

Sudden Gastrointestinal Deaths of a Child due to a Fecaloma: a Multilevel Forensic Pathological Approach

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Abstract

Background. Sudden gastrointestinal deaths refer to unexpected fatalities that occur due to severe gastrointestinal conditions. They are rare, especially when compared to sudden deaths caused by cardiovascular, respiratory or neurological events. It is even more rare that a sudden death due to gastrointestinal causes occurs in a child.

Results. We presented a unique case of sudden death of a 9-year-old child with no pertinent medical history, presenting abdominal pain and diarrhea and deceased shortly after the onset of symptoms. The autopsy showed a severe intestinal obstruction due to a rectosigmoid fecaloma. In addition, the immunohistochemical evaluation revealed an inflammation of the intestine.

Conclusion. In case of sudden deaths, forensic pathological investigation is essential to determine the cause of death. Diagnosis can only be made through a rigorous and multi-level post-mortem approach in which immunohistochemical investigation can also make an important contribution. *Clin Ter 2025; 176 (3):271-277 doi: 10.7417/CT.2025.5221*

Keywords: Bowel obstruction, Autopsy, Unexpected death, Immunohistochemistry, Infant death, Natural death

Introduction

Sudden death (SD) refers to a natural death that occurs quickly, unexpectedly and only shortly after the presentation of symptoms or signs of a potentially fatal condition. (1). In particular, the World Health Organisation defines “sudden” as those non-violent, unexpected deaths that occur within 1 hour of the onset of symptoms. (2). The one-hour time interval refers to so-called “witness SD”; it can extend up to 24 hours for victims of “unwitness SD” known to be healthy or otherwise not in lethal condition 1 day before being found dead. (3).

Although the lack of a coherent definition of the SD time window limits the collection of epidemiological data and the identification of risk factors (4), the most prevalent

cause of SD is related to cardiovascular disease, followed by neurological and respiratory events. Compared to other disorders, gastrointestinal disease rarely leads to SD. This is probably because gastrointestinal disorders most often occur with typical symptoms that guarantee early health intervention. (5). Therefore, gastrointestinal disorders, even in case of a fatal outcome, often allow to define *antemortem* the cause of death. Therefore, sudden deaths caused by gastrointestinal pathologies are rare and poorly described in the pathological-forensic field. (6, 7).

Gastrointestinal causes of SD include both congenital abnormalities and acquired conditions. Some congenital causes are intestinal obstruction, Meckel’s diverticulum, or mesenteric defects. Acquired causes include intussusception, volvulus, tumors, perforation secondary to ulcers, and blood vessel pathologies (8). Intestinal (or bowel) obstruction is a partial or complete blockage of the transit of the intestinal contents; it is a medical emergency requiring prompt treatment. The causes of intestinal blockage can be functional or mechanical due to an intraluminal or extraluminal compression. In case of mechanical intestinal obstruction, the clinical presentation and evolution depend on the site of blockage. Symptoms include constipation, nausea or vomiting, abdominal pain, and abdominal distension (9,10). Abdominal distension is massive in case of large bowel obstruction, with an increased risk of parietal perforation. Morbidity and mortality due to intestinal obstruction are still high, especially in colonic occlusion and ischemia (11). Bowel obstruction in children is uncommon; this condition may be caused by helminth infection, bezoars (solid masses of indigestible material), congenital abnormal bands, intestinal malrotation, Hirschsprung disease, or Meckel’s diverticulum (12, 13).

A fecaloma is a large, hard mass of stool that remains lodged in the lumen of the last tract of the intestine (typically the colon or rectum) that cannot be evacuated spontaneously (14). It occurs when stool accumulates over time, becoming dry, hardened, and difficult to pass. Conditions leading to the formation of a fecaloma are chronic constipation, poor fiber or fluid intake, neurological dysfunction. Also, certain medications such as opiates, anticholinergics, diuretics, anti-

depressants and antipsychotics can induce gastrointestinal hypomotility and cause its formation (15 - 20)

Chronic constipation and fecal impaction are common in elderly and can be found in young patients with psychiatric and cognitive disorders. Chronic constipation and fecal impaction are common in elderly people and can be found in young patients with psychiatric and cognitive disorders. Globally, the prevalence of chronic constipation in children and adolescents is estimated at 30%, and only 5% are caused by organic diseases (such as Hirschsprung disease, cystic fibrosis, Down syndrome, neural tube abnormalities and celiac disease). Functional constipation is more difficult to diagnose not fitting into a pathological picture that itself manifests with red flag signs and symptoms. (21). It is determined by multifactorial causes, including withholding behaviour, anorectal dysfunction, diet, physical activity, genetic predisposition and psychological issues. (22).

