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| RESEARCH ARTICLE

Convulsions and Catastrophe: Status Epilepticus Complicated by Massive Pneumoperitoneum and Bowel Perforation: A Case Report

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| ABSTRACT

We report the case of a sixteen-year-old female with a known history of epilepsy who presented to the emergency department following prolonged generalized tonic-clonic seizures at home, consistent with status epilepticus, after recent noncompliance with her antiepileptic regimen. She had experienced multiple convulsive episodes over twenty minutes without full recovery between seizures, prompting urgent ambulance transport. On arrival, she remained confused, drowsy, and tachycardic, with stable oxygenation on supplemental oxygen. Initial examination revealed subtle postictal features including tongue biting, but no focal neurological deficits were detected. Unexpectedly, progressive abdominal distension was noted, with the abdomen tense and tympanic on percussion, raising concern for an acute intra-abdominal process. Urgent chest radiography demonstrated massive bilateral subdiaphragmatic free air, confirmed by contrast-enhanced CT of the abdomen, which revealed focal distal ileal perforation with surrounding inflammation and small amounts of free fluid, consistent with pneumoperitoneum secondary to bowel perforation. Laboratory studies demonstrated mild leukocytosis, elevated creatine kinase, and metabolic acidosis, reflecting physiological stress from prolonged seizures. The patient underwent multidisciplinary management, including intravenous anticonvulsants for seizure control, supportive care with fluids and oxygen, broad-spectrum antibiotics, and urgent surgical intervention with resection of the perforated ileal segment and primary anastomosis. Postoperatively, she was monitored in the intensive care unit, with gradual neurological recovery, resolution of abdominal distension, and restoration of bowel function. This case illustrates the rare but serious occurrence of seizure-induced gastrointestinal perforation, emphasizing the importance of vigilant clinical assessment, early imaging, and coordinated multidisciplinary care in adolescents with status epilepticus to identify and manage life-threatening complications promptly.

| KEYWORDS

Epilepsy, Seizure, Status Epilepticus, Perforation, Surgical Abdomen, Air Under Diaphragm, Pneumoperitoneum, Benzodiazepines

