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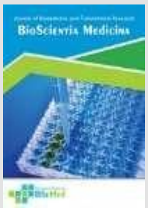

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High-Dose Sucralfate as a Mucosal-Protective Cornerstone in the Organ-Sparing Management of Pediatric Grade 2A Corrosive Gastroduodenitis: A Case Report and Pathophysiological Review

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ABSTRACT

Background: Accidental corrosive ingestion is a formidable pediatric emergency that can lead to severe gastrointestinal injury and long-term sequelae. Sulfuric acid, a common agent, induces coagulative necrosis, primarily affecting the stomach. The optimal management for moderate-grade injuries (Zargar Grade 2A) is debated, with a focus on preventing stricture formation. This report details a case managed with an aggressive pharmacotherapeutic protocol centered on high-dose sucralfate. **Case presentation:** A 2-year-10-month-old boy was admitted following accidental ingestion of battery acid. His presentation was atypical, with vomiting but no oropharyngeal lesions. Initial investigations revealed a significant systemic inflammatory response (leukocytosis: 19,220/mm^3; thrombocytosis: 581,000/mm^3) and aspiration pneumonitis. Despite a 12-day delay in endoscopy due to parental refusal, an aggressive conservative regimen was initiated upon admission. This protocol included high-dose, frequent-interval sucralfate (80 mg/kg every 2 hours), intensive intravenous acid suppression (omeprazole and ranitidine), and prophylactic antibiotics. The endoscopy on day 12 confirmed Zargar Grade 2A burns in the gastric fundus, pylorus, and proximal duodenum, with the esophagus spared. The patient improved rapidly, tolerated an oral diet by day 11, and was discharged on day 14. **Conclusion:** Follow-up endoscopy at 6 weeks and 6 months confirmed complete mucosal healing without any evidence of stricture or gastric outlet obstruction. This case suggests that an immediate, aggressive, non-surgical protocol featuring high-dose sucralfate can be effective in managing pediatric Grade 2A corrosive gastroduodenal burns, promoting complete healing and preventing long-term complications. The findings underscore the potential of this pharmacotherapeutic strategy and warrant further investigation.

1. Introduction

Accidental ingestion of corrosive substances represents a persistent and devastating challenge in pediatric emergency medicine and critical care.[1] Globally, it is estimated that children under the age of six account for up to 80% of all corrosive ingestion incidents. This alarming statistic is disproportionately concentrated in low- and middle-income countries, where a confluence of socioeconomic factors creates a high-risk environment.[2] A particularly hazardous and common practice in these settings is the storage of potent industrial and household chemicals—such as drain cleaners containing sodium hydroxide, industrial solvents, and battery acid—in unlabeled, repurposed beverage or food containers.[3] These innocuous-looking bottles become tragic traps, easily accessible to unsuspecting and curious toddlers, leading to catastrophic internal injuries. The prevention of these incidents through public health campaigns and safe storage practices remains a paramount, yet challenging, global health goal. In their absence, clinicians are faced with the complex task of managing the severe and often life-altering consequences.[4]

The pathophysiology of corrosive injury is dictated by the chemical nature of the ingested agent, with acids and alkalis producing distinct patterns of tissue destruction.[5] Alkaline agents, with a high pH, cause liquefactive necrosis. This process involves saponification of fats and solubilization of proteins, leading to deep, penetrating injuries that readily perforate the full thickness of the esophageal wall. In contrast, acidic agents such as sulfuric acid (H_2SO_4), the primary component of battery acid, induce coagulative necrosis upon contact with mucosal tissue. Sulfuric acid is a powerful oxidizing and dehydrating agent that rapidly denatures cellular and extracellular proteins, causing them to precipitate into a firm, adherent eschar.[6] This eschar, while itself a product of profound tissue damage, can paradoxically limit the depth of acid penetration, often sparing the deeper muscular layers of the esophagus. However, the journey of the acid through the gastrointestinal tract dictates the ultimate pattern of injury. Owing to its high specific gravity and the rapid esophageal transit time, the esophagus is often relatively spared. The acid then pools in the dependent portions of the stomach, primarily the antrum and pylorus. This prolonged contact time, combined with the acid overwhelming the stomach's natural protective mechanisms and inducing pylorospasm, traps the corrosive agent and leads to severe, often circumferential, gastric injury. Consequently, gastric outlet obstruction is a more frequent and dreaded complication of acid ingestion than esophageal strictures.[7]

