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DIAGNOSIS AND TREATMENT OF INTERRELATED COMPLICATIONS FOLLOWING SPLENIC INJURY

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The article presents a clinical case of diagnosis and treatment of a patient who was admitted to the surgical department with signs of acute peritonitis and a history of a previous catatrauma. In the preoperative period, abdominal abscesses were detected in the patient during an ultrasound examination, and during the operation, the cause of their occurrence was established – a microrupture of the spleen, which occurred during a fall from a height. Also, the patient had diffuse peritonitis as a result of perforation of the duodenal ulcer, which was provoked by prolonged use of nonsteroidal anti-inflammatory drugs, which she took to reduce the intensity of the abdominal pain after the injury. The patient was discharged from the hospital on the 7th day in satisfactory condition. Therefore, in patients who indicate a previous catatrauma, it is necessary to diagnose damage to the internal organs of the abdominal cavity and their possible complications, and when performing surgical intervention – perform a thorough inspection of the abdominal cavity for the detection of such pathology.

Key words: splenic trauma, diffuse peritonitis, abdominal abscesses, nonsteroidal anti-inflammatory drugs, perforated duodenal ulcer, diagnostics, treatment.

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ДІАГНОСТИКА І ЛІКУВАННЯ ВЗАЄМОПОВ'ЯЗАНИХ УСКЛАДНЕНЬ, ЯКІ ВИНИКЛИ ПІСЛЯ ПОШКОДЖЕННЯ СЕЛЕЗІНКИ

У статті наведений клінічний випадок діагностики і лікування пацієнтки, яка надійшла до хірургічного відділення із ознаками гострого перитоніту та наявністю у анамнезі отриманої напередодні кататравми. У передопераційному періоді під час проведення ультразвукового обстеження у хворої виявлено абсцеси черевної порожнини, а під час операції встановлено причину їх виникнення – мікророзрив селезінки, який виник під час падіння з висоти. Також, у хворої відзначено розлитий перитоніт в результаті перфорації виразки дванадцятипалої кишки, яка спровокована тривалим прийомом нестероїдних протизапальних препаратів, які вона приймала для зменшення інтенсивності болю в животі після травми. Хвора виписана із стаціонару на 7 добу у задовільному стані. Тому, у пацієнтів, які вказують на отриману напередодні кататравму, необхідно проводити діагностику пошкоджень внутрішніх органів черевної порожнини та їх можливих ускладнень, а при виконанні оперативного втручання – виконувати ретельну ревізію черевної порожнини на предмет виявлення такої патології.

Ключові слова: травма селезінки, розлитий перитоніт, абсцеси черевної порожнини, нестероїдні протизапальні препарати, перфоративна виразка дванадцятипалої кишки, діагностика, лікування.

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The spleen is the visceral organ most frequently injured in blunt abdominal trauma, accounting for approximately 20 % of all abdominal injury cases [8]. Motor vehicle accidents are responsible for 50 % to 75 % of these injuries, followed by direct injuries to the abdomen and falls from a height. Splenic rupture may occur either immediately at the time of trauma or at a later stage [1]. Splenic injury does not always present with a typical clinical picture; indeed, rupture can occur following minor trauma or even in the absence of any identifiable precipitating event [19]. The primary complication of splenic injury is the development of hemoperitoneum. If blood accumulates in the abdominal cavity, abscesses and adhesions may subsequently form at the sites of fluid collection [14].

Localized collections of pus within the abdominal cavity can occur in various anatomical

regions, including the peritoneal cavity, the retroperitoneal space, and on the surface of visceral organs [7]. The most common symptoms of intra-abdominal abscesses are persistent abdominal pain, fever, and general weakness. These symptoms are often accompanied by nausea, loss of appetite, and weight loss. The abdominal pain is typically constant and may be localized or diffuse, depending on the location of the abscess. The clinical presentation ranges from mild to severe; some patients develop systemic inflammatory response syndrome which, in advanced cases, progress to sepsis [4].

Persistent pain often leads to the use of analgetic drugs. Non-steroidal anti-inflammatory drugs (NSAIDs) possess such properties, offering significant anti-inflammatory and analgesic effects; they account for 8 % of prescribed medications worldwide. However, there has recently been an

increase in their use without a prescription. Notably, 26 % of patients use doses exceeding the recommended limits, often without consulting healthcare professionals [20]. The most well-known adverse effects of these drugs include upper gastrointestinal ulcers, which can lead to bleeding or perforation. However, such complications can occur with or without symptoms, but with presence of mucosal damage in the upper, middle, or lower gastrointestinal tract; deep ulcers may progress to perforation [15]. The risk of upper gastrointestinal bleeding or perforation depends on the specific type of NSAID. For instance, selective COX-2 inhibitors are less gastrointestinally harmful than non-selective ones; ibuprofen is generally safer, whereas diclofenac or naproxen carry a higher risk of mucosal injury [3].