If not timely diagnosed and treated, chronic constipation can result in the retention of faeces, formation of a fecaloma that can cause the blockage of intestinal transit, increased pressure on the intestinal wall and on adjacent viscera. In severe cases the increase of intraluminal pressure may cause intestinal ischemia, stercoral colitis, intestinal necrosis, intestinal perforation with stercoral peritonitis, and death (23-25).

Few cases of fecaloma deaths have been reported in the literature; they mostly concern psychiatric subjects under treatment with antipsychotic drugs, elderly subjects, and/or suffering from neurological diseases. (26 - 28.). To the best of our knowledge, there are no reported cases in literature of children dying from a fecaloma.

We describe a unique case of sudden death from bowel obstruction due to a rectosigmoid fecaloma, unusually observed in the pediatric population.

Materials and methods

Case presentation

A 9-year-old boy started complaining of severe abdominal pain around 10 pm. At 4:00 am, the mother called the emergency service, explaining that the child suffered from intense abdominal pain and diarrhea. She was advised by

the doctor to only hydrate the child and to wait until the morning. At about six o'clock in the morning the mother took the unconscious child to the pediatric emergency room. At the access the child, now lifeless, were unresponsive to resuscitation measures and medical professionals declared death at 06.00. Based on the report provided by the attending pediatrician, the child was born at full term of natural birth, had no pathology and was not taking any medication. No pertinent family history was also found. The mother also ruled out ingestion of foreign substances by the child who had been in close contact with her during the previous two days. In addition, the mother reported that the son had not had any symptoms on previous days. Abdominal pain and diarrhea - not accompanied by fever or other symptoms - had suddenly arisen only the night before death. The mother also denied a history of chronic constipation.

In order to clarify the cause of death, a complete autopsy was performed.

Autopsy and laboratory findings

The forensic pathologist performed an autopsy three days after death. The corpse's abdomen was distended and entirely affected by the putrefactive green stain on external examination. The body had no external injuries (Fig.1).

Upon opening the peritoneal cavity, there was a massive expansion of the intestinal loops without apparent necrosis (Fig. 2A).

Additionally, the peritoneum did not show macroscopic signs of inflammation. The intestine was isolated and opened on its anti-mesenteric side. In the lumen of the rectum-sigma, about 18 cm from the anus, there was a large hard fecal mass, measuring 7x5.5x4 cm (Fig. 3).

The fecal mass obstructed the lumen. The intestine was not perforated, although the large bowel walls were thin and dark-colored. The whole gastrointestinal system contained fecaloid material, while the esophagus was full of blackish fecal-like material (Fig. 2B). The organs did not show macroscopic alterations. The heart was regular, as were the coronaries. The toxicological tests carried out on blood and urine were negative. The histological examination of samples, stained with hematoxylin-eosin, revealed visceral stasis, massive pulmonary edema, and interfascicular

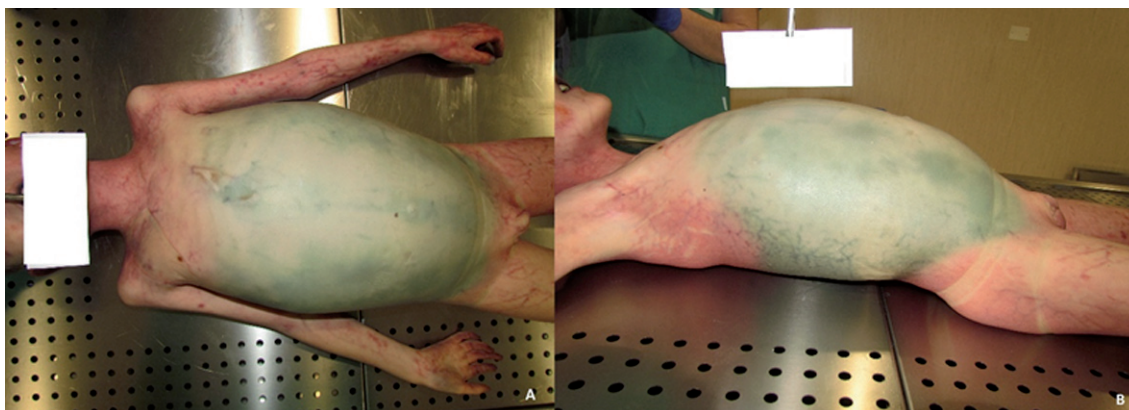


Fig. 1. External examination of the body.