The severity of mucosal damage is systematically graded using the Zargar classification, an endoscopic scoring system that is the global standard for prognosis and management guidance. Grade 0 indicates a normal examination, while Grade 1 shows mucosal edema and hyperemia. Grade 2 injuries are subdivided: Grade 2A, the focus of this report, is defined by superficial ulcerations, friability, hemorrhages, and exudates without circumferential involvement; Grade 2B involves deep or circumferential ulceration. Grade 3 injuries involve focal (3A) or extensive (3B) necrosis. While Grade 2A injuries carry a lower risk of perforation and stricture formation (estimated at 0-10%) compared to more severe grades, the potential for long-term morbidity is not negligible, and the optimal management strategy remains an area of active clinical investigation and debate. Preventing the progression of inflammation and fibrosis that culminates in a luminal stricture is the primary therapeutic goal.[8] A non-functional, strictured gastric outlet necessitates multiple high-risk endoscopic dilatations or surgical bypass, imposing a significant physical, emotional, and economic burden on the child and their family. Therefore, an initial investment in an aggressive and effective medical therapy that can prevent these outcomes is of profound clinical and economic importance.

Traditional management has centered on supportive care (fluid resuscitation, nutritional support) and acid suppression with proton pump inhibitors (PPIs). The role of adjuvant therapies remains controversial. Corticosteroids have been largely abandoned for acid-induced injuries due to a lack of proven efficacy in preventing strictures and a concerning potential to increase infection risk and mask signs of perforation. Prophylactic antibiotics are typically reserved for cases with suspected perforation or co-existing infection.[9] In recent years, significant attention has shifted towards the therapeutic potential of cytoprotective agents, with sucralfate emerging as a leading candidate. Sucralfate, a complex of sucrose octasulfate and polyaluminum hydroxide, acts locally to form a protective barrier over ulcerated tissue, stimulate endogenous prostaglandins and growth factors, and enhance mucosal blood flow—all critical processes for healing. While standard-dose sucralfate is widely used, emerging evidence suggests that high-dose, frequent-interval regimens may offer superior mucosal protection and significantly reduce stricture rates in severe corrosive injuries. However, robust clinical data, particularly for pediatric Grade 2A gastric and duodenal burns caused by sulfuric acid, are scarce.[10]

The novelty of this report lies in its detailed, day-by-day description of an aggressive, early-initiated conservative protocol centered on high-dose, frequent-interval sucralfate therapy in a young child. The aim of this case report is to provide a comprehensive account of the clinical, laboratory, and nutritional progression of a toddler with Zargar Grade 2A gastric and duodenal burns, and to explore the deep pathophysiological rationale for the intensive pharmacological regimen employed. This case is particularly instructive as successful management was achieved despite a significant delay in endoscopic assessment, a challenging scenario commonly encountered in clinical practice. We propose that this intensive, multi-modal pharmacological approach represents a viable and highly effective primary management strategy that can promote complete mucosal healing and prevent both acute and long-term complications in this specific patient population.

2. Case Presentation

A 2-year-10-month-old, previously healthy Indonesian boy (Weight: 13 kg, WHO Weight-for-Age Z-score: -1 SD) was brought to the pediatric emergency department of Dr. M. Djamil General Hospital, a tertiary referral center in Padang, Indonesia. He presented approximately 90 minutes after the accidental ingestion of an unknown quantity of battery acid. According to the parents' frantic account, the acid was stored in a discarded plastic mineral water bottle and left on the floor of the family's home workshop. The father was alerted by the child's sudden cry and found him with the open bottle. In a state of panic, the parents attempted inappropriate home remedies, including inducing vomiting and administering sweetened condensed milk. Subsequently, the child experienced five episodes of non-bloody, non-bilious vomiting that had a distinct, acrid chemical odor (Table 1a).

Upon arrival, the patient was awake, alert, irritable, and crying but was consolable in his mother's arms. His vital signs were stable for his age: heart rate: 110 beats per minute; respiratory rate: 25 breaths per minute; blood pressure: 90/50 mmhg; oxygen saturation: 99% on room air; temperature: 36.8°c (afebrile). The physical examination was most notable for its lack of overt findings, a deceptively benign presentation that masked the severity of the internal injury. There was no evidence of oropharyngeal burns, erythema, edema, or ulceration on his lips, tongue, or buccal mucosa. His airway was patent, with no stridor, hoarseness, or drooling to suggest supraglottic or laryngeal involvement. The neck was supple. The chest examination revealed clear, equal breath sounds bilaterally on auscultation. His abdomen was soft, non-distended, and non-tender to superficial or deep palpation, with normoactive bowel sounds present in all four quadrants.