Therefore, the uncontrolled use of non-steroidal anti-inflammatory drugs (NSAIDs) can lead to serious complications, primarily involving the gastrointestinal tract. Indeed, the primary established risk factor for perforated gastric or duodenal ulcers is the long-term use of NSAIDs; other factors include advanced age, a history of peptic ulcer disease, and the consumption of herbal supplements, which may cause mild irritation of the gastric mucosa. However, their role is minimal compared to the direct impact of NSAIDs on the development of severe gastrointestinal complications. Delayed diagnosis and treatment of perforated ulcers are closely linked to increased morbidity and mortality; therefore, early recognition of this condition is crucial [13, 16]. NSAIDs, as well as certain herbal supplements and alternative medicine products, cause gastric mucosal injury by inhibiting cyclooxygenase enzymes and reducing prostaglandin synthesis; this leads to decreased blood flow and reduced secretion of bicarbonate and mucus, ultimately resulting in an increased susceptibility to ulcer formation and perforation [5, 10]. At the same time, according to estimates by some researchers, the annual incidence of perforated ulcer ranges from 3.8 to 14 per 100,000 population, making it the second most common complication of peptic ulcer disease after upper gastrointestinal bleeding [6, 18].

Thus, the combination of abdominal trauma and the uncontrolled, self-administered use of NSAIDs to relieve abdominal pain leads to serious gastrointestinal disorders that can even be life-threatening.

The purpose of the study was to illustrate, through a clinical case, the complications that may arise from splenic injury combined with the uncontrolled use of non-steroidal anti-inflammatory drugs, and to describe the specifics of their diagnosis and treatment.

Materials and methods. This article presents a detailed description of the clinical case of female patient K. (born in 1992), focusing on the diagnosis and treatment of a perforated chronic duodenal ulcer. The perforation occurred on the background of uncontrolled use of non-steroidal anti-inflammatory drugs (NSAIDs) and analgesics, taken to manage abdominal pain resulting from a fall from a tree – an

injury that had caused splenic damage and subsequent development of abdominal abscesses. A detailed analysis was conducted of the medical records of the patient, who was treated in the surgical department of the Municipal Enterprise "2-nd City Clinical Hospital of the Poltava City Council" from June 18 to June 24, 2025. Clinical monitoring of the patient included assessment of her condition during the preoperative and early postoperative periods, as well as follow-up observations at 1 and 6 months.

On June 18, 2025, at 12:00 PM, the patient has been transported by an emergency team to the admission department of the Municipal Enterprise "2nd City Clinical Hospital of the Poltava City Council" with a diagnosis of "ruptured ectopic pregnancy." She presented with complaints of acute (described as a "dagger-like" sensation) and unbearable pain throughout the abdomen – most intense in the epigastrium and right hypochondrium – as well as nausea, vomiting, and general weakness. Her medical history indicates that acute upper abdominal pain had appeared suddenly four hours prior admission; taking painkillers provided no relief – on the contrary, the pain spread across the entire abdomen, prompting her to seek medical attention. Further history-taking revealed that on June 5, 2025, she had fallen from a tree, subsequently developing mild pain in the epigastric region and left hypochondrium. She did not ask for medical help at that time, even though the pain gradually intensified, leading her to self-medicate with non-steroidal anti-inflammatory drugs (NSAIDs), which provided temporary relief. Her use of these medications was uncontrolled; she would have taken the pills almost immediately upon the worsening of pain, doing them up to 5-7 times a day. Gradually, the pain spread to the upper abdomen, accompanied by an increase of body temperature to 37.4–38.3°C, which she thought was an acute respiratory disease. Her medical history indicates satisfactory living conditions. She denies a history of tuberculosis, infectious hepatitis, venereal diseases, or chronic illnesses. There is no significant history of allergies. She reports no prior surgical interventions.