| ARTICLE INFORMATION

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Introduction

Status epilepticus is one of the most serious neurological emergencies encountered in clinical practice. It is characterized by prolonged or recurrent seizure activity that persists without recovery of consciousness between episodes. If not treated promptly, ongoing seizure activity can lead to significant neurological injury, systemic complications, and death. Despite advances in emergency and critical care management, status epilepticus continues to carry considerable morbidity and mortality, particularly when treatment is delayed or when complications develop during the course of illness [1,3]. Early recognition and rapid initiation of therapy are therefore essential in improving outcomes for affected patients. The current clinical definition of status epilepticus emphasizes the importance of early intervention. Continuous seizure activity lasting more than five minutes or recurrent seizures without full recovery of consciousness are now generally considered sufficient to establish the diagnosis and initiate treatment. This definition reflects the understanding that seizures persisting beyond this time frame are unlikely to stop spontaneously and may rapidly progress to a more refractory state. As seizure activity continues, a cascade of metabolic and neuronal changes occurs that can make the condition increasingly resistant to treatment [1,2]. The pathophysiology of status epilepticus involves complex neurochemical and cellular mechanisms. Persistent neuronal excitation leads to excessive release of excitatory neurotransmitters, particularly glutamate, along with reduced inhibitory signaling through gamma aminobutyric acid pathways. These changes promote ongoing electrical activity within the brain and contribute to neuronal injury when seizures are prolonged. In addition to direct neurological effects, systemic complications may occur as a result of increased metabolic demand, hypoxia, acidosis, and autonomic instability [1]. Patients with status epilepticus often require urgent stabilization in the emergency department or intensive care unit. Initial management focuses on airway protection, circulatory support, and prompt administration of anticonvulsant therapy. Benzodiazepines are typically used as first line agents because of their rapid onset of action and effectiveness in terminating seizures. If seizures persist, additional medications such as levetiracetam, phenytoin, or valproate may be administered. In refractory cases, continuous infusion of anesthetic agents and intensive monitoring may be required [2,4]. Although neurological injury is the most recognized consequence of prolonged seizures, status epilepticus can also lead to complications involving multiple organ systems. Prolonged muscle activity during convulsive seizures may result in metabolic acidosis, hyperthermia, rhabdomyolysis, and electrolyte disturbances. Cardiovascular instability, aspiration pneumonia, and acute respiratory failure may also occur during severe episodes. These systemic complications often contribute significantly to patient morbidity and may complicate the clinical course in critically ill individuals [3,6]. In some cases, the severity of generalized convulsive activity can produce unexpected complications involving the thoracic or abdominal cavities. Forceful muscle contractions, increased intrathoracic pressure, and repeated Valsalva maneuvers during seizures may lead to rare but clinically important conditions such as pneumomediastinum, pneumothorax, or pneumoperitoneum. Although these complications are uncommon, they can create significant diagnostic challenges when they occur in patients with ongoing neurological illness [11,12]. Pneumoperitoneum refers to the presence of free air within the peritoneal cavity. In most clinical settings, pneumoperitoneum is strongly associated with perforation of a hollow abdominal organ and is generally considered a surgical emergency. Common causes include perforated peptic ulcer disease, bowel ischemia, trauma, and complications related to abdominal procedures. The presence of intraperitoneal free air on imaging often prompts urgent surgical evaluation because untreated gastrointestinal perforation can rapidly lead to peritonitis and sepsis [11]. However, not all cases of pneumoperitoneum are caused by visceral perforation. In a small proportion of patients, free intraperitoneal air may occur without an identifiable gastrointestinal rupture. This condition is sometimes described as spontaneous or nonsurgical pneumoperitoneum. Reported mechanisms include barotrauma from mechanical ventilation, thoracic air leak syndromes, and migration of air from the mediastinum into the abdominal cavity through anatomical fascial planes. In such cases, careful clinical assessment is required to distinguish benign causes from those requiring urgent surgical intervention [11,14]. Another condition that may contribute to pneumoperitoneum is pneumatosis intestinalis, which refers to the presence of gas within the wall of the gastrointestinal tract. Pneumatosis intestinalis can occur in association with a variety of conditions including bowel ischemia, infection, inflammatory disorders, and certain medications. In severe cases, disruption of the intestinal wall may lead to leakage of gas into the peritoneal cavity and the development of pneumoperitoneum. The clinical significance of pneumatosis intestinalis varies widely, ranging from benign incidental findings to life threatening intestinal ischemia or perforation [10]. Bowel perforation remains one of the most serious causes of pneumoperitoneum and requires prompt diagnosis and treatment. Perforation of the gastrointestinal tract allows leakage of intestinal contents into the peritoneal cavity, resulting in chemical and bacterial peritonitis. Patients may present with acute abdominal pain, abdominal distension, fever, and signs of systemic infection. Imaging studies such as abdominal radiography or computed tomography are often crucial in detecting free intraperitoneal air and identifying the site of perforation [12]. The development of pneumoperitoneum in critically ill patients may be particularly challenging to recognize. Many patients with severe neurological illness are sedated, intubated, or unable to communicate symptoms clearly. As a result, abdominal complications may initially be overlooked or attributed to other causes. Subtle clinical signs such as abdominal distension, unexplained hypotension, or worsening metabolic abnormalities may provide early clues that an intra abdominal complication has developed [12]. Rare combinations of neurological and abdominal emergencies present significant diagnostic and therapeutic challenges for clinicians. In patients with status epilepticus, attention is often focused primarily on seizure control and stabilization of neurological function. However, clinicians must also remain vigilant for secondary complications that may arise during the course of illness. Failure to recognize associated surgical

conditions such as bowel perforation can result in delayed intervention and increased risk of morbidity or mortality. Reports describing pneumoperitoneum associated with systemic or non abdominal conditions highlight the complexity of diagnosing free intraperitoneal air in unusual clinical contexts. In some cases, air may dissect from the thoracic cavity into the abdominal cavity through the mediastinum and retroperitoneal spaces. This process has been observed in patients with severe coughing, mechanical ventilation, or pulmonary air leak syndromes. These mechanisms demonstrate that pneumoperitoneum does not always originate directly from gastrointestinal perforation, although distinguishing between these possibilities remains critical in clinical decision making [12,13]. Because pneumoperitoneum is commonly linked to gastrointestinal perforation, its identification usually triggers urgent surgical consultation. Computed tomography has become the most sensitive imaging modality for detecting free intraperitoneal air and evaluating associated abdominal pathology. CT imaging may also reveal findings such as pneumatosis intestinalis, bowel wall thickening, or localized inflammation that can help determine the underlying cause. Early imaging therefore plays a key role in guiding management decisions in patients with suspected intra abdominal emergencies [10,12]. Case reports have an important role in highlighting unusual clinical presentations and rare complications of common conditions. While status epilepticus is well described in neurological literature, reports describing its association with severe intra abdominal complications are limited. The coexistence of prolonged seizure activity with conditions such as pneumoperitoneum or bowel perforation is particularly rare and may create significant diagnostic uncertainty during the acute phase of management. Documenting such cases can contribute to greater awareness among clinicians regarding potential complications that may arise during severe neurological illness. Recognition of unexpected abdominal pathology in patients with status epilepticus requires careful clinical observation, appropriate imaging, and multidisciplinary collaboration between emergency physicians, neurologists, intensivists, and surgeons. Early detection and timely intervention may be critical in preventing further deterioration. The present case report describes a patient who developed status epilepticus complicated by massive pneumoperitoneum and bowel perforation. The case highlights the diagnostic challenges encountered when a neurological emergency is accompanied by an acute abdominal catastrophe. Through detailed description of the clinical course, investigations, and management, this report aims to emphasize the importance of maintaining a broad differential diagnosis when unexpected complications arise in critically ill patients. Increased awareness of such rare presentations may assist clinicians in recognizing similar situations and initiating timely intervention to improve patient outcomes.

