported as showing no growth.

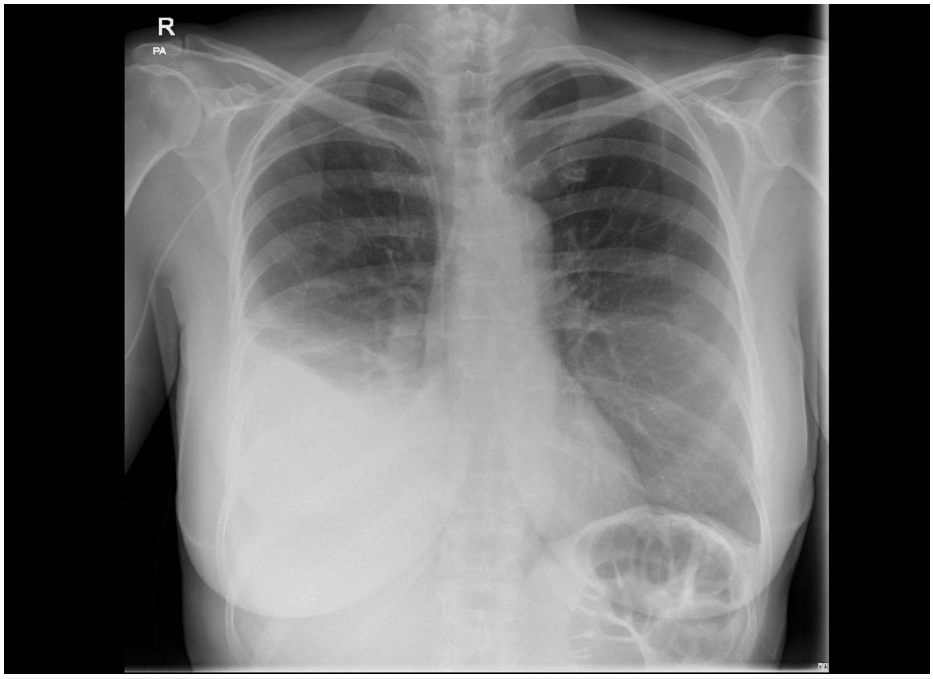


FIGURE 3: Posteroanterior chest X-ray demonstrating a right-sided pleural effusion with associated basal atelectatic change.

In view of the subsequent clinical deterioration, an intercostal chest drain was inserted the following day. Haemoserous fluid was obtained; however, the fluid did not drain freely and required syringe aspiration, with approximately 300 mL obtained. A sample was sent for cytology, acid-fast bacilli (AFB), microbiological culture and sensitivity (MCS), and biochemical analysis. The procedure was subsequently escalated to the respiratory consultant, who advised stopping the procedure and removing the drain due to the difficulty with drainage. A follow-up appointment was arranged with the pleural clinic two weeks after discharge. A prolonged course of antibiotics was also commenced, with microbiology advice sought regarding antibiotic choice and duration.

Microbiology initially advised that co-amoxiclav was an appropriate empirical antibiotic, which was commenced intravenously on postoperative day eleven, and the previous antibiotic regimen of amoxicillin, metronidazole, and gentamicin was stopped. A four-week course of co-amoxiclav was initially considered. However, given the possibility of either a reactive postoperative pleural effusion or a secondary pleural infection related to an ongoing intra-abdominal or subdiaphragmatic source, the team decided that the final duration of treatment should be guided by the pleural fluid results and clinical progress.

The patient subsequently showed clinical improvement and was weaned off supplemental oxygen three days later. However, her CRP initially remained elevated at approximately 100 mg/L. A subsequent pleural fluid analysis demonstrated a pH of 7.31, glucose of 4.6 mmol/L, and protein level of 44 g/L. These findings did not provide definitive evidence of a pleural infection. However, the significant septations identified on ultrasound remained concerning for possible loculated infected fluid.

