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Case Report

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Gastrointestinal involvement and obstructed hernia in a diabetic patient with Stevens-Johnson Syndrome: A case report

Abstract

Stevens-Johnson syndrome (SJS) is a rare but potentially life-threatening mucocutaneous hypersensitivity reaction, most commonly triggered by medications such as carbamazepine. Gastrointestinal (GI) involvement in SJS is uncommon and often under-recognized. We report the case of a 46-year-old diabetic woman recently diagnosed with carbamazepine-induced SJS, who presented with an obstructed paraumbilical hernia and diffuse intraoperative inflammation involving the entire bowel. This case underscores the importance of thorough intraoperative evaluation in patients with recent or ongoing SJS. Surgeons should be cautious in making decisions regarding bowel resection based solely on abnormal serosal appearance, as inflammation may not necessarily indicate irreversible pathology. In this patient, conservative surgical management combined with appropriate postoperative supportive care resulted in a favorable outcome without unnecessary bowel resection.

Keywords: Stevens-Johnson syndrome, gastrointestinal involvement, carbamazepine, abdominal surgery, colonic inflammation, paraumbilical hernia

INTRODUCTION

Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are rare, severe cutaneous adverse reactions characterized by widespread epidermal necrosis and mucosal sloughing. Drug exposure—particularly to anticonvulsants such as carbamazepine—is the most common precipitating factor (1,2). While cutaneous and mucosal involvement is well documented, gastrointestinal (GI) tract manifestations are rare and often under-recognized (3,4).

The GI tract may be affected at various levels, with histopathological findings typically showing epithelial necrosis and lymphocytic infiltration (4-6). This becomes especially significant in vulnerable populations such as diabetic patients, who are predisposed to impaired immune responses, delayed wound healing, and increased surgical risk (7).

In this report, we describe a diabetic patient recently diagnosed with carbamazepine-induced SJS who presented with an obstructed paraumbilical hernia and intraoperative findings of unusual colonic inflammation.

CASE REPORT

A 46-year-old woman with a 10-year history of poorly controlled type 2 diabetes mellitus presented with acute abdominal pain and an irreducible, tender paraumbilical hernia. One month earlier, she had undergone partial toe amputation due to diabetic foot gangrene. She had been prescribed carbamazepine for diabetic neuropathy, but subsequently developed widespread

mucocutaneous lesions, oral erosions, and conjunctival hyperemia—findings consistent with SJS.

On examination, the patient was alert and oriented. She had hyperpyrexia and generalized maculopapular and bullous eruptions affecting the neck, face, and external ears. The trunk and extremities showed well-developed, variably sized, target-like lesions (Figure 1, Figure 2). She also reported burning micturition, and vaginal lesions were clinically confirmed.

The patient was hospitalized in the burns unit, where she received supportive care, systemic corticosteroids, and immediate withdrawal of carbamazepine.

Shortly after recovering from the Stevens-Johnson syndrome episode, the patient presented with acute abdominal pain, persistent vomiting, and a tender swelling around the umbilicus, ongoing for two days. The pain was progressive, colicky in nature, and accompanied by absolute constipation and abdominal distension.

On examination, the patient appeared acutely ill. She was febrile (38.2°C), tachycardic (110 bpm), hypotensive (100/60 mmHg), and clinically dehydrated. Abdominal examination revealed a tender, irreducible paraumbilical hernia with localized signs of peritonitis. The abdomen was distended, and bowel sounds were sluggish.

Laboratory investigations showed leukocytosis (WBC: 18,000/mm³) with a neutrophilic shift, elevated C-reactive protein (CRP), hyperglycemia (blood glucose: 280 mg/dL), and mild metabolic acidosis. Abdominal ultrasound revealed a hernial sac containing non-vascularized bowel loops suggestive of strangulation. After fluid resuscitation, broad-spectrum antibiotics, and optimization, the patient underwent emergency exploratory laparotomy. Intraoperatively, the hernial sac was found to contain congested small bowel loops (Figure 3). On careful examination, the entire small and large intestines exhibited a dusky red, inflamed, and congested appearance, with edematous and friable serosa (Figure 4).

The hernia was reduced, and a primary repair was performed without mesh placement due to the contaminated surgical field. Peritoneal lavage was carried out, and a drain was placed.

The patient was admitted to the intensive care unit (ICU) for close monitoring. She was managed conservatively with bowel rest, parenteral nutrition, and gradual reintroduction of oral intake as her condition stabilized.

Histopathological analysis of serosal biopsies revealed non-specific inflammatory changes consistent with hypersensitivity-related injury, supporting the clinical suspicion of SJS-related gastrointestinal involvement.

The postoperative course was uneventful. The patient was discharged home in stable condition on postoperative day 10 and scheduled for follow-up in the outpatient clinic.