Case Presentation

Patient's history and Physical Examination

This case involves a sixteen year old female who was brought to the emergency department by ambulance after experiencing prolonged generalized seizures at home. The patient lived with her parents and two younger siblings and was a secondary school student. According to her family, she had been functioning well academically and socially prior to this event and had no recent limitations in her daily activities. The patient had a known history of epilepsy that had been diagnosed approximately three years earlier following an episode of generalized tonic clonic seizures. At that time she underwent neurological evaluation and was started on antiepileptic medication. Her seizures had been relatively well controlled over the past few years with oral levetiracetam. However, according to her mother, the patient had recently missed several doses of her medication during the previous week. The family believed this occurred because of school examinations and irregular sleep during that period. She had experienced a brief seizure episode about four months earlier but had recovered quickly and did not require hospital admission. On the day of presentation, the patient had attended school in the morning and returned home in the afternoon. Her mother reported that she complained of mild headache and appeared somewhat tired but otherwise behaved normally. She had eaten a light meal and was resting in her room when the first seizure episode occurred. According to her family, she suddenly developed generalized body stiffening followed by rhythmic jerking movements of the arms and legs. She lost consciousness and fell to the floor during the episode. The initial seizure reportedly lasted several minutes. After the episode ended, the patient did not regain full awareness and appeared confused and drowsy. Within a short period she developed another generalized seizure. Over the next twenty minutes she experienced repeated convulsive episodes without full recovery between them. Her parents became increasingly concerned and contacted emergency medical services. When paramedics arrived at the home, the patient was still having intermittent tonic clonic movements and remained unresponsive. Initial seizure control measures were administered by the emergency team, and she was transported urgently to the hospital. During transport she remained confused and poorly responsive. There was no history of recent head trauma. The family denied any recent fever, cough, or symptoms suggestive of infection. There was also no history of alcohol use, substance exposure, or ingestion of toxic substances. According to her parents, the patient had not complained of abdominal pain, nausea, or vomiting earlier in the day. Her appetite had been normal during the previous days and there had been no recent gastrointestinal complaints. Her past medical history was significant only for epilepsy. She had no history of chronic medical illnesses such as diabetes, heart disease, or chronic gastrointestinal disorders. She had never undergone any previous surgical procedures. Her immunizations were reported to be up to date according to the national vaccination schedule. There was no known family history of epilepsy or other neurological conditions. Upon arrival to the emergency department, the patient remained in an altered state of consciousness. Active seizure activity had stopped shortly

before arrival following treatment provided by the paramedics, but she remained confused and poorly responsive. She was placed on continuous monitoring while the emergency team began the initial assessment and stabilization. At the time of evaluation, the patient appeared acutely ill and was not responding appropriately to verbal commands. Her vital signs showed a temperature of 36.8 degrees Celsius, blood pressure of 112 over 70 millimeters of mercury, heart rate of 124 beats per minute, respiratory rate of 26 breaths per minute, and oxygen saturation of 97 percent while receiving supplemental oxygen through a face mask. She appeared tachycardic and mildly tachypneic but maintained stable oxygen saturation. General examination showed an adolescent girl lying on the stretcher with reduced level of consciousness. There were no visible signs of external trauma. Her skin appeared slightly sweaty and flushed. Evidence of tongue biting was noted along the lateral edge of the tongue, which was consistent with recent generalized seizure activity. No active bleeding was seen. There was no cyanosis or jaundice on general inspection. Neurological examination was limited due to her reduced responsiveness. The patient opened her eyes briefly in response to painful stimulation but did not follow verbal commands. Her pupils were equal in size and reactive to light. There was no obvious facial asymmetry. Spontaneous movement of all four limbs was observed during brief periods of restlessness. There was no clear evidence of focal neurological deficit during the initial examination. Deep tendon reflexes appeared symmetrical in the upper and lower limbs. Plantar responses could not be reliably assessed due to her agitation. Cardiovascular examination revealed sinus tachycardia with regular rhythm. Heart sounds were normal and no murmurs were appreciated. Peripheral pulses were present and symmetrical. Capillary refill time appeared normal. There was no evidence of peripheral edema. Respiratory examination showed good air entry bilaterally on auscultation. No wheezing or crackles were heard. Her breathing appeared somewhat rapid but there were no clear signs of respiratory distress. Chest expansion appeared symmetrical. Given her recent seizures, the team remained attentive to the possibility of aspiration or airway compromise. During the course of the initial assessment, the patient was noted to have progressive abdominal distension that appeared unusual for her body habitus. Her abdomen appeared visibly enlarged and tense on inspection. According to her parents, her abdomen had not appeared distended earlier in the day prior to the seizure episodes. On abdominal examination, the abdomen was markedly distended. Palpation revealed generalized abdominal tenderness, although the assessment was limited due to the patient's reduced level of consciousness and intermittent agitation. The abdominal wall felt tense and somewhat firm. There were no visible surgical scars. Guarding was suspected but difficult to clearly evaluate because of her limited cooperation during the examination. Percussion of the abdomen produced a predominantly tympanic sound across most areas. Bowel sounds were present but appeared reduced in frequency. No clear abdominal masses were palpated during the initial examination. The sudden development of abdominal distension raised concern for an acute intra abdominal process. Because of these findings, the emergency team proceeded with an urgent chest radiograph as part of the initial evaluation. The imaging demonstrated large amounts of free air beneath both hemidiaphragms, consistent with massive pneumoperitoneum (Figure 1).