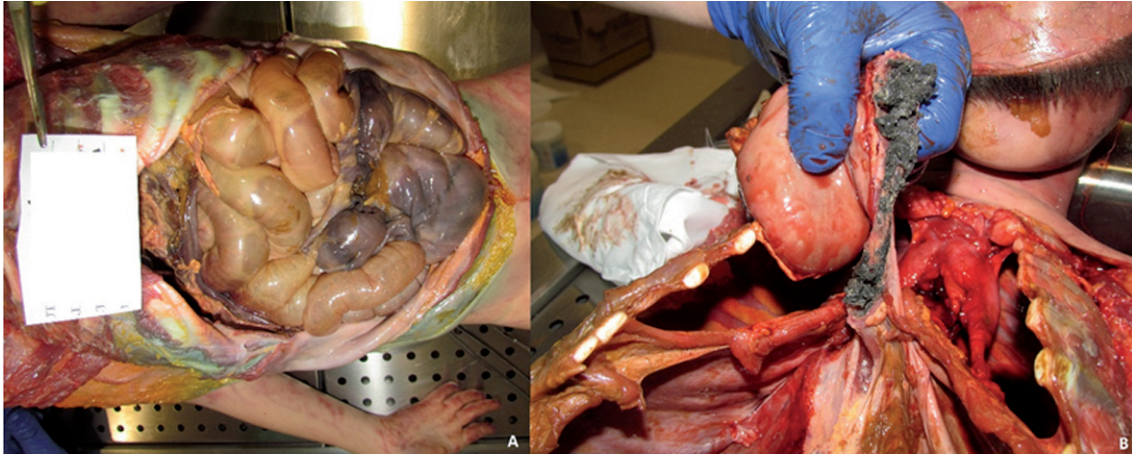


Fig. 2. A Massive expansion of the intestinal loops without apparent necrosis. B Esophagus full of blackish fecal-like material.



Fig. 3. Large hard fecal mass in rectum-sigma.

edema of the myocardium. The histological examination of the intestinal wall revealed no necrosis and the presence of ganglia. Immunohistochemical analysis was performed using antibodies, as shown in Table 1.

The intestinal wall showed intense positivity for CD68 antibodies on the mucosal side. The other markers were noticeable on the serous side. The immunohistochemical positivity thus demonstrated the presence of inflammation of the intestinal wall (Fig. 4).

The cause of the inflammation was the increase in intraluminal and colon wall pressure due to visceral distention caused by the obstructing fecaloma. Combining the data obtained, the pathologist excluded bleeding, ulceration, intestinal perforation, peritonitis, and cardiac-related death. Considering all the evidence, the death was due to hypovolemic shock. Indeed, the inflamed intestinal wall led to a hydrolytic imbalance due to the lack of reabsorption of fluids.

Table 1. Types of antibodies used in immunohistochemical staining.

CD-68: sc-66204	CD68 is a mouse monoclonal antibody raised against peripheral blood monocyte components of human origin. CD68 is a macrophages markers. Macrophages are tissue-localized, differentiated cells derived from circulating monocytes. They are phagocytic cells that engulf antibody-coated pathogens, which are subsequently degraded in intracellular vesicles.
MCP-1 (ECE.2): sc-52701	MCP-1 (ECE.2) is a rat monoclonal antibody raised against murine MCP-1 of mouse origin. MCPs are monocyte chemotactic proteins, they are produced by a variety of cells, including T lymphocytes, subsequent to their activation with cytokines. MCP-1 levels are increased during infection and inflammation.
TNF α (N-19): Sc-1350	TNF α is an affinity purified goat polyclonal antibody raised against a peptide mapping at the N-terminus of TNF α of human origin. Tumor necrosis factor α is a small cytokine that binds to the same receptors producing a vast array of effects similar. It is produced by several different cell types, such as lymphocytes, neutrophils and macrophages. TNF α can modulate many immune and inflammatory functions, also inhibit tumor growth.
COX-2 (M-19): Sc-1747	COX-2 is goat polyclonal affinity purified antibody raised against a peptide mapping at the C-terminus of COX-2 of mouse origin. COX-2 is a prostaglandin synthases that catalyze the formation of PGH ₂ from arachinoidic acid (AA). COX-2 is induced in migratory cells responding to pro-inflammatory stimuli and is considered to be an important mediator of inflammation.