In the emergency department, the patient underwent a clinical examination by a gynecologist, a surgeon, and an internist. A physical examination was performed, focusing on signs of injury resulting from a fall (catatrauma) – specifically, the presence of hematomas and skin lesions on the patient's body. Laboratory and instrumental tests were also done. Laboratory assessments included a complete blood count, coagulation studies, biochemical blood analysis (measuring serum electrolytes, bilirubin fractions, glucose, liver enzymes, renal function markers, and C-reactive protein), blood typing and Rh factor determination, and urine analysis. Instrumental examinations included abdominal ultrasound, electrocardiography, and chest X-ray. The patient provided written consent for the examinations, as well as for the surgical and conservative treatments.

To monitor the course of the disease, the patient was examined by the attending physician one and six

months after the surgery. During the one-month follow-up visit, the patient underwent a fibrogastroduodenoscopy. According to the decision of the Bioethics Commission of Poltava State Medical University, the study materials complied with relevant ethical principles, specifically the provisions of the Declaration of Helsinki and current Ukrainian legislation.

Results of the study. During the physical examination by the surgeon, the patient was noted to be pale and thin build; she was in conscious but somewhat sluggish. She lay in a forced position on her left side with her legs drawn up toward her abdomen. An older (up to 2 weeks) subcutaneous hematoma measuring 3x4 cm was observed on the medial surface of the upper third of the right shin, and a similar older subcutaneous hematoma with a diameter of up to 6.0 cm was noted on the lateral surface of the upper third of the right thigh. An older subcutaneous hematoma with a diameter of up to 4.5 cm was also visible on the lateral surface of the upper third of the left shin. Edema was noted in both shins; finger-pressure marks persisted for 7-8 seconds. Vesicular breath sounds were heard throughout the lungs, though slightly diminished in the lower zones. Heart sounds were distinct and rhythmic. Pulse was 112 beats per minute; blood pressure was 120/70 mmHg. The tongue was dry, with a white coating. The abdomen was symmetrical and distended, and did not participate in respiratory movements. Upon superficial palpation, the abdomen was tense and tender in all quadrants; deep palpation was not possible due to pain. Signs of peritoneal irritation were positive. Abdominal auscultation revealed diminished bowel sounds. There were no reported disturbances of urination or defecation. Digital rectal examination reveals satisfactory sphincter tone; the rectal ampulla is filled with liquid stool, and no organic pathology is detected in the lumen; stool of normal color was present on the glove. Neurological status is unremarkable. The patient was examined by a gynecologist in the emergency department, and no evidence of acute gynecological pathology was found.

An ultrasound examination of the abdominal cavity performed by a surgeon revealed a significant amount of fluid and free gas throughout the abdomen, as well as presence of a suprahepatic, subhepatic, and interintestinal abscesses. A hyperechogenic mass with fluid content, measuring up to 5.0 cm in diameter, was identified near the upper pole of the spleen. The patient was urgently admitted to the surgical department, where she underwent further assessment and preoperative preparation.

Laboratory test results. Complete blood count (CBC) dated June 18, 2025: erythrocytes – $1.78 \times 10^{12}/L$, hemoglobin – 54 g/L, leukocytes – $13.9 \times 10^9/L$, platelets – $236 \times 10^9/L$, ESR – 39 mm/h, neutrophils – 92.8 %, lymphocytes – 5.5 %, monocytes – 1.6 %, eosinophils – 0.1 %, basophils – 0 %. Blood glucose (June 18, 2025) – 4.4 mmol/L. Coagulation profile (June 18, 2025): prothrombin time – 12.18 seconds, fibrinogen – 7.14 g/L, activated

partial thromboplastin time (aPTT) – 22.3 seconds, international normalized ratio (INR) – 1.02. Biochemical blood analysis dated June 18, 2025: total protein – 36.8 g/L, aspartate aminotransferase (AST) – 66.8 U/L, alanine aminotransferase (ALT) – 39.9 U/L, total bilirubin – 7.02 $\mu\text{mol}/L$ (direct – 4.03 $\mu\text{mol}/L$, indirect – 2.99 $\mu\text{mol}/L$), urea – 8.27 mmol/L, creatinine – 80.2 $\mu\text{mol}/L$. Urine analysis dated June 18, 2025: specific gravity 1015, pH 5.0, color amber, protein 0.25 g/L; no other abnormalities detected. ECG dated June 18, 2025: heart rate 120 bpm, sinus tachycardia, vertical electrical axis of the heart, repolarization abnormalities in the posterior wall myocardium, depression in the anterior wall myocardium. Chest X-ray dated June 18, 2025 (supine position, anteroposterior view): lung fields clear of pathological opacities, slightly accentuated lung markings, lung roots well-defined, costophrenic angles clear, mediastinal shadow normal.