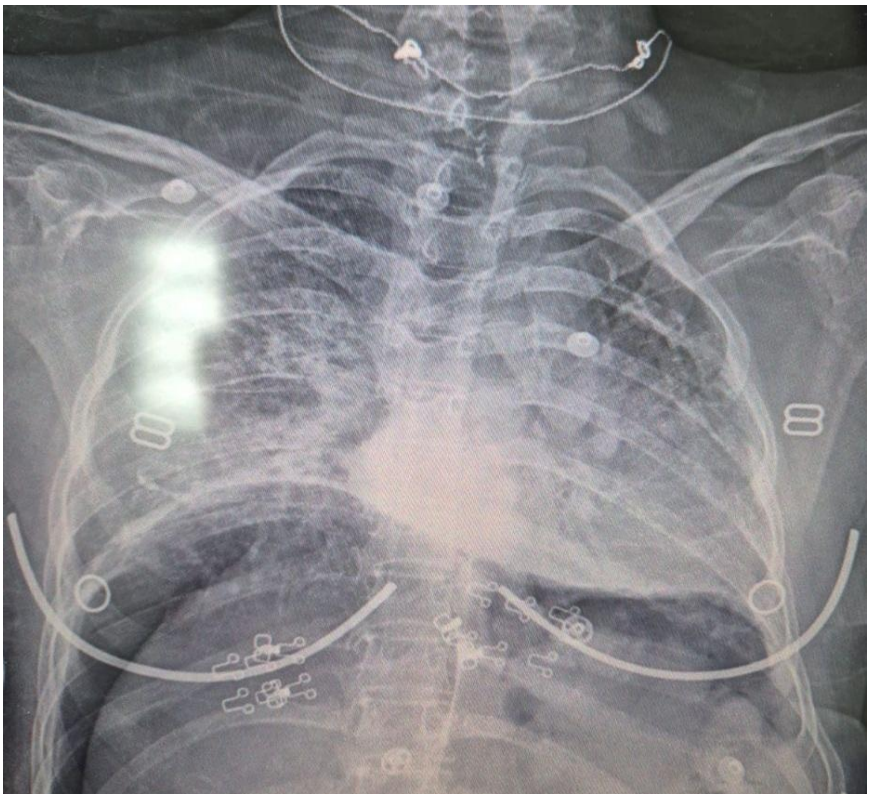

Figure 1: Chest radiograph demonstrating bilateral subdiaphragmatic free air consistent with massive pneumoperitoneum.

The presence of significant free intraperitoneal air raised immediate concern for gastrointestinal perforation, particularly in the context of acute abdominal distension. An urgent surgical consultation was requested while further stabilization and evaluation of the patient continued in the emergency department.

Diagnostic Workup:

Following the initial assessment in the emergency department, the patient remained under close monitoring while further diagnostic evaluation was initiated. At this stage, the clinical team was dealing with two major concerns. The first was the neurological emergency related to recent prolonged seizure activity and suspected status epilepticus. The second was the unexpected finding of significant abdominal distension with radiographic evidence of free air beneath the diaphragm. The presence of massive pneumoperitoneum raised strong concern for gastrointestinal perforation, which required urgent confirmation and further characterization. Initial laboratory investigations were obtained as part of the emergency evaluation. Complete blood count showed a hemoglobin level of 12.1 grams per deciliter, which was within the normal range for the patient's age and sex. The white blood cell count was elevated at 15,200 cells per microliter with a predominance of neutrophils. Platelet count was 295,000 per microliter. The elevated white cell count was considered consistent with physiological stress and possible early inflammatory response, although it was not specific for any particular diagnosis. Serum electrolytes were assessed to evaluate possible metabolic contributors to the seizure episode. Sodium level was 134 millimoles per liter, potassium was 4.1 millimoles per liter, and chloride was 102 millimoles per liter. Serum calcium and magnesium levels were also within normal range. Blood glucose measured on arrival was 118 milligrams per deciliter. These findings did not suggest a clear metabolic trigger for the seizure activity. Renal function tests showed blood urea nitrogen of 16 milligrams per deciliter and serum creatinine of 0.7 milligrams per deciliter, both within normal limits. Liver function tests were also within normal range. Serum lactate was mildly elevated at 3.4 millimoles per liter, which was attributed to recent prolonged seizure activity and increased muscular activity during the convulsive episodes. Because of the history of multiple seizures, serum creatine kinase was also measured to assess for muscle injury related to prolonged convulsions. Creatine kinase level was moderately elevated at 820 units per liter. This finding was consistent with muscle breakdown following repeated tonic clonic seizures. Arterial blood gas analysis was performed to evaluate the patient's respiratory and metabolic status following the seizure episodes. The results showed a mild metabolic acidosis with partial respiratory compensation. The pH was 7.31, bicarbonate level was 19 millimoles per liter, and partial pressure of carbon dioxide was slightly reduced. Oxygenation remained adequate while the patient was receiving supplemental oxygen. These findings were considered consistent with the metabolic stress associated with recent seizure activity. In parallel with the laboratory evaluation, imaging studies were arranged to further assess the source of the pneumoperitoneum that had been identified on the initial chest radiograph. Because massive free air beneath the diaphragm is most commonly associated with perforation of a hollow abdominal organ, the surgical team recommended urgent cross sectional imaging of the abdomen. A contrast enhanced computed tomography scan of the abdomen and pelvis was therefore performed. The CT scan confirmed the presence of large amounts of free intraperitoneal air distributed throughout the abdominal cavity. Significant air accumulation was seen anterior to the liver and beneath both hemidiaphragms. The imaging also demonstrated marked abdominal distension with displacement of several bowel loops. Further evaluation of the gastrointestinal tract on CT imaging revealed focal discontinuity along the anterior wall of the distal ileum with surrounding inflammatory changes. Small pockets of free air were seen adjacent to this region, raising strong suspicion for bowel perforation at that site. Mild thickening of the nearby bowel wall was also noted. There was a small amount of free fluid within the pelvis, but no large fluid collections or abscess formation were identified. The stomach and proximal small bowel appeared mildly distended but without clear evidence of perforation. The colon was visualized and did not demonstrate obvious masses or areas of obstruction. The liver, spleen, pancreas, kidneys, and adrenal glands appeared unremarkable on imaging. There was no evidence of intra abdominal tumors or structural abnormalities that could explain the perforation. Because of the recent seizure activity and altered level of consciousness, neuroimaging was also considered necessary to evaluate for possible intracranial causes or complications. A non contrast computed tomography scan of the brain was performed as part of the emergency workup. The scan did not demonstrate acute intracranial hemorrhage, mass lesions, or signs of cerebral edema. The ventricular system appeared normal and there were no areas of focal brain injury. These findings suggested that the seizures were likely related to her known epilepsy rather than an acute structural brain lesion. Continuous cardiac monitoring was maintained throughout the evaluation period. Electrocardiography performed in the emergency department demonstrated sinus tachycardia but no evidence of arrhythmia or ischemic changes. This was considered consistent with the physiological stress associated with recent seizures and acute illness. Given the imaging findings suggesting bowel perforation with large volume pneumoperitoneum, the surgical team was consulted urgently. The CT scan findings were reviewed jointly by the emergency physicians, radiologists, and surgeons. Based on the presence of free intraperitoneal air, focal bowel wall disruption, and the patient's worsening abdominal distension, the leading diagnosis was perforation of the small intestine with secondary pneumoperitoneum. At this stage, the patient remained hemodynamically stable but continued to demonstrate signs of abdominal distension and reduced level of consciousness following the seizure episodes. The combination of neurological instability and suspected gastrointestinal perforation represented a complex clinical situation requiring prompt multidisciplinary

management. The diagnostic workup therefore confirmed two critical conditions occurring simultaneously. The patient had experienced prolonged seizure activity consistent with status epilepticus, and imaging studies revealed massive pneumoperitoneum with evidence suggestive of small bowel perforation. These findings prompted immediate preparation for urgent surgical intervention while neurological stabilization and supportive care continued in the emergency department.