The respiratory team advised ongoing clinical monitoring, with consideration of pleural drain insertion in the event of clinical deterioration. The CRP subsequently began to downtrend, while the patient remained clinically stable, with no further episodes of fever or oxygen requirement. Functional recovery also continued with ongoing physiotherapy and improvement in oral nutritional intake. Given the clinical improvement and downtrending inflammatory markers, microbiology advised reducing the planned antibiotic duration from four weeks to two weeks.

The patient was discharged after being successfully weaned off supplemental oxygen and following stabilisation of her inflammatory markers on day fourteen. At discharge, she was independently mobile, passing soft stools, and tolerating oral intake in small amounts, demonstrating marked clinical improvement in her condition. In terms of the clinical results, pleural fluid cytology demonstrated a bloodstained specimen containing occasional macrophages, while microbiological analysis showed scanty white cells with no organisms identified. Although pleural fluid analysis did not provide definitive evidence of infection, the effusion was managed as a possible infective parapneumonic effusion in view of the septations on ultrasound and the patient's preceding fever and inflammatory response. She was discharged with a further 11 days of oral co-amoxiclav to complete a 14-day course.

Appropriate follow-up was arranged, with review in the outpatient Respiratory Pleural Clinic and same day emergency care (SDEC) two weeks following discharge. At the SDEC review, the patient reported resolution of her presenting symptoms and continued clinical improvement. She had substantially improved her oral intake and resumed her daily activities.

Discussion

Bowel injury is a recognised but uncommon complication of gynaecological surgery [4]. Following hysteroscopic surgery, bowel injury is most commonly recognised as a potential consequence of uterine perforation; however, it is difficult to establish the incidence and characterise the clinical association between bowel injury and hysteroscopy [5]. The French National College of Gynaecologists and Obstetricians (CNGOF) guidelines recommend that laparoscopy should be performed to exclude bowel injury when uterine perforation is recognised during operative hysteroscopy using a monopolar or bipolar loop [3]. This emphasises the potential for significant intra-abdominal visceral injury to occur despite the minimally invasive nature of the initial procedure.

In this case, the clinical presentation raised concern for possible postoperative complications; however, it was not clearly suggestive of a bowel injury. The diagnosis was challenging, given that the marked abdominal pain and raised inflammatory markers were initially compatible with several differential diagnoses, such as endometritis, ovarian pathology, or uterine perforation. Initially, the lactate was within normal limits, and there was no marked drop in haemoglobin postoperatively, illustrating the possibility of bowel injury without early haemodynamic compromise or major blood loss. The persistent abdominal pain and signs of peritonism were key indicators prompting investigation for intra-abdominal pathology. The CT scan was pivotal, demonstrating intraperitoneal air and significant small-bowel abnormalities warranting urgent surgical intervention.

The precise mechanism of the bowel injury in this case remains uncertain. Possible mechanisms include direct mechanical injury following uterine perforation and thermal injury associated with electrosurgical instruments [6]. The patient had plausible factors that may have contributed to increased procedural complexity, including significant pelvic pathology, such as multiple fibroids, and previous pelvic surgery. Previous pelvic surgery may alter anatomy; however, no significant adhesions or anatomical abnormalities were documented at the time of laparotomy to explain the bowel injury. Although the close temporal relationship does suggest an association with the preceding hysteroscopic procedure, the absence of documented uterine perforation or an identifiable mechanism does not allow a definitive causal relationship to be established.

The subsequent development of a significant right-sided pleural effusion is an unusual feature and a key diagnostic challenge in this case. The initial CT scan demonstrated a small-to-moderate right-sided hydropneumothorax with an underlying lung collapse. Prevot et al. reported a case of pyopneumothorax and peritonitis which was secondary to a perforated duodenal ulcer, attributing the pleural findings to a possible pleuroperitoneal communication and transdiaphragmatic passage of infected fluid [8]. In this case, the aetiology of this thoracic finding was uncertain; however, the co-existence with bowel injury raises suspicion of a possible related inflammatory or infective process between the abdominal and pleural cavities.

