

A Rare Case of Ogilvie Syndrome Associated with Herpes Zoster

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Acute colonic pseudo-obstruction (Ogilvie syndrome) associated with herpes zoster is extremely rare, and few cases have been reported. An 81-year-old woman diagnosed with herpes zoster was referred for accompanying colonic ileus. The diameter of the cecum was 7 cm and a computed tomographic scan showed no definite obstructive cause. Because the patient showed minimal improvement with conservative treatment, endoscopic colonic decompression was performed successfully. Previous studies revealed that the treatment of Ogilvie syndrome associated with herpes zoster does not differ from that of other conditions, and the role of the varicella-zoster virus in this syndrome is unclear. Here, we present the first case of Ogilvie syndrome associated with herpes zoster in Korea, which was improved by endoscopic colonic decompression. (**Intest Res 2012;10:379-382**)

Key Words: Colonic Pseudo-obstruction; Ogilvie Syndrome; Herpes Zoster

INTRODUCTION

Acute colonic pseudo-obstruction is characterized by massive colonic dilatation with symptoms and signs of colonic obstruction in the absence of a mechanical blockage.¹ Because it was first described by the British surgeon Sir William Heneage Ogilvie, it is also known as Ogilvie syndrome.² This disease is often associated with life-threatening complications, and the mortality rate is estimated to be at least 50%.¹ The majority of cases are associated with several causes or predisposing factors, such as surgery, trauma, electrolyte imbalance, sepsis, viral infection, medication, cardiac disorders, or renal insufficiency.³ Herpes zoster is a very rare cause of Ogilvie syndrome, and few cases have been reported.^{2,4,5} Here, we present the first case of Ogilvie syndrome associated with herpes zoster in Korea.

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CASE REPORT

An 83-year-old woman was admitted to a local hospital be-



Fig. 1. Herpes zoster skin lesion on the abdominal wall.

cause of herpes zoster on her abdominal wall (Fig. 1). Three days later, she complained that her abdominal distension and pain were worsening. An abdominal X-ray showed colonic dilatation with no definite obstructive cause, and the patient was referred to our hospital (Figs. 2, 3). She had no significant medical or surgical history and she took no regular medication. On admission, her vital signs were: blood pressure 100/70 mmHg, pulse rate 74 beats/min, temperature 36.3°C, and respiratory rate 18/min. Abdominal distension was observed with tenderness and decreased bowel sounds, with no sign of peritoneal irritability. Laboratory findings were: white blood cell count 8,340 cell/ μ L, hemoglobin 13.9 g/dL, platelet count 404,000/mm³, serum sodium 144 mEq/L, potassium 4.76 mEq/L, chloride 102 mEq/L, aspartate aminotransferase 21 IU/L, alanine aminotransferase 16 IU/L, blood urea nitrogen 16 mg/dL, creatinine 0.7 mg/dL, C-reactive protein 3.23 mg/L, T3 1.20 ng/mL, free T4 1.16 ng/dL, and thyroid-

stimulating hormone 1.08 μ IU/mL. As the patient's general condition was good and the cecal diameter was only 7 cm, we decided to maintain conservative treatment, such as a nasogastric tube, for gut decompression and maintenance of nil per os with parenteral support. Antiviral therapy for herpes zoster had begun before admission, and we completed the 7 day therapeutic course. After 24 hours from admission, abdominal distension was not improved and an abdominal X-ray showed persisting colonic and small bowel dilatation. We checked the CT scan to exclude mechanical obstruction, and confirmed colonic dilatation with no evidence of an obstructive lesion. Therefore, we strongly suspected Ogilvie syndrome induced by herpes zoster. Although the patient complained frequently of abdominal pain, most episodes were abdominal wall pain due to herpes zoster and she showed no sign of peritonitis during treatment. Several guidelines rec-



Fig. 2. Initial plain abdominal radiograph showing colonic dilatation.



Fig. 4. Plain abdominal radiograph after colonoscopic decompression showing markedly decreased bowel dilatation.