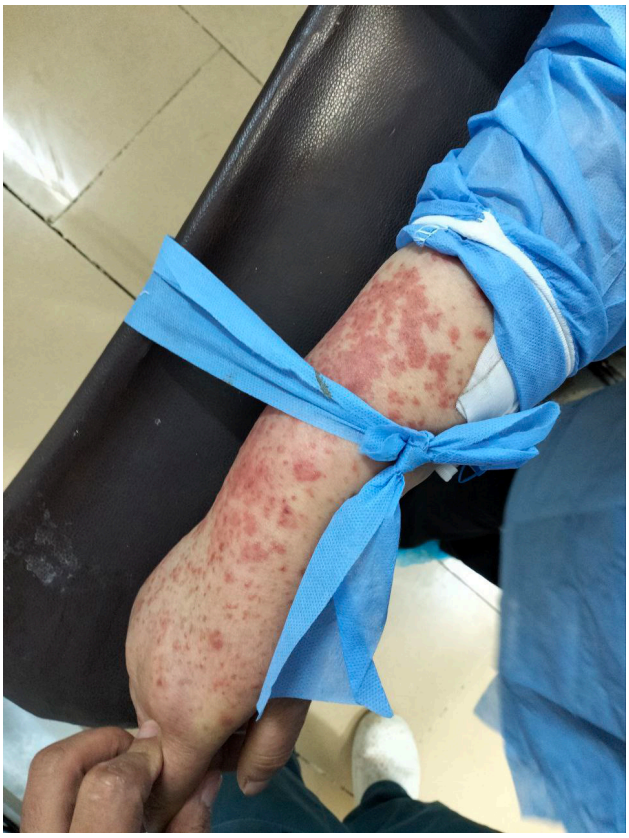

Figure 1. Cutaneous lesions on the forearm during the healing phase

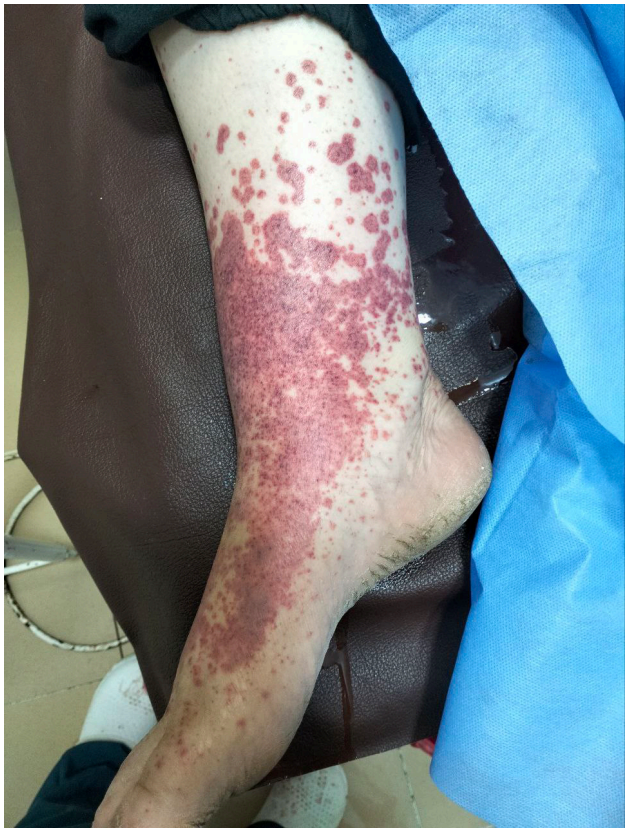

Figure 2. Cutaneous lesions on the leg during the healing phase

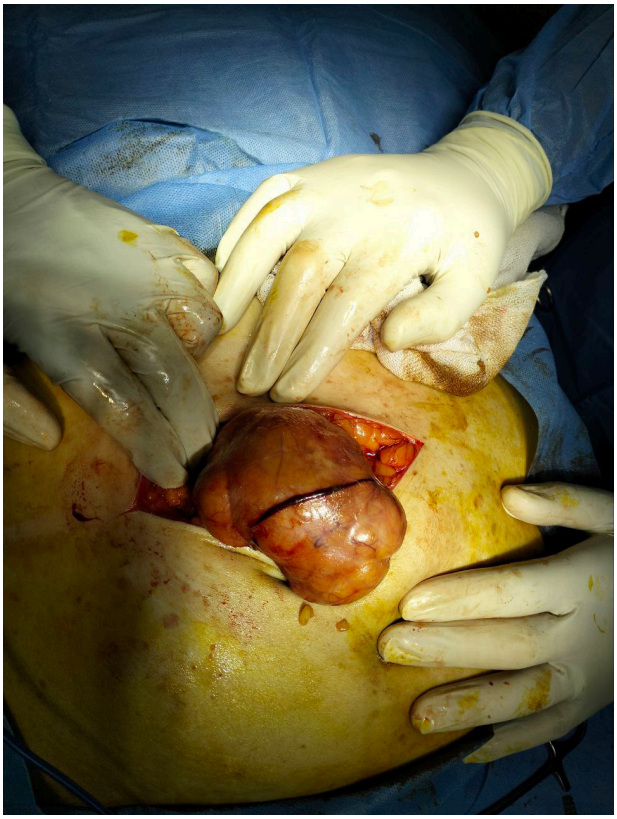

Figure 3. Intraoperative finding showing the hernial sac containing congested small bowel loops