The subsequent thoracic ultrasound demonstrated significant septations, while the patient developed a fever, new oxygen requirement, and a rising inflammatory response, raising concern for a pleural infection; however, an infective or reactive parapneumonic effusion could not be confirmed. The pleural fluid analysis demonstrated a pH of 7.31 and glucose of 4.6mmol/l. The 2023 British Thoracic Society guideline identifies a pleural fluid pH between 7.2 and 7.4 as an intermediate risk category in suspected pleural infection requiring interpretation alongside other clinical, biochemical and radiological features [9]. In this case, the absence of organisms on microbiological analysis, the non-diagnostic cytology and the subsequent clinical improvement made a reactive or parapneumonic effusion more likely than a confirmed empyema. However, the presence of significant septations meant that an infected or loculated component could not be initially excluded. Conversely, the temporal relationship with recent intra-abdominal pathology and surgery raised the possibility of postoperative effusion. In view of the non-diagnostic results and clinical improvement of the patient, the mechanism or cause of the effusion remains uncertain in this case, highlighting the importance of respiratory follow-up and consideration of repeat drain insertion in the event of clinical deterioration.

To date, only a small number of cases of bowel perforation following hysteroscopy have been reported in the literature, with only two, including the present case, describing bowel injury in the absence of documented uterine perforation [10-12]. Hsu et al. reported a 30-year-old woman who developed spontaneous small-bowel perforation following operative hysteroscopy, without evidence of uterine perforation, with similarities to the presentation in our patient [10]. In contrast, Watkins et al. described two ileal perforations following hysteroscopic myomectomy in a 33-year-old patient, in whom uterine perforation was suspected alongside bowel injury [11]. Dofutsu et al. reported simultaneous uterine and sigmoid colon perforation following hysteroscopy-guided microwave endometrial ablation in a patient with a bicornuate uterus and multiple myomas, providing a more apparent anatomical context for the associated bowel injury [12]. Our

case is notable for the identification of a discrete small-bowel perforation remote from the pelvis, in the absence of documented uterine perforation or an identifiable intra-operative mechanism. Therefore, significant intra-abdominal visceral injury should remain an important differential diagnosis in patients who develop severe abdominal pain or peritonism following hysteroscopic surgery, even in the absence of haemodynamic instability or documented uterine perforation.

Overall, this case highlights several key clinical considerations following operative hysteroscopy. Significant visceral injury should be considered as a differential diagnosis following the development of postoperative pain and signs of peritonism, even in the absence of haemodynamic compromise. This reinforces the importance of prompt reassessment rather than attributing symptoms to expected postoperative findings. This case demonstrates the importance of timely imaging when clinical findings are concerning but non-specific. Furthermore, the subsequent development of a pleural effusion in the context of intra-abdominal pathology should prompt consideration of not only pleural infection but also reactive causes. Repeated clinical reassessment and integration of evolving clinical, radiological, and biochemical findings are therefore essential when managing unexpected postoperative complications.

Conclusions

In conclusion, bowel perforation is a rare but potentially serious complication of operative hysteroscopy and should remain an important differential diagnosis of intra-abdominal injury in patients presenting with severe and persistent abdominal pain and signs of peritonism, even in the absence of haemodynamic instability. Early imaging can facilitate prompt diagnosis and facilitate timely surgical management.

This case also highlights the diagnostic complexity of a subsequent pleural effusion in the context of intra-abdominal pathology. Although pleural infection should be considered in patients who are febrile, have raised inflammatory markers and septated pleural fluid; a reactive pleural effusion secondary to intra-abdominal inflammation cannot be excluded. Thus, careful interpretation of pleural fluid results should be considered alongside the overall clinical, biochemical, and radiological findings. Overall, this case emphasises the importance of maintaining a broad differential diagnosis, timely investigation and repeat clinical assessment when unexpected complications arise following hysteroscopic surgery.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Gina Abdel-Hady, Amirah Bhatti

Acquisition, analysis, or interpretation of data: Gina Abdel-Hady, Amirah Bhatti

Drafting of the manuscript: Gina Abdel-Hady, Amirah Bhatti

Critical review of the manuscript for important intellectual content: Gina Abdel-Hady, Amirah Bhatti

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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Gina Abdel-Hady and Amirah Bhatti contributed equally to the work and should be considered co-first authors.

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