The clinical team, maintaining a high index of suspicion based on the history alone, immediately placed the patient on nil per os (NPO) status. Two large-bore (22-gauge) peripheral intravenous lines were established, and aggressive fluid resuscitation was initiated with Kaen 1B solution at a maintenance rate of 1,150 mL/day (approximately 88 mL/kg/day) to maintain hydration and renal perfusion. A size 8 French nasogastric tube (NGT) was carefully inserted by an experienced pediatric resident to a depth of 25 cm. The tube was lubricated generously and passed gently to minimize the risk of iatrogenic injury to potentially damaged esophageal mucosa. Gastric contents were aspirated for decompression, and tube placement was confirmed by auscultation over the stomach during air insufflation. This NGT would serve for gastric decompression and, critically, as the route for administration of medication.

Table 1a. Patient Clinical Findings

Summary of Demographics, History, and Physical Examination on Admission

Patient Demographics	
Age	2 years, 10 months
Sex	Male
Weight	13 kg (WHO WAZ: -1 SD)
History of Present Illness	
Chief Complaint	Accidental ingestion of unknown quantity of battery acid.
Time Since Ingestion	~90 minutes prior to arrival.
Presenting Symptoms	Five episodes of non-bloody, non-bilious vomiting with a distinct chemical odor. Irritable and crying.
Vital Signs on Admission	
Heart Rate	110 beats/min
Respiratory Rate	25 breaths/min
Blood Pressure	90/50 mmHg
Oxygen Saturation	99% on room air
Temperature	36.8°C (Afebrile)
Physical Examination Findings	
General	Alert, irritable but consolable. No acute distress.
Oropharynx	**Absence** of burns, erythema, or ulcerations. Moist mucosa.
Respiratory	Clear, equal bilateral breath sounds. No stridor or hoarseness.
Abdomen	Soft, non-distended, non-tender. Normoactive bowel sounds.

Initial laboratory investigations were sent to assess for systemic inflammation, end-organ damage, and metabolic derangements (Table 1b). Table 1b presents the ancillary data, which unmasks the true severity of the patient's condition. The laboratory results reveal a potent systemic inflammatory response syndrome (SIRS). The marked leukocytosis (19,220/mm^3), profound reactive thrombocytosis (581,000/mm^3), and massively elevated C-Reactive Protein (48.5 mg/L) are objective biochemical evidence of massive tissue injury and necrosis. These are not localized findings; they represent a body-wide inflammatory cascade triggered by the corrosive insult. The elevated chloride level further suggests an evolving metabolic acidosis, a common consequence of systemic shock and tissue damage. The radiological findings add another crucial dimension. The diagnosis of bilateral aspiration pneumonitis confirms that the corrosive gastric contents were aspirated into the lungs, contributing significantly to the systemic inflammation and adding a major pulmonary complication to the primary gastrointestinal injury. Critically, the absence of free air on the radiograph rules out an initial perforation, confirming that a non-surgical, conservative approach is a viable initial strategy.

Table 1b. Laboratory & Radiological Findings

Summary of Ancillary Investigations on Admission

Key Laboratory Results

Parameter	Result	Reference Range
WBC Count	**19,220 /mm³**	5,000-15,000
Platelet Count	**581,000 /mm³**	150,000-450,000
C-Reactive Protein (CRP)	**48.5 mg/L**	< 5.0
Chloride	**117 mmol/L**	95-108

Radiological Findings

Modality	Chest Radiograph (AP, Upright)
Impression	• Bilateral aspiration pneumonitis. • No radiographic evidence of esophageal or gastric perforation.

The patient was admitted to the pediatric ward for intensive monitoring and management. The medical team engaged in extensive counseling with the parents, explaining the gravity of the potential internal injuries and the critical need for an early upper gastrointestinal endoscopy to guide further management. However, the parents, overwhelmed with fear and anxiety, initially refused to provide consent for the procedure, citing concerns about its invasiveness and potential complications. While respecting their autonomy, the clinical team continued daily discussions and decided to proceed immediately with a maximalist, aggressive medical management protocol in the interim, aiming to mitigate damage and promote healing while awaiting consent. A detailed day-by-day summary of the hospital course is provided in Table 2.

