



Internal Medicine

HERPES ZOSTER-INDUCED SMALL INTESTINAL PSEUDO-OBSTRUCTION MASQUERADING AS AUTOIMMUNE ENTEROPATHY: A CASE REPORT

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ABSTRACT

Varicella-zoster virus (VZV) establishes latency not only in dorsal root and cranial nerve ganglia but also within enteric ganglia, and its reactivation there can produce gastrointestinal dysfunction, including acute intestinal pseudo-obstruction. We describe an 82-year-old woman who presented with subacute small intestinal obstruction. Evaluation, including a positive antinuclear antibody/anti-double-stranded DNA and an extended extractable nuclear antigen panel, prompted a working diagnosis of late-onset autoimmune enteropathy within an evolving connective-tissue overlap syndrome; diagnostic laparoscopy excluded a mechanical cause, and she improved on corticosteroids. Two days after discharge, she developed a unilateral, dermatomal vesicular rash, later diagnosed as herpes zoster. Re-examination of the timeline revealed that her pseudo-obstruction had begun approximately two weeks before the rash, a pattern consistent with previously reported herpes zoster-induced pseudo-obstruction. She was treated with oral antivirals with continuation of her corticosteroid taper, and at six weeks was symptomatically improved. Enteric zoster should be considered in the differential diagnosis of unexplained intestinal pseudo-obstruction, even in the absence of a rash at presentation, since the cutaneous eruption may lag visceral symptoms by up to several days to weeks.

KEYWORDS : Herpes Zoster; Enteric Zoster; Varicella-Zoster Virus; Intestinal Pseudo-Obstruction; Ogilvie Syndrome; Autoimmune Enteropathy

INTRODUCTION

Acute intestinal pseudo-obstruction, first described by Ogilvie in 1948, denotes massive dilatation of the bowel in the absence of a mechanical obstructing lesion [1]. While adhesions, electrolyte disturbance, opioid use, and neuromuscular disease account for most cases, viral infection is an under-recognised cause [2]. Varicella-zoster virus (VZV), which causes chickenpox on primary infection and shingles on reactivation, establishes lifelong latency not only in dorsal root and cranial nerve ganglia but also within the ganglia of the enteric nervous system [3]. Reactivation confined to, or predominantly affecting, these enteric ganglia has been termed “enteric zoster,” a concept described by Gershon and colleagues, who demonstrated that gastrointestinal dysfunction from VZV reactivation can occur with a delayed or even absent cutaneous eruption [4]. Because the classic dermatomal rash which is the usual trigger for clinical suspicion may follow the visceral symptoms by days to weeks, enteric zoster is frequently missed, and patients may undergo extensive, sometimes invasive, evaluation for alternative diagnoses. We report a case in which small intestinal pseudo-obstruction, initially attributed to a late-onset autoimmune enteropathy was ultimately recognised as enteric zoster only after a dermatomal rash appeared two days after hospital discharge.

Case Presentation

An 82-year-old woman with a background of hypothyroidism, dyslipidaemia, and benign paroxysmal positional vertigo presented with a two-day history of diffuse periumbilical abdominal pain, three episodes of large-volume watery loose stools, and multiple episodes of bilious vomiting. She denied fever, recent travel or outside food intake. On examination she was haemodynamically stable but pale, with diffuse periumbilical tenderness and no peritonism. She was admitted to the intensive care unit for management of subacute intestinal obstruction.

Contrast-enhanced computed tomography of the abdomen showed dilated distal jejunal and mid-ileal loops with submucosal oedema and a smooth transition point at the distal ileum, without an identifiable mechanical obstructing lesion. Given ileal inflammation on imaging together with a positive antinuclear antibody (ANA) and anti-double-stranded DNA (anti-dsDNA), rheumatology was consulted. An extended extractable nuclear antigen (ENA) panel showed nRNP/Sm 1+, SSA 3+, PM-Scl 1+, and ribosomal-P 1+, with a negative antiphospholipid antibody panel; C-reactive protein was 20 mg/L and serum creatinine had risen from 0.9 to 1.1 mg/dL. An initial working impression of autoimmune overlap syndrome with small intestinal involvement was considered.

To exclude a mechanical cause before escalating immunosuppression, diagnostic laparoscopy was performed on hospital day five. This

revealed dilated bowel from the duodenojejunal flexure to the proximal ileum, with the proximal ileum and distal jejunum adherent to the pelvis. The terminal ileum and ascending colon were collapsed, and the remaining small bowel was grossly dilated with an ischaemic-appearing serosa, although peristalsis was preserved throughout. Adhesiolysis and a bowel walk were performed, and no mechanical obstruction was identified. In the absence of a structural cause, systemic corticosteroids (intravenous hydrocortisone, later transitioned to oral prednisolone) were initiated for a presumed immune-mediated process.