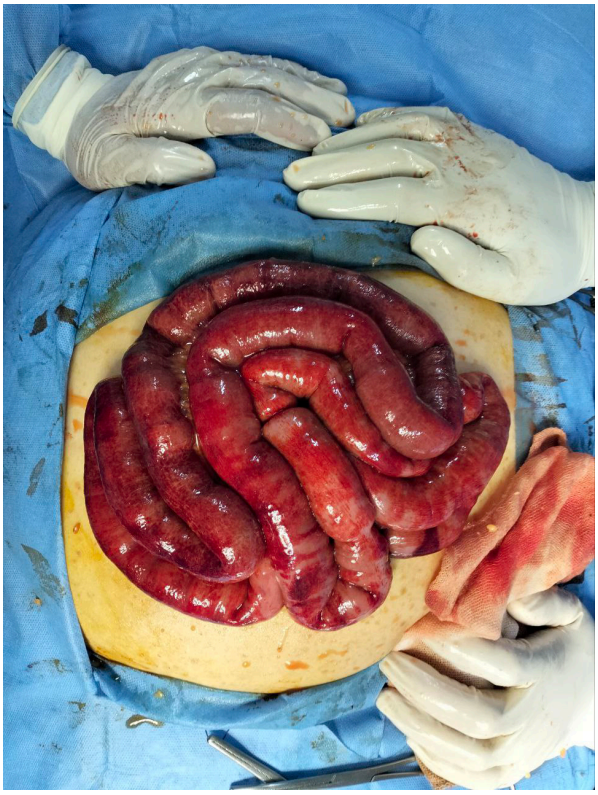

Figure 4. Diffusely inflamed small and large intestines with dusky red discoloration, congestion, and edematous, friable serosal surfaces

DISCUSSION

SJS is a rare but potentially devastating hypersensitivity reaction, most commonly triggered by medications such as carbamazepine, allopurinol, and certain antibiotics (8). It is primarily characterized by widespread mucocutaneous blistering and epithelial necrosis. While cutaneous and mucosal involvement is well established, systemic organ involvement—including GI tract inflammation—has been increasingly recognized in recent years (9).

Gastrointestinal manifestations of SJS may present with abdominal pain, vomiting, diarrhea, gastrointestinal bleeding, or signs of an acute abdomen due to transmural inflammation, ischemia, or even perforation (9,10). The underlying pathophysiology is thought to parallel that of the skin, involving T-cell-mediated cytotoxicity that induces epithelial apoptosis and widespread mucosal injury (10).

In our case, the patient presented with an obstructed paraumbilical hernia. However, intraoperatively, the entire small and large intestines appeared dusky red, inflamed, and congested, with marked bowel wall edema and serosal friability. These findings were more consistent with SJS-related gastrointestinal involvement than with simple mechanical obstruction. Notably, no areas of frank necrosis or perforation were identified, highlighting the systemic hypersensitivity response of SJS rather than localized ischemia as the primary pathophysiological mechanism.

Reviewing the available literature, gastrointestinal (GI) involvement in SJS is reported in fewer than 10% of cases (9,11). The severity of involvement can range from superficial mucosal sloughing to deep ulceration and perforation. Notably, most published reports highlight catastrophic complications such as small bowel perforation, colonic perforation, or severe hemorrhagic enterocolitis (11,12). However, only a few cases have described diffuse, non-necrotic bowel inflammation similar to what was observed in our patient.

Wang et al. (11) reported a series of SJS patients who developed small intestinal perforations requiring extensive bowel resection, with a high associated mortality rate. Similarly, Hong et al. (10) documented cases of gastrointestinal bleeding and mucosal erosions that necessitated aggressive medical management. In contrast, the intraoperative finding of widespread, inflamed, yet viable bowel in our patient is atypical and infrequently reported in the literature.

The management of GI involvement in SJS remains primarily supportive, with an emphasis on fluid and electrolyte balance, infection control, nutritional support, and prevention of further mucosal injury. The use of corticosteroids and immunosuppressive agents in GI SJS remains controversial; however, some studies suggest that early administration may help mitigate immune-mediated epithelial injury (12).

This case also highlights the critical importance of thorough intraoperative assessment. In patients with recent or active

SJS, surgeons should exercise caution before performing bowel resection based solely on abnormal serosal appearance. Judicious intraoperative decision-making, combined with appropriate postoperative supportive care, can lead to favorable outcomes without unnecessary bowel resection.

CONCLUSION

This case highlights a rare but clinically significant presentation of gastrointestinal involvement in SJS, characterized by diffuse inflammatory changes in the bowel without evidence of necrosis or perforation. Awareness of such atypical presentations is essential for surgeons, as early recognition and conservative surgical management may help avoid unnecessary morbidity. Further case reporting and research are warranted to better define the full clinical spectrum and to establish evidence-based management strategies for GI manifestations of SJS.

Conflict of Interests
The authors declare that there is no conflict of interest in the study.

Financial Disclosure
The authors declare that they have received no financial support for the study.

Patient Informed Consent
Written informed consent was obtained from the patient for publication of this case report and accompanying images.

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