Table 2. Hospital Course and Daily Progress

A Longitudinal Summary of Clinical, Laboratory, and Therapeutic Progression

Day	Clinical Status & Events	WBC ($x10^3/mm^3$)	Platelets ($x10^3/mm^3$)	CRP (mg/L)	Key Interventions & Nursing Notes
1	Admitted. Alert but irritable. NPO. Vomiting ceased. Stable hemodynamics.	19.2	581	48.5	IV fluids, IV Omeprazole, IV Ranitidine, IV Ampicillin/Gentamicin. High-dose sucralfate via NGT q2h. NGT on free drainage.
2	Afebrile. Less irritable. Abdomen remains soft. Bowel sounds present.	17.5	610	62.1	Continued all medications. Strict NPO. Monitoring urine output (2 mL/kg/hr).
3	Clinically stable. Enteral nutrition initiated via NGT (Peptamen Junior).	15.8	605	55.4	Cautious initiation of enteral feeds at 10 mL/hr. Feeds held 30 mins pre/post sucralfate.
4	Tolerating feeds well. No vomiting or distension. Passed first stool.	14.2	570	41.2	Advanced enteral feeds to 20 mL/hr. Child more interactive.
5	Continued clinical improvement. Passing flatus. Showing interest in toys.	12.9	545	30.8	Advanced enteral feeds to goal rate of 35 mL/hr.
6	**Fever spike to 38.8°C. No new localizing signs. Blood cultures obtained.**	16.5	590	58.9	**Antibiotics broadened to IV Cefotaxime. Tepid sponge bath administered.**
7	Fever resolved. Clinically well.	14.1	560	35.7	Continued Cefotaxime. Blood cultures reported no growth.
8	Afebrile. Tolerating full enteral feeds. Bowel movements regular.	12.0	510	22.4	Continued all medications. Discussed plan for oral intake with parents.
9	Well-appearing. Showing interest in oral intake.	10.5	480	15.1	Parents consented to endoscopy, scheduled for Day 12.
10	Feeds transitioned to full-strength formula.	9.8	440	8.9	Sucralfate frequency decreased to q4h.
11	**NGT removed. Trial of oral fluids and soft diet well-tolerated.**	8.5	415	< 5.0	**IV antibiotics D/C'd. Switched to oral Omeprazole. Ranitidine D/C'd. Sucralfate q6h.**
12	Upper GI Endoscopy performed. Tolerated well. Resumed oral diet post-op.	-	-	-	Continued oral diet and medications.
13	Eating and drinking normally. No complaints. Playful.	-	-	-	Discharge planning initiated.
14	**Discharged home with extensive counseling and prescriptions.**	-	-	-	**Discharged with follow-up appointment for 6 weeks.**

Following the child's marked clinical improvement and extensive counseling, the parents provided informed consent for an upper gastrointestinal endoscopy, which was performed on hospital day 12 under general anesthesia. Table 3 provides the definitive anatomical diagnosis of the patient's internal injuries and is a cornerstone for understanding the clinical picture. The report from the upper GI endoscopy on Day 12 offers a detailed map of the damage wrought by the sulfuric acid, with findings that are both classic for this type of injury and prognostically significant. First, the complete sparing of the esophagus is a critical finding. This confirms the pathophysiological principle that high-density acids transit rapidly through the esophagus, often causing minimal damage before pooling in the stomach. This immediately rules out the risk of a long-term esophageal stricture, a major source of morbidity in other types of corrosive ingestions. The distribution of the injury within the stomach and duodenum is highly informative. The damage is concentrated in the dependent portions of the stomach—the fundus and, most severely, the antrum and pylorus—and extends into the proximal duodenum. This pattern perfectly illustrates the path the acid took, pooling and causing maximum damage where contact time was longest. The extensive inflammation and ulceration in the pre-pyloric antrum and duodenal bulb highlight the area of greatest concern for future complications, namely gastric outlet obstruction.

Crucially, the injury is classified as Zargar Grade 2A. This is the most important prognostic detail in the report. Grade 2A signifies superficial ulceration, friability, and exudates, but it is explicitly non-circumferential. This distinction is vital because circumferential burns (Grade 2B) have a significantly higher risk of healing with fibrotic scarring that leads to luminal strictures. The finding that the pylorus, while edematous, remained patent for the endoscope is another favorable sign. In essence, the endoscopy confirms a severe, widespread chemical burn but one that, anatomically, has a good potential for healing without the most dreaded long-term complication of obstruction. This report provides the essential baseline against which the success of the therapy and the completeness of healing on follow-up endoscopy can be accurately measured.