Management course

Management of the patient began immediately after the diagnostic workup confirmed two major clinical problems occurring at the same time. The first was status epilepticus related to her known history of epilepsy, and the second was massive pneumoperitoneum with imaging findings strongly suggestive of small bowel perforation. Because of the potential for rapid deterioration in both neurological and abdominal conditions, the patient was managed jointly by the emergency medicine, neurology, anesthesia, and general surgery teams. Initial management in the emergency department focused on stabilization of airway, breathing, and circulation while controlling ongoing seizure activity. The patient was placed on continuous cardiac and oxygen saturation monitoring. Intravenous access was secured through two peripheral lines to allow rapid administration of medications and fluids. Supplemental oxygen was provided through a face mask, and suction equipment was kept readily available because of the risk of aspiration following repeated seizures. To prevent recurrence of seizures, the patient received intravenous benzodiazepine followed by a loading dose of intravenous levetiracetam as recommended by the neurology team. After medication administration, no further seizure activity was observed in the emergency department. Her level of consciousness gradually improved over the following hour, although she remained drowsy and confused for some time, which was consistent with a postictal state after prolonged seizures. Because of the elevated creatine kinase and risk of dehydration following prolonged convulsive activity, intravenous fluids were started using isotonic saline to maintain adequate hydration and renal perfusion. Serial neurological observations were performed to monitor improvement in mental status and detect any recurrence of seizure activity. At the same time, the surgical team proceeded with urgent evaluation of the abdominal findings. The CT scan results indicating free intraperitoneal air with suspected distal small bowel perforation were reviewed carefully, and the decision was made to proceed with emergency surgical exploration. Given the large volume of pneumoperitoneum and the patient's worsening abdominal distension, conservative management was considered inappropriate. The patient was therefore prepared for urgent operative intervention. Prior to surgery, broad spectrum intravenous antibiotics were initiated to reduce the risk of intra abdominal infection and peritonitis. A combination of ceftriaxone and metronidazole was administered to provide coverage against both gram negative organisms and anaerobic bacteria commonly associated with gastrointestinal perforation. The patient was kept nil by mouth and a nasogastric tube was inserted to decompress the stomach and reduce further accumulation of air within the gastrointestinal tract. A urinary catheter was also placed to allow monitoring of urine output as an indicator of renal perfusion and overall fluid status during the perioperative period. After initial stabilization and preparation, the patient was transferred to the operating theater for emergency exploratory laparotomy under general anesthesia. During the procedure, a large amount of free air was released upon entering the abdominal cavity, confirming the presence of massive pneumoperitoneum. Surgical exploration revealed a small perforation located in the distal ileum with surrounding inflammation of the adjacent bowel segment. No evidence of widespread bowel ischemia or additional perforations was identified during inspection of the remaining small and large intestine. The affected segment of the ileum containing the perforation was resected, and primary end to end anastomosis was performed. The abdominal cavity was then irrigated thoroughly with warm saline to remove any contaminated fluid and reduce the risk of postoperative infection. No large abscess collections were identified during the procedure. After completion of the surgical repair, a drain was placed in the abdominal cavity for postoperative monitoring and the abdominal wall was closed in layers. The patient was then transferred to the intensive care unit for postoperative observation because of the combination of recent status epilepticus and emergency abdominal surgery. In the intensive care unit she remained on close neurological and hemodynamic monitoring. Antiepileptic therapy was continued with scheduled doses of levetiracetam to prevent further seizures. Her neurological status gradually improved over the next several hours as the postictal state resolved. Pain control was provided using intravenous analgesics while avoiding excessive sedation that might interfere with neurological assessment. Intravenous antibiotics were continued to prevent intra abdominal infection. Fluid balance was carefully monitored through regular assessment of urine output and daily laboratory tests. Over the following days, the patient's abdominal distension gradually improved and bowel function began to recover. The nasogastric tube was removed once bowel sounds returned and there were no signs of persistent ileus. Oral intake was then slowly reintroduced beginning with clear fluids and advancing as tolerated. The surgical drain showed minimal output and was removed after several days once there were no signs of ongoing intra abdominal leakage. Neurological evaluation was repeated during her recovery period, and the neurology team emphasized the importance of strict adherence to antiepileptic medication to prevent further seizure episodes. The patient and her family were counseled regarding medication compliance, seizure precautions, and the need for regular neurological follow up after discharge. By the end of her hospital stay the patient had recovered well from both the seizure episode and the surgical intervention. Her vital signs were stable, abdominal pain had significantly improved, and she was able to tolerate oral intake without difficulty. She was discharged home with scheduled follow up appointments with both the neurology and surgical teams for continued monitoring and long term management.