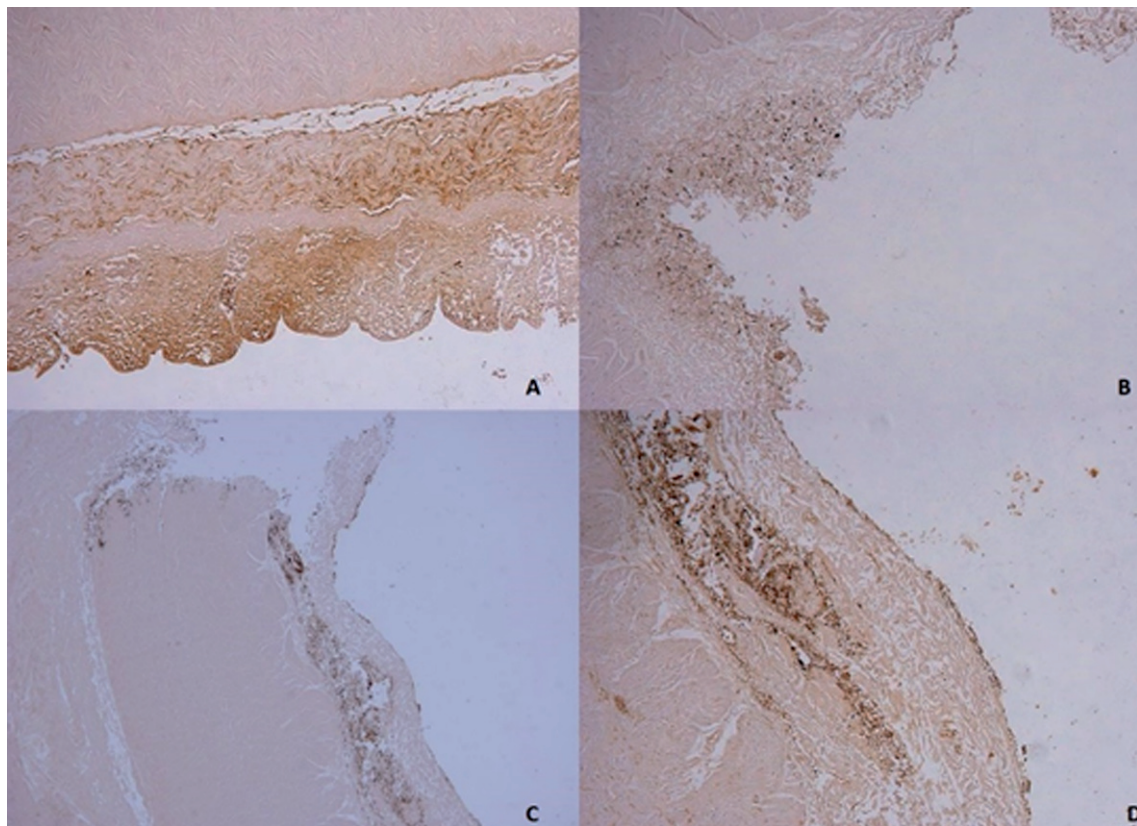


Fig. 4. Immunohistochemical positivity with inflammation of the intestinal wall.

Discussion

The pathological-forensic investigation is a crucial one to clarify the cause of sudden deaths. In such circumstances, post-mortem examination is essential not only to allow scientific progress, but also in terms of prevention for family members in detecting congenital diseases and/or malformations. In addition, the rapid, abrupt and unexpected nature of SD sometimes requires a differential diagnosis with a violent death. This can only be clarified by post-mortem investigation. Finally, SDs are often the subject of criminal

and civil proceedings for medical liability claims (29). For these reasons, the forensic pathologist is often called upon to define the cause of death in cases of SD, helping to provide epidemiological and preventive data that would otherwise be lost. The forensic pathological approach in DS cases requires an accurate collection of circumstances and medical data of the deceased. However, as in the case described, in DS the clinical history is usually negative and not decisive. The cause of death must then be determined by a comprehensive autopsy, toxicological and histological examinations.

In the case described, the autopsy showed a voluminous fecaloma in the lumen of the rectum-sigma, actually the most frequently obstructed tract by faecal material (30). There were no macroscopic or microscopic signs of necrosis or intestinal perforation; the peritoneum was free from inflammation. The absence of postmortem signs of peritoneal irritation probably explain the absence of red flag symptoms in the case described. As stated by the mother, the child complained of abdominal pain, had diarrhea, but was substantially stable and his medical condition was not life-threatening until a few minutes before arrival in the emergency room. In case of intestinal obstruction, there is an increased intestinal secretion, which can cause "paradoxical diarrhea" (14). Diarrhea could "reassure" the patient or caregiver and can lead them to rule out a severe condition. As mentioned, chronic constipation is frequent in children and is mainly functional. In such circumstances the frequency of defecation of the child should be carefully monitored to avoid the formation of fecaloma that require a medical-surgical evacuation (31, 32). In the case described, both the pediatrician and the mother denied the presence of chronic constipation and/or other previous gastrointestinal disorders.