Table 3. Formal Endoscopy Report

Diagnostic Upper GI Endoscopy on Hospital Day 12

Procedural Details	
Date of Procedure	Hospital Day 12
Instrument	Olympus GIF-XP190N Pediatric Video Gastroscope
Anesthesia	General Anesthesia (Midazolam, Fentanyl, Propofol)
Anatomical Findings	
Esophagus	Completely normal from UES to GE junction. Smooth, pink mucosa with a normal vascular pattern. No evidence of erythema, ulceration, or exudate.
Stomach	• **Fundus:** Multiple, discrete, linear ulcers on the greater curvature, covered with grayish-white exudate. Evidence of contact bleeding (friability). • **Body:** Largely spared, with minimal scattered petechiae. • **Antrum & Pylorus:** Extensive, non-circumferential patches of superficial ulceration and severe mucosal inflammation. Pyloric ring was edematous but patent, allowing passage of the endoscope.
Duodenum	• **Bulb (D1):** Changes similar to the gastric antrum, with patches of exudate and friable, ulcerated mucosa. • **Second Part (D2):** Injury extended into the proximal second portion. Mucosa appeared hemorrhagic and ulcerated. • **Distal Duodenum:** Normal.
Conclusion	
Final Diagnosis	**Corrosive Injury, Zargar Classification Grade 2A** Affecting the gastric fundus, gastric antrum/pylorus, and the proximal duodenum (D1/D2). The esophagus is spared.

Table 4 outlines the aggressive, multi-modal, and evidence-based therapeutic strategy employed to manage this patient's severe corrosive injury. This protocol was not a single intervention but a carefully orchestrated symphony of treatments designed to counter the chemical assault from multiple angles simultaneously, creating an optimal environment for healing and organ preservation. The clear cornerstone of the therapy was the use of high-dose, frequent-interval sucralfate. The regimen of 1 gram every two hours is exceptionally aggressive and highlights a proactive strategy focused on direct mucosal defense. The rationale is twofold: first, to provide a continuous, physical "shield" over the raw, ulcerated surfaces of the stomach and duodenum, protecting them from the digestive insults of acid and pepsin; and second, to actively stimulate the body's own healing mechanisms by concentrating growth factors and prostaglandins at the site of injury. This dual-action approach—both passive barrier and active healing agent—was the central pillar of the management plan.

This mucosal protection was supported by intensive acid suppression using a dual-agent approach. The combination of an IV proton pump inhibitor (Omeprazole) and an H2-receptor antagonist (Ranitidine) reflects a sophisticated understanding of pharmacodynamics. The Ranitidine provides a rapid onset of action to immediately raise gastric pH, while the more potent Omeprazole provides profound, long-lasting suppression. This "shock and awe" tactic ensures that the corrosive environment is neutralized as quickly and completely as possible, preventing further damage and optimizing the conditions for sucralfate to polymerize and adhere to the ulcer craters.

The management of antibiotic therapy demonstrates a responsive and vigilant clinical approach. The initial regimen of Ampicillin and Gentamicin was appropriately chosen for prophylactic coverage against aspiration pneumonitis. The decision to broaden coverage to Cefotaxime following the fever spike on Day 6, while ultimately perhaps unnecessary given the negative blood cultures, was a prudent and safe decision in the face of potential sepsis. Finally, the explicit exclusion of corticosteroids is a critical detail that underscores the protocol's adherence to modern, evidence-based practice, which has shown them to be ineffective and potentially harmful in cases of acid ingestion. Collectively, these interventions represent a holistic strategy that combines direct mucosal defense, environmental control, infection prevention, and supportive care to maximize the potential for a successful outcome.

Table 5 provides the ultimate validation of the therapeutic strategy, documenting the patient's complete and robust recovery over a six-month period. This longitudinal data is essential, as the true success of corrosive injury management is not just surviving the acute phase, but avoiding the devastating long-term sequelae. The 6-Week Follow-up marks the first critical checkpoint and reveals an outstanding outcome. Clinically, the patient was thriving, asymptomatic, and gaining weight, indicating that the gastrointestinal tract was functioning normally. However, the most definitive evidence comes from the follow-up endoscopy. The findings of "complete mucosal healing" are profound. This means the extensive, raw ulcerations seen initially had been entirely replaced by healthy, intact epithelial tissue. The notation of only "faint, flat scarring" is significant; it indicates that the healing process occurred without the excessive deposition of collagen and fibrotic tissue that leads to problems. The most crucial finding is the "widely patent pylorus" with "no evidence of stricture formation." This confirms that the primary therapeutic goal—preventing the development of gastric outlet obstruction—was successfully achieved. The 6-Month Follow-up serves to confirm the durability of this excellent outcome. The patient's continued asymptomatic status and normal growth, months after all medications were discontinued, demonstrate that the healing was stable and self-sustaining.