Discussion

Status epilepticus represents one of the most critical neurological emergencies encountered in clinical medicine and requires rapid recognition and immediate treatment to prevent permanent neurological injury and systemic complications. The present case illustrates an unusual and clinically challenging scenario in which prolonged seizure activity was accompanied by massive pneumoperitoneum due to bowel perforation. Although seizures themselves are relatively common in emergency practice, the coexistence of status epilepticus with acute abdominal catastrophe is rare and can easily lead to diagnostic delay if clinicians focus solely on neurological stabilization without considering secondary complications. Status epilepticus is defined as continuous seizure activity lasting longer than five minutes or recurrent seizures without recovery of consciousness between episodes. This modern definition reflects current understanding that seizures persisting beyond this period are unlikely to terminate spontaneously and require urgent treatment to prevent progression to refractory status epilepticus [1,2]. Epidemiological studies suggest that status epilepticus occurs in approximately 10 to 40 cases per 100,000 individuals annually, with higher incidence in pediatric and elderly populations. Mortality rates vary widely depending on the underlying cause and duration of seizures, but reports suggest that mortality can reach 15 to 20 percent in severe cases, particularly when systemic complications develop [3]. The pathophysiology of prolonged seizures involves complex neurochemical processes that lead to sustained neuronal excitation. Excessive release of excitatory neurotransmitters combined with reduced inhibitory signaling promotes continuous electrical activity in cortical networks. As seizure activity persists, metabolic demand increases significantly, leading to systemic stress responses that may affect multiple organs [1]. These physiological changes explain why prolonged seizures are frequently associated with complications such as metabolic acidosis, hyperthermia, rhabdomyolysis, and electrolyte disturbances. In addition to these well recognized systemic effects, prolonged tonic clonic activity can also produce marked increases in intrathoracic and intra abdominal pressure. Repeated forceful contraction of the diaphragm and abdominal musculature during generalized convulsions may lead to unusual mechanical complications involving the thoracic or abdominal cavities. Although rare, several reports have described the occurrence of pneumomediastinum, pneumothorax, and pneumoperitoneum following severe convulsive episodes [12]. The present case highlights how these mechanical forces may contribute to gastrointestinal injury and ultimately bowel perforation. Pneumoperitoneum is defined as the presence of free gas within the peritoneal cavity and is traditionally considered a radiological sign of hollow viscus perforation. In clinical practice, more than 90 percent of cases of pneumoperitoneum are associated with perforation of a gastrointestinal organ such as the stomach, small intestine, or colon. Because leakage of gastrointestinal contents into the peritoneal cavity can rapidly lead to peritonitis and sepsis, the detection of free intraperitoneal air typically warrants urgent surgical evaluation [11]. The diagnostic importance of pneumoperitoneum is well established in emergency medicine and surgery, and the classic radiographic finding of air beneath the diaphragm on an upright chest radiograph remains one of the most recognized signs of gastrointestinal perforation. However, not all pneumoperitoneum is caused by direct bowel rupture. A smaller proportion of patients develop what is described as spontaneous or non surgical pneumoperitoneum. In these situations, air may enter the peritoneal cavity through mechanisms unrelated to gastrointestinal perforation. Examples include barotrauma from mechanical ventilation, thoracic air leak syndromes, or dissection of air along fascial planes from the mediastinum into the retroperitoneum and peritoneal cavity [11,12]. Distinguishing between surgical and non surgical causes is therefore an important diagnostic challenge. Computed tomography has become the most sensitive imaging modality for evaluating pneumoperitoneum because it allows identification of the source of free air as well as associated signs such as bowel wall thickening, focal discontinuity, or inflammatory changes around the perforation site. In the present case, CT imaging demonstrated focal bowel wall disruption with surrounding inflammation, findings that strongly suggested true gastrointestinal perforation rather than benign pneumoperitoneum. Early CT evaluation in patients with unexplained abdominal distension or radiographic free air is therefore critical in guiding appropriate management. Another potential contributor to pneumoperitoneum in critically ill patients is pneumatosis intestinalis, which refers to the presence of gas within the wall of the gastrointestinal tract. Pneumatosis intestinalis is not a disease itself but rather a radiological finding associated with a variety of conditions ranging from benign to life threatening. It may occur in association with bowel ischemia, infection, inflammatory diseases, or increased intraluminal pressure. When the integrity of the intestinal wall is compromised, gas may escape into the peritoneal cavity and produce pneumoperitoneum [10]. The clinical significance of pneumatosis intestinalis depends on the underlying cause and the patient's overall clinical condition. In unstable patients or those with signs of peritonitis, the finding often indicates severe intestinal pathology requiring urgent surgical intervention. One of the key learning points from this case is the importance of maintaining a broad differential diagnosis when evaluating patients with prolonged seizures. Emergency clinicians naturally focus on terminating seizure activity and preventing neurological injury, which is appropriate and consistent with current treatment guidelines. Rapid administration of benzodiazepines followed by second line antiepileptic medications remains the recommended approach for early seizure control [2,4]. However, once seizures have been stabilized, careful reassessment of the patient is essential to identify complications that may have developed during the episode. Physical examination should not be limited to neurological assessment alone. In particular, unexpected findings such as abdominal distension, tenderness, or hemodynamic instability should prompt further investigation. In the present case, recognition of progressive abdominal distension during the initial examination played a critical role in identifying the underlying surgical emergency. Without careful