The post-mortem histological examination of the intestinal walls showed no evidence of necrosis. In addition, examination of samples from other organs also showed nonspecific signs.

The urine and blood toxicology tests were negative for substances of abuse and psychotropic drugs. This allowed the violent cause of death by intoxication to be excluded. In SD this differential diagnosis is fundamental precisely because the sudden and unexpected character of death leaves open even hypotheses of violent death. In addition, the immunohistochemical examination of the colon wall showed intense positivity for CD68 antibodies, an histochemical marker of inflammation associated with monocyte/macrophage involvement (33).

The whole of the post-mortem investigations allowed to attribute the death to a sudden gastrointestinal deaths due to a hypovolemic shock induced by fecaloma.

The case presented is quite peculiar in the literature. In fact, sudden death in children and adolescents is often due to heart problems. Genetic analysis makes it possible to identify heart diseases in children at risk, thus avoiding death (34). Another cause of child death is sudden infant death syndrome (SIDS). SIDS is the sudden unexpected death of a healthy infant younger than 12 months, whose cause of death remains unknown and occurs during sleep (35, 36). Sudden death from a gastrointestinal condition is rare, soprattutto in bambini. In their review, Menezes et al. (8) consider hypovolemic shock and hydroelectrolytic imbalance (following bowel obstruction) as rare causes of death. Byard and Simpson (37) investigated the cases of fatal intussusceptions in children over 20 years, which were caused by many gastrointestinal conditions. Death occurs from fluid and electrolyte imbalances, peritonitis, generalized sepsis, and shock from intestinal infarction due to compromised blood supply. In a unique case in the literature, the sudden unexpected death of a 5-year-old boy was due to the forced ingestion of vinegar (38). The pathologists attributed the mechanism of death to electrolyte

imbalance with necrosis of the gastrointestinal and respiratory tract (caused by the corrosive effects of vinegar) or cardiac arrhythmia. Sato et al. (39) described a fatal case of small bowel herniation due to an undiagnosed mesenteric defect. They found a herniation of an intestinal loop in a congenital mesenteric defect, with complete obstruction of the lumen. The intestinal wall became ischemic, leading to sudden death due to septic shock.

To the best of our knowledge, in literature there are no reported infant sudden deaths due to fecaloma. In addition, the particularity of the case is related to the pathological history of the child, negative for chronic functional constipation or other predisposing conditions. In addition, the macroscopic and microscopic post-mortem examination allowed to exclude organic pathologies or gastrointestinal malformations.

We believe that the post-mortem investigation was crucial to shed light on an apparently unexplained death of a healthy child. Our study also showed that immunohistochemistry can be a valuable tool for forensic purposes, especially if the autopsy does not show any relevant macroscopic and histological alterations (e.g.: ischemia, necrosis). Indeed, immunohistochemistry can reveal the organic results underlying the metabolic disease causing death (40-44). In our case, the immunohistochemical signs of inflammation of the colon have allowed us to trace the final cause of the decess to an electrolyte imbalance induced by intestinal inflammation.

Conclusion

In the case of sudden death, post-mortem examination is the only scientific means to determine the cause of death. A multi-level pathological-forensic approach (reconstruction of the clinical history of the deceased, external examination, autopsy, toxicological examination and immunohistochemical investigation) is essential to allow a differential diagnosis between natural and violent death and to arrive at the definition of the cause of death. In particular, immunohistochemistry can be useful in cases of sudden death where the absence of clinical-anamnestic information hinders the determination of the cause of death. In fact, immunohistochemistry can highlight alterations at the cellular and molecular level that are not visible through conventional macroscopic or histological observation.

In our case, immunohistochemistry identified the presence of inflammation in the intestinal wall, thus excluding other possible causes of death and directing the diagnosis towards hypovolemic shock secondary to hydro-electrolyte imbalance. Immunohistochemistry, autoptic and microscopic results as well as toxicological tests, has allowed a diagnosis of a infant SD due to fecaloma which, to the best of our knowledge, is unique in the scientific literature.

Declaration of conflicting interests

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