The patient's symptoms gradually improved, and she tolerated oral feeds. At discharge on hospital day eleven, her working diagnoses were late-onset autoimmune enteropathy. She was discharged on a tapering course of oral prednisolone and co-trimoxazole prophylaxis. Two days after discharge, she developed a painful, grouped vesicular and crusted eruption over the lower thoraco-abdominal wall. The rash was strictly unilateral, confined to a dermatomal band, and did not cross the midline. There were no preceding trauma and no similar lesions elsewhere. A clinical diagnosis of herpes zoster was made in this elderly patient on recent high-dose corticosteroid therapy.

Reviewing the timeline retrospectively, the onset of abdominal symptoms preceded the cutaneous eruption by approximately fifteen days. This interval, together with the absence of any mechanical or other identifiable cause for her pseudo-obstruction, supported enteric zoster which is the reactivation of VZV within enteric ganglia was considered as the unifying explanation for her presentation.

Oral antiviral therapy was commenced at the time of rash diagnosis. The corticosteroid taper already in progress was continued. She received analgesia and local wound care for the cutaneous lesions, with close monitoring of renal function, glycaemic status, and bowel symptoms. At six-week follow-up, she reported normal bowel habits and was tolerating a regular diet; the cutaneous lesions had healed with residual post-inflammatory pigmentation and no post-herpetic neuralgia.

DISCUSSION

This case illustrates a recognised but frequently overlooked pattern: gastrointestinal dysfunction from VZV reactivation in enteric ganglia can precede, rather than follow, the cutaneous eruption that ordinarily prompts diagnosis [4,5]. In a systematic review of 28 published cases of herpes zoster-associated colonic pseudo-obstruction, pseudo-obstruction preceded the rash in 58% of patients, by a range of one to sixteen days; the mean patient age was 60 years, and nearly half had an underlying immunosuppressive condition [6]. Our patient's interval of approximately fifteen days sits at the upper end of this reported range and, notably, involved the small bowel rather than the colon, a less

commonly reported site [7,8].

The proposed mechanisms linking VZV reactivation to bowel dysmotility include direct viral injury to the myenteric and submucosal plexuses and the muscularis propria, invasion of the coeliac plexus with disruption of sympathetic and parasympathetic outflow, haemorrhagic infarction of the abdominal sympathetic ganglia, interruption of afferent C-fibres producing intestinal hypomotility, and parietal or visceral peritoneal inflammation [9,10]. Because VZV can reach enteric ganglia either by retrograde axonal transport from the dorsal root ganglia or via infected T-lymphocytes during viraemia, visceral symptoms can plausibly arise from ganglionic reactivation that has not yet, or never will, manifest cutaneously as described by Gershon et al. [4].

Confirmatory testing for enteric zoster remains challenging when a rash is absent or has yet to appear. Tzanck smear from a vesicle base, when present, offers rapid but poorly sensitive and non-specific support. Polymerase chain reaction for VZV DNA in vesicle fluid, rash-biopsy tissue, saliva, or gut biopsy where obtained, is considered the reference standard, and immunohistochemical demonstration of VZV antigen within the myenteric or submucosal plexus has confirmed the diagnosis in surgically resected or biopsied specimens in prior reports [4,9]. In our patient, the diagnosis rested on the classical clinical evolution and temporal correlation rather than virological confirmation in bowel tissue, since no rash was present at the time of laparoscopy to prompt such testing; this is a limitation shared by most reported cases and reflects the retrospective nature of the diagnosis in the absence of an early cutaneous or mucosal clue.

A second point of interest in this case is the diagnostic overlap with autoimmune disease. Low-titre positivity for multiple autoantibodies (nRNP/Sm, SSA, PM-Scl) in an acutely unwell, elderly patient is a well-recognised confounder; such positivity may reflect a genuine evolving connective-tissue disease, but can equally represent a transient epiphenomenon of immune dysregulation [11]. This uncertainty underscores the value of maintaining a broad differential and revisiting the diagnosis when new clinical information like development of a delayed rash.

From a management perspective, this case reinforces existing guidance that acute intestinal pseudo-obstruction is a diagnosis of exclusion, and that mechanical causes should be ruled out, where feasible by minimally invasive means, before instituting or escalating immunosuppression [12]. It further highlights a practical safety benefit of considering enteric zoster leads to earlier recognition and prompt antiviral therapy.

CONCLUSION

Enteric zoster is an under-recognised cause of intestinal pseudo-obstruction that can present without, or well before, its characteristic cutaneous eruption, particularly in elderly or immunosuppressed patients. Clinicians should consider VZV reactivation in the differential diagnosis of unexplained pseudo-obstruction even when autoimmune serology appears to offer an alternative explanation and should remain alert to a delayed dermatomal rash as a retrospective diagnostic clue. Early recognition can avoid unnecessary escalation of immunosuppression and direct patients toward timely antiviral therapy.

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Ethical Approval: Not required

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