physical assessment, the pneumoperitoneum might have been overlooked until the patient developed more advanced signs of peritonitis or septic shock. This highlights an important clinical principle that even in the context of a clear neurological diagnosis, clinicians must remain alert to additional pathology. From a surgical perspective, bowel perforation represents a life threatening condition that requires rapid intervention. Mortality rates for untreated gastrointestinal perforation remain high because bacterial contamination of the peritoneal cavity can rapidly lead to diffuse peritonitis and systemic infection. Early surgical repair combined with broad spectrum antibiotic therapy significantly improves survival outcomes. Current surgical practice emphasizes prompt exploration once perforation is suspected, particularly in patients with imaging findings demonstrating free intraperitoneal air and focal bowel injury [12]. In the present case, emergency exploratory laparotomy allowed identification and resection of the affected bowel segment before widespread intra abdominal infection developed. The relationship between severe seizures and gastrointestinal perforation remains poorly understood because such cases are rarely reported in clinical literature. Several mechanisms have been proposed to explain how seizures might contribute to bowel injury. One possibility involves sudden and repeated increases in intra abdominal pressure during tonic contraction of abdominal muscles. These pressure changes may place mechanical stress on the bowel wall, particularly if the intestine is already weakened by inflammation or other underlying pathology. Another potential mechanism involves transient hypoperfusion of the intestinal wall during prolonged seizures. Severe systemic stress may lead to temporary reduction in splanchnic blood flow, which could theoretically contribute to localized ischemia and increased susceptibility to perforation. Although direct causal relationships are difficult to prove, the temporal association between status epilepticus and the development of pneumoperitoneum in this case suggests that seizure related physiological stress may have played a contributing role. The present case also emphasizes the importance of multidisciplinary management in complex medical emergencies. Successful outcome required coordination between emergency physicians, neurologists, radiologists, anesthesiologists, and surgeons. Early recognition of both neurological and surgical problems allowed timely treatment of each condition before irreversible complications occurred. Multidisciplinary collaboration is particularly important when patients present with overlapping medical and surgical issues that fall outside the boundaries of a single specialty. Another relevant clinical consideration involves prevention of recurrent seizures after stabilization. Poor adherence to antiepileptic medication is a well recognized risk factor for breakthrough seizures and status epilepticus. Studies have shown that medication noncompliance accounts for a substantial proportion of emergency admissions related to epilepsy. Ensuring patient education and appropriate follow up is therefore an essential component of long term management [4]. In adolescent patients, additional attention should be given to counseling regarding medication adherence, sleep patterns, and avoidance of potential seizure triggers. Finally, this case highlights the ongoing importance of detailed clinical examination in modern medical practice. Although advanced imaging techniques provide valuable diagnostic information, initial bedside observations often provide the first clue that a serious complication is developing. In the present case, the observation of rapidly increasing abdominal distension prompted further imaging that ultimately revealed massive pneumoperitoneum. Without this clinical suspicion, the underlying bowel perforation might have been recognized much later in the patient's course. Careful integration of clinical findings, laboratory data, and imaging results remains the cornerstone of accurate diagnosis in complex medical cases. In summary, this case demonstrates a rare but clinically significant association between status epilepticus and massive pneumoperitoneum due to bowel perforation. The case underscores several important lessons for clinical practice, including the need for rapid seizure control, thorough physical examination after stabilization, early imaging when unexpected findings arise, and prompt surgical intervention when gastrointestinal perforation is suspected. Recognition of such unusual complications can improve clinical awareness and help clinicians manage similar presentations more effectively in the future.

Conclusion

This case underscores the critical need for clinicians to maintain a high index of suspicion for life threatening secondary complications in patients presenting with status epilepticus. While seizure control is the immediate priority, rapidly developing abdominal distension or unexplained systemic changes should trigger prompt evaluation for rare but serious complications such as pneumoperitoneum and bowel perforation. Early bedside recognition, combined with targeted imaging, allows timely surgical intervention and prevents progression to sepsis or multi organ failure. The case also illustrates how the mechanical and metabolic stresses of prolonged seizures can precipitate uncommon gastrointestinal emergencies, reinforcing the importance of multidisciplinary care, continuous reassessment, and vigilance beyond the primary neurological presentation.

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