The patient was examined by an internist; the established diagnosis was: arterial hypertension (Stage I, Grade 2), moderate risk; duodenal ulcer disease complicated by perforation (June 18, 2025); moderate secondary anemia.

Thus, based on complaints, medical history, and objective, laboratory, and instrumental examination findings, the patient was diagnosed with: acute duodenal ulcer disease complicated by perforation; chronic splenic abscess with microperforation; suprahepatic, subhepatic, and interloop abscesses; diffuse fibrinous-purulent peritonitis; and arterial hypertension (Stage I).

The patient was urgently taken to the operating room at 14:30 on June 18, 2025. The procedure performed was laparotomy, excision of a perforated duodenal ulcer with duodenoplasty, splenectomy, visceral adhesiolysis, peritoneal lavage, and drainage of the abdominal cavity.

Following preparation of the operative field, general intravenous anesthesia with endotracheal intubation was administered. An upper midline laparotomy was performed. Upon exploration of the abdominal cavity, approximately 1,500 mL of purulent-fibrinous and hemorrhagic exudate was identified and evacuated, and the abdominal cavity was dried. The liver was dark brown in color, covered with fibrin, moderately enlarged, with a rounded inferior edge. The gallbladder was located in its usual anatomical position, measuring $7.0 \times 4.0 \times 2.5$ cm, with no gross pathological changes. Subhepatic, suprahepatic, and interloop abscesses were identified. Approximately 10 cm of the small bowel loops adjacent to the abscesses were moderately adherent to each other; visceral adhesiolysis was therefore performed. The small bowel loops were not distended, peristalsis was active, the serosal surface was shiny, and mesenteric arterial pulsation was satisfactory. In the pyloric region, along the anterior wall of the duodenal bulb, a duodenal ulcer measuring approximately 2.0×2.5 cm was identified. The ulcer had indurated margins and a central perforation measuring up to 1.5 cm in diameter. A nasogastroduodenal tube was placed. The ulcer

margins were excised, followed by two-layer closure (duodenoplasty) of the duodenal wall. Exploration of the spleen revealed a dark-red organ covered with fibrin. An abscess measuring up to 5.0 cm in diameter was identified at the upper pole, with perforation and evacuation of pus and liquefied blood. A blood clot measuring approximately 100 mL was removed from the splenic site. A decision was made to perform splenectomy. The splenic vascular pedicle was doubly ligated and transected, and the spleen was removed. Hemostasis was checked and found to be adequate, with no active bleeding. Repeat exploration revealed no other pathological findings within the abdominal cavity. The abdominal cavity was thoroughly irrigated with antiseptic solutions, dried, and drained using two Blake drains placed in the pelvis, one Blake drain in the subhepatic space, and Redon and Blake drains in the left upper quadrant. The abdominal incision was closed in layers, and sterile dressings were applied.

In the postoperative period, the patient was treated in the intensive care unit, where she received detoxification, antibacterial, anticoagulant, and analgesic therapy, as well as transfusions of red blood cells and plasma. On the second day after surgery, the patient was transferred to the surgical department. The early postoperative course was uncomplicated; body temperature normalized on the second day, and abdominal drains were removed between the third and fifth days. Follow-up laboratory tests were performed. Complete blood count (June 21, 2025): erythrocytes – $3.24 \times 10^{12}/L$, hemoglobin – 97 g/L, leukocytes – $12.3 \times 10^9/L$, platelets – $360 \times 10^9/L$, ESR – 27 mm/h, neutrophils – 87.8 %, lymphocytes – 8.2 %, monocytes – 3.5 %, eosinophils – 0.4 %, basophils – 0.1 %. Coagulation profile (June 21, 2025): prothrombin time – 11.76 seconds, fibrinogen – 5.19 g/L, aPTT – 24.45 seconds, INR – 0.98. Biochemical blood analysis dated June 21, 2025: total protein – 58.4 g/L, AST – 56.3 U/L, ALT – 43.2 U/L, total bilirubin – 5.08 $\mu\text{mol/L}$ (direct – 2.8 $\mu\text{mol/L}$, indirect – 2.28 $\mu\text{mol/L}$), urea – 7.5 mmol/L, creatinine – 71.4 $\mu\text{mol/L}$. Urine analysis dated June 21, 2025 – no abnormalities.

