

# Ogilvie's Syndrome and Herpes Zoster: A Case Report

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## Abstract

A case of intestinal pseudo-obstruction (Ogilvie's syndrome) secondary to herpes zoster infection. We discuss the association of Ogilvie's syndrome with herpes zoster as well as investigations and course of treatment of these cases. An understanding of the rare association is important in correct management of such cases to prevent associated morbidity and mortality.

**Keywords:** Ogilvie's syndrome, Herpes zoster infection, Intestinal pseudo-obstruction

## Introduction

Intestinal pseudo-obstruction is a disorder characterized by dilatation of the bowel in the absence of anatomical obstruction. This condition can begin at any age, and it can be primary (idiopathic or congenital) or secondary to another disease. It most commonly involves Colon, and can be classified to acute and chronic forms.

Ogilvie's syndrome is defined as acute colonic dilatation and is most commonly seen in hospitalized patients with severe stress due to infections or in the postoperative settings. Drugs that disturb the colonic motility can also contribute to this condition. Imbalance in the regulation of colonic motility by the autonomic nervous system is the postulated mechanism for Ogilvie's syndrome.

The management of Ogilvie's syndrome primarily involves stopping oral ingestion, nasogastric tube placement, pharmacological agents like neostigmine, and colonoscopic decompression. It is important to address the underlying cause in cases of secondary pseudo-obstruction.

## Case Presentation

A 61 year old man who was a known diabetic and hypertensive not compliant with medications presented with abdominal pain for 3 days. Pain was episodic, moderate in intensity, non-radiating and was present over left lower quadrant of the abdomen. The pain was associated with abdominal distension, constipation and obstipation. There was no history of any prior abdominal operations or similar episodes in the past. He also complained of fluid filled painful blisters on the left side of the abdomen for 2 days.

On examination, he was afebrile and hemodynamically stable. Multiple grouped vesicles were present over the left T10 dermatome (Figure 1). Abdomen was distended, with no tenderness or guarding and bowel sounds were sluggish. There was no abnormality on digital rectal examination. Supine abdominal X-ray showed dilated colon. Contrast enhanced Computed tomography of the abdomen and pelvis was done which revealed dilated ascending and transverse colon with narrowing at the splenic flexure (Figure 2). There was no abnormality in the colonic wall at the site of narrowing. This was followed by gastrograffin enema which did not reveal any mechanical cause of obstruction (Figure 3). He was managed non operatively with bowel rest, nasogastric decompression and IV fluids. Dermatology consultation was sought for the vesicular lesion over abdomen. He was diagnosed with herpes zoster following a Tzanck smear and started on IV Acyclovir (10 mg/kg Q8h). He passed stool and flatus within 24 hours of admission and was allowed a normal diet from the 2<sup>nd</sup> day. A Colonoscopy was done which was normal and he was discharged from hospital after 3 days. He came

for regular follow up on outpatient basis and did not have recurrence of symptoms over the next 3 months.

## Discussion

The primary infection by Varicella Zoster virus causes chicken pox which is characterised by diffuse papulovesicular rash. During the primary infection, the virus seeds in the dorsal root ganglion, cranial nerve ganglion and enteric nervous system and establishes latency [1,2]. The virus is transported via retrograde transmission through axons or seeds as a result of viremia [1,3]. Subclinical viremia secondary to vaccination also results in latency in dorsal root ganglion, cranial nerve ganglion as well as enteric nervous system [3,4]. Reactivation of the latent virus in dorsal root ganglion and cranial nerve ganglion, seen in 30% of individuals presents with papulovesicular lesions over single dermatome or cranial nerve distribution. Similarly, reactivation of the virus in enteric nervous system can cause several gastrointestinal disorders like acute or chronic colonic pseudo-obstruction and



Figure 1: Multiple grouped vesicles present over the left T10 dermatome.

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**Figure 2:** CECT abdomen: narrowing at the splenic flexure.



**Figure 3:** Gastrograffin enema: free flow of contrast into distal ileum.

idiopathic gastroparesis [2,3]. Apart from reactivation involving enteric nervous system, three other mechanism by which varicella zoster can cause gastrointestinal diseases have been proposed. Firstly, involvement of the extrinsic autonomic nervous system either through infection of motor neurons in anterior horn of spinal cord, involvement of celiac plexus ganglion or infection of sacral lateral columns [4-7]. Secondly, localised parietal and visceral peritoneal inflammation secondary to

vesicular eruption of the overlying dermatome [4,7]. Thirdly, infection of the smooth muscle of muscularis propria [7].

A review of 29 cases published in 21 reports between of 1950 to 2008 revealed that cutaneous manifestations may appear one day to one month before the pseudo-obstruction, appear simultaneously or 1 day to several weeks after the pseudo-obstruction [8]. History of vesicular lesions in recent past and detailed examination to look for herpes zoster should be part of evaluation of cases of Ogilvie's syndrome. Routine microbiological investigation like PCR, immunofluorescence testing, cell cultures to detect VZV in stool, blood or colonic wall is not recommended because of variable sensitivity and specificity of these and high cost [2]. The diagnosis of varicella zoster should be sufficient to establish association and initiate pharmacological treatment with Acyclovir.

## Conclusion

The pathogenesis of Ogilvie's syndrome secondary to herpes zoster involves reactivation of the VZV as a result of which initiation of Acyclovir along with the management of pseudo-obstruction is of primary importance for treatment as well as prevention of morbidity and mortality associated with Ogilvie's syndrome.

## Conflict of interest

Authors have no conflict of interest to disclose.

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