Table 4. Therapeutic Interventions

A Detailed Summary of the Multi-Modal Management Protocol

Intervention	Clinical Rationale
Mucosal Protection	
High-Dose Sucralfate **Dose:** 1 gram (approx. 80 mg/kg) **Route:** NGT, then Oral **Timeline:** q2h for 10 days, then tapered. Continued for 6 weeks post-discharge.	**Clinical Rationale:** To form a viscous, adherent barrier over ulcerated tissue, protecting it from acid and pepsin. Stimulates endogenous prostaglandins and growth factors to accelerate healing. This was the cornerstone of therapy.
Intensive Acid Suppression	
Omeprazole (PPI) **Dose:** 1 mg/kg/dose **Route:** IV, then Oral **Timeline:** IV for 11 days, then oral until 6 weeks post-discharge.	**Clinical Rationale:** Potent, long-acting inhibition of the H+/K+ ATPase pump to profoundly reduce gastric acid secretion and create an optimal environment for healing.
Ranitidine (H_2RA) **Dose:** 1 mg/kg/dose q12h **Route:** IV **Timeline:** Days 1-11	**Clinical Rationale:** Provides rapid onset of acid suppression to complement the slower-acting PPI, ensuring immediate elevation of gastric pH in the critical initial phase.
Antibiotic Therapy	
Initial & Modified Regimens **Initial:** Ampicillin & Gentamicin **Modified:** Cefotaxime **Timeline:** Days 1-11	**Clinical Rationale:** Prophylactic coverage for aspiration pneumonitis and to prevent secondary bacterial infection of necrotic tissue. Broadened in response to fever spike on Day 6.
Supportive Care	
Nutritional Support **Timeline:** NPO for 2 days, followed by cautious, stepwise introduction of enteral and then oral nutrition.	**Clinical Rationale:** To support metabolic demands and promote healing while minimizing stress on the injured gut.
Excluded Therapies	
Corticosteroids	**Clinical Rationale:** Not administered, in line with current evidence showing no benefit for acid ingestions and potential for harm (increased infection risk, masking perforation).

The fact that no further investigations were needed underscores the clinical team's confidence in the complete resolution of the injury. Finally, the declaration of "Complete Recovery" is the definitive conclusion. This is not merely a statement about the absence of symptoms; it is a scientific assessment that the patient suffered no lasting anatomical or functional consequences from a severe, life-threatening chemical burn. The organ was preserved, and the long-term prognosis is excellent, effectively returning the child to his pre-injury state of health.

Table 5. Follow-up and Long-Term Outcomes

Assessment of Recovery and Final Disposition

6-Week Follow-up	
Clinical Status	Patient was thriving. Remained completely asymptomatic with no complaints of dysphagia, regurgitation, vomiting, or abdominal pain.
Nutritional Status	Tolerating a normal, age-appropriate diet without issue. Appropriate interval weight gain of **0.8 kg** noted.
Follow-up Endoscopy	**Findings:** • **Complete mucosal healing in the stomach and duodenum.** • **Previous ulcers and inflammation had fully resolved.** • **Only faint, flat scarring remained in the antrum.** • **Crucially, no evidence of stricture formation; pylorus was widely patent.**
6-Month Follow-up	
Clinical Status	Patient remained completely asymptomatic with appropriate growth and development tracking along his expected curve.
Medication Status	All medications (Omeprazole, Sucralfate) had been successfully discontinued after the 6-week mark.
Further Investigations	Given the excellent endoscopic result at 6 weeks and the complete lack of symptoms, no further imaging or endoscopy was deemed necessary.
Final Outcome	
Disposition	**Complete Recovery** The patient achieved full clinical and anatomical recovery with no long-term sequelae, such as gastric outlet obstruction or chronic dyspepsia.

3. Discussion

This case report presents a compelling example of successful non-surgical management of significant Zargar Grade 2A corrosive gastroduodenitis in a toddler. The complete clinical and endoscopic resolution, particularly the absence of stricture formation, highlights the potential efficacy of an immediate and aggressive pharmacological protocol.[11] This discussion will focus on the deep pathophysiology of the injury and the multimodal pharmacodynamic rationale for the therapeutic interventions employed.