Pathological examination results: the spleen shows congestion of the red pulp and a focus of necrosis with marked leukocyte infiltration in the upper pole. Duodenal ulcer margins: the small intestine wall exhibits focal mucosal necrosis with leukocyte infiltration, as well as sclerosis of the submucosa, muscularis, and blood vessels, consistent with a chronic ulcer.

The patient was discharged from the surgical department in satisfactory condition on June 24, 2025. At the time of discharge, she reported no abdominal pain and a normal body temperature; postoperative wounds showed no signs of inflammation, and the sutures were intact. The patient was referred to an outpatient surgeon for follow-up. The patient was examined one and six months after surgery; her condition is satisfactory. She reports no abdominal pain, does not strictly adhere to a specific diet, and leads a full life. Fibrogastroduodenoscopy

performed one month after hospital discharge showed healing of the duodenal ulcer with scar formation.

Discussion. Analysis of this clinical case led to the conclusion that, following a fall from a tree (June 6, 2025), the patient sustained a closed abdominal injury involving damage to the upper pole of the spleen. This was complicated by hemoperitoneum – manifesting as progressively worsening pain – and the accumulation of blood in the subdiaphragmatic and subhepatic spaces, as well as between intestines and the spleen, resulting in abscess formation in these areas. Due to persistent abdominal pain, the patient engaged in prolonged, uncontrolled use of oral non-steroidal anti-inflammatory drugs (NSAIDs). In the presence of an asymptomatic chronic duodenal ulcer, this medication use triggered an ulcer perforation (June 18, 2025), leading to diffuse fibrinous-purulent peritonitis.

In this case, the patient presented with a classic "acute abdomen" clinical picture, characterized by the sudden onset of severe epigastric pain radiating across the entire abdomen, nausea, vomiting, and general weakness. Clinical findings included peritoneal signs and the presence of significant fluid and free gas throughout the abdominal cavity, along with suprahepatic, subhepatic, and inter-intestinal abscesses detected by ultrasound. The timely detection of these signs and immediate referral for surgical treatment played a decisive role in the patient's favorable outcome, as a diagnostic delay exceeding 24 hours is invariably associated with a significant increase in mortality. The patient's laboratory findings were also consistent with this clinical presentation; specifically, the presence of leukocytosis with marked neutrophilia indicated an acute inflammatory response to generalized peritonitis. These pathological changes further confirmed the clinical diagnosis and the necessity for urgent surgical intervention [18].

Gastroduodenal ulcer disease arises from an imbalance between aggressive factors (primarily gastric acid and pepsin) and mucosal protective mechanisms, such as mucus and bicarbonate secretion, mucosal blood supply, and prostaglandin synthesis [9, 17]. In this context, the patient's use of NSAIDs remains one of the most well-established risk factors for mucosal injury and the development of associated complications. This clinical case involves the prolonged, uncontrolled, and self-administered use of such drugs, which generally inhibit cyclooxygenase-1 and -2 (COX-1 and COX-2) activity, thereby reducing prostaglandin synthesis [2]. COX-1 inhibition compromises gastrointestinal mucosal defense by reducing mucus and bicarbonate secretion, impairing blood supply, and slowing epithelial repair, which increases susceptibility to injury. This mechanism is associated with serious gastrointestinal complications – specifically bleeding, ulcer formation, and perforation [11, 12] – which was observed in this patient.

Thus, this clinical case demonstrates the interconnected progression of complications following blunt abdominal trauma involving

splenic injury. Based on the pathomorphological findings, the final clinical diagnosis is formulated as follows: blunt abdominal trauma with splenic injury (June 5, 2025); chronic splenic abscess with micro-perforation; suprahepatic, subhepatic, and inter-intestinal abscesses; chronic duodenal ulcer disease, acute stage, complicated by perforation

(June 18, 2025); diffuse fibrinous-purulent peritonitis; stage I arterial hypertension; and subcutaneous hematomas of both lower extremities. Surgical intervention (June 18, 2025): laparotomy, ulcer excision with duodenoplasty, splenectomy, viscerolysis, and lavage and drainage of the abdominal cavity.

Conclusion

Consequently, when patients are admitted to a surgical unit presenting with signs of acute peritonitis and a history of a recent fall (catatrauma), it is essential to conduct an assessment aimed at detecting injuries to abdominal organs and associated complications; furthermore, during surgical intervention, a full exploration of the abdominal cavity must be performed to identify such pathology.

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Conflict of interest. The authors have no conflicts of interest to declare.

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