The pathophysiology of acid-induced injury is fundamentally distinct from that caused by alkali. Sulfuric acid (H_2SO_4) is a potent oxidizing and dehydrating agent that inflicts damage through coagulative necrosis.[12] Upon mucosal contact, the acid's hydrogen ions rapidly denature intracellular and extracellular proteins, causing them to precipitate and cross-link. This process instantly kills the affected cells and creates a firm, leathery, adherent eschar on the tissue surface.[13] At a cellular level, the acid causes immediate disruption of cell membranes, inactivation of cytoplasmic and mitochondrial enzymes, and cessation of all metabolic activity. This eschar, a tomb of necrotic tissue, acts as a physical barrier that can limit the depth of further acid penetration, a key feature distinguishing it from the saponifying, deeply penetrating liquefactive necrosis seen with alkaline agents.[14]

However, the anatomical journey of the ingested acid dictates the ultimate landscape of injury. The esophagus, with its protective stratified squamous epithelium and rapid peristaltic transit, often escapes severe damage, as was observed in this case.[15] Upon reaching the stomach, the acid's high specific gravity causes it to flow along the lesser curvature and pool in the most dependent parts of the stomach—the greater curvature, antrum, and pylorus. Here, two factors amplify the injury.[16] First, the stomach's protective mucus-bicarbonate layer is rapidly overwhelmed and denuded. Second, the acid often induces intense pylorospasm, effectively trapping the corrosive agent in the antrum for a prolonged period. This sustained contact allows for extensive and severe mucosal damage, creating a major risk factor for inflammation, fibrosis, and the subsequent development of gastric outlet obstruction. The injury pattern in this patient—sparing the esophagus but severely affecting the fundus, antrum, pylorus, and proximal duodenum—is a classic signature of a significant acid ingestion.[17]

Figure 1. Pathophysiology of sulfuric acid-induced coagulative necrosis.

The management strategy was designed to aggressively counter the pathophysiological insults from the moment of admission. A central component of this successful management was the early and aggressive use of high-dose, frequent-interval sucralfate. The regimen employed—1 gram (approx. 80 mg/kg) every 2 hours—represents a significant departure from standard peptic ulcer dosing. This intensive approach is designed to maintain a constant, durable, protective coating over the vast area of denuded and ulcerated mucosa, and its rationale is rooted in sucralfate's multiple, synergistic mechanisms of action at the molecular and cellular levels.

In an acidic environment with a pH below 4, sucralfate undergoes extensive polymerization and cross-linking. The sucrose octasulfate anion dissociates, leaving a highly polar polyanion that binds electrostatically with positively charged proteins like albumin and fibrinogen that are exposed in the ulcer crater. This creates a viscous, sticky, paste-like complex that selectively and strongly adheres to the damaged tissue, forming a physical "mucosal bandage." This barrier shields the underlying regenerating cells from further chemical insult by luminal acid, pepsin, and refluxed bile salts, creating a stable microenvironment conducive to healing. The frequent two-hourly dosing is critical to maintain the integrity of this barrier, as it can be mechanically sloughed off by gastric peristalsis. Furthermore, sucralfate is not merely a passive barrier; it is an active biopharmaceutical agent. It directly stimulates the gastric mucosa to increase the synthesis and secretion of prostaglandins, particularly PGE_2 and prostacyclin (PGI_2). These eicosanoids are powerful local hormones that exert profound cytoprotective effects by stimulating the secretion of protective mucus and bicarbonate from epithelial cells. Most importantly, they are potent vasodilators that significantly enhance mucosal blood flow. This hyperemia is critical for delivering oxygen, nutrients, and systemic growth factors necessary for cellular regeneration, while simultaneously removing toxic metabolic byproducts from the site of injury. Perhaps most critically, the healing of deep ulcers is a complex process orchestrated by peptide growth factors such as epidermal growth factor (EGF) and basic fibroblast growth factor (bFGF). Sucralfate has been shown to bind these crucial growth factors within its polymeric matrix, protecting them from luminal degradation and concentrating them at the site of injury. This action effectively creates a high-potency, localized growth factor delivery system, directly accelerating the processes of re-epithelialization and angiogenesis, which are essential for rebuilding the damaged tissue.

The dosing regimen used in this case is at the high end of the protocols described in the literature. Some studies on severe esophageal burns used 1g every 4 hours, which was shown to reduce stricture rates. The more aggressive two-hourly regimen in our patient was chosen to maximize barrier maintenance in the dynamic gastric environment. While effective, such high doses necessitate vigilance for potential side effects. Constipation, due to the aluminum hydroxide component, is the most common and must be monitored.[18] In patients with renal insufficiency, impaired clearance of absorbed aluminum raises a theoretical concern for systemic toxicity, making this regimen relatively contraindicated in that setting.

While sucralfate provides a protective barrier, reducing the corrosivity of the luminal environment is equally critical. This was achieved with intensive, dual-agent acid suppression using both an IV PPI (omeprazole) and an IV H_2RA (ranitidine). While using both may appear redundant, there is a physiological rationale in the acute, critical setting of a severe corrosive burn. PPIs provide the most potent suppression by irreversibly inhibiting the final step in acid secretion—the H^+/K^+ ATPase pump—but they require 24-48 hours to reach maximal effect as they only inhibit actively secreting pumps.[19] In contrast, H_2RAs competitively block the histamine H2 receptor, providing a more rapid onset of action that can reduce nocturnal acid breakthrough and immediately decrease acid production stimulated by histamine. The combination, therefore, theoretically provides both a more rapid and a more profound elevation of intragastric pH in the crucial first 48 hours. This is beneficial because it reduces further direct acid-related damage to the compromised mucosa, minimizes the activity of the proteolytic enzyme pepsin, and optimizes the acidic environment required for the initial polymerization and activation of sucralfate.

The patient’s initial laboratory results—marked leukocytosis, profound reactive thrombocytosis, and a highly elevated CRP—are the classic signature of a potent systemic inflammatory response syndrome (SIRS). This was not a localized reaction but a systemic firestorm triggered by massive tissue injury and necrosis in the gut. The damaged gastric and duodenal tissues release a flood of pro-inflammatory cytokines (such as tumor necrosis factor-alpha, Interleukin-1, and Interleukin-6) and damage-associated molecular patterns (DAMPs) into the circulation. These mediators trigger a systemic response: they stimulate the bone marrow to dramatically increase the production and release of neutrophils (leukocytosis) and platelets (thrombocytosis), and they stimulate the liver to produce acute-phase reactants like CRP. The concurrent aspiration pneumonitis would have undoubtedly contributed to this inflammatory state. These markers served as an invaluable clue to the true severity of the internal injury, belying the benign external physical examination. The steady, day-by-day normalization of these markers provided objective, biochemical evidence of the patient's positive response to the therapeutic regimen, long before endoscopic confirmation of healing was possible.

This case offers several important clinical learning points. First and foremost, the absence of oral lesions or early alarm symptoms like hematemesis does not exclude severe underlying gastrointestinal injury. A high index of suspicion based on the history of ingestion alone is paramount. Second, systemic inflammatory markers (WBC, platelets, CRP) are invaluable tools for gauging the initial severity of the insult and for monitoring the response to therapy, providing a crucial window into the patient's internal state.[20]

Third, this case powerfully illustrates that a positive outcome is possible even when early endoscopy is not feasible. While early endoscopy (within 24-48 hours) remains the undisputed gold standard for diagnosis, prognostication, and guiding therapy, this case demonstrates that in situations where it is delayed (due to patient instability, lack of resources, or parental refusal), immediate institution of maximal medical therapy should not be postponed. The delayed endoscopy on day 12 must be viewed as a circumstance of necessity, not a strategic choice. However, it fortuitously avoided the "blind period" between days 5 and 15 post-ingestion, when the necrotic tissue sloughs off, leaving behind a highly friable and weakened granulation tissue bed that is at the highest risk of iatrogenic perforation during an endoscopic procedure. This case should not be interpreted as an endorsement for delaying endoscopy, but rather as evidence that aggressive, barrier-forming, and acid-suppressing therapy may provide a crucial bridge to a safer, later endoscopic evaluation if necessary. While Grade 2A injuries have a lower intrinsic risk of stricture than more severe grades, the extensive nature of the burns in this patient across multiple anatomical regions (fundus, antrum, duodenum) likely placed him at a higher risk than a more localized Grade 2A injury. The intensive, multi-modal therapy may have been critical in preventing the progression of inflammation to fibrosis in this specific context.

4. Conclusion

This case report details the successful non-surgical management of extensive Zargar Grade 2A gastric and duodenal burns in a toddler following sulfuric acid ingestion. An aggressive, early-initiated conservative protocol—featuring high-dose, frequent-interval sucralfate, intensive dual-agent acid suppression, and appropriate nutritional and antibiotic support—was associated with a complete clinical and endoscopic recovery. This approach appeared effective in preventing both acute complications and the significant long-term sequela of stricture formation, even in a challenging clinical scenario involving delayed endoscopic evaluation. The findings highlight the profound pathophysiological insults of acid ingestion and provide a strong rationale for a multi-modal pharmacological defense. This management strategy, particularly the central role of high-dose sucralfate, shows significant promise and warrants further rigorous investigation in larger, prospective pediatric studies.

5. References

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