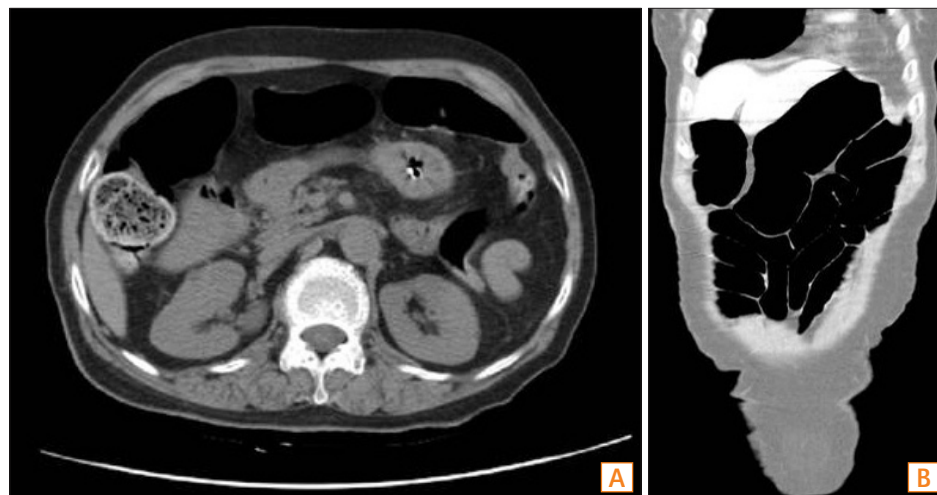


Fig. 3. Computed tomographic image showing colonic dilatation with no structural obstruction. (A) Axial view. (B) Coronal view.

ommend conservative treatment for 24-48 hours; however, we prolonged treatment due to the patient's good performance.³ After 4 days from admission, despite improved herpes zoster symptoms and general condition, an abdominal X-ray showed no improvement in colonic dilatation. Instead of a pharmacological therapy such as neostigmine, we decided to try colonoscopic decompression due to the patient's age and the probability of cardiac side effects. She underwent colonoscopic decompression with no bowel preparation. Colonoscopy showed marked dilatation of the ascending colon, with fecal contents in several locations. The whole colon was observed and decompressed. No complication of the colonoscopy occurred. Colonic dilatation was again observed after 3 days, and colonoscopic decompression was performed once more. Abdominal distension and colonic dilatation were much improved after the second procedure; flatus and bowel sounds were also noticed. The patient was then able to begin eating a soft-food diet, and her abdominal X-ray results continued to show improvement (Fig. 4). She was discharged 2 weeks after admission without recurrence and required no further medication or procedure.

DISCUSSION

Acute colonic pseudo-obstruction (Ogilvie syndrome) is a rare disease. It was first described in 1948 as acute massive colonic dilatation, usually associated with an underlying disease.⁴ It is associated most commonly with intraperitoneal or extraperitoneal surgery. Trauma, electrolyte imbalance, sepsis, medication, cardiac disorders, and renal insufficiency can also be associated with Ogilvie syndrome; viral infection, such as herpes zoster, is a rare cause. The most common clinical manifestation of herpes zoster is a vesicular eruption with dermatomal distribution, and gastrointestinal symptomatology is an extremely rare complication.

Edelman et al.² searched the MEDLINE database for articles published between 1950 and 2008 describing patients affected simultaneously by herpes zoster and Ogilvie syndrome. They identified 20 studies of 28 adult patients, with a variable onset time of skin eruption with pseudo-obstruction. Three hypotheses have been proposed to describe the role of varicella-zoster virus (VZV) infection in Ogilvie syndrome:⁴ involvement of the extrinsic autonomic nervous system through the anterior horn of the spinal cord or celiac plexus ganglion, involvement of the intrinsic nervous system through the myenteric plexus, and localized parietal and visceral peritoneal inflammation caused by the overlying dermatome. Pui et al.⁵ suggested another hypothesis based that was treated by a partial ileocolectomy. They demonstrated evidence of VZV infection in the muscularis propria and myenteric plexi of the resection specimen and postulated that the viral infection in the neuronal plexi and muscularis propria of the colon caused muscle injury leading to pseudo-obstruction. The exact mechanism of herpes zoster involve-

ment in Ogilvie syndrome is unclear; thus, further research is necessary.

Previous studies have reported that the treatment of Ogilvie syndrome associated with herpes zoster does not differ from the treatment of other conditions. The American Society for Gastrointestinal Endoscopy³ suggested increased risk thresholds of 9 cm for the transverse colon and 12 cm for the cecum. The initial step in the management of Ogilvie syndrome is conservative therapy, including evaluation of the contributing factor, electrolyte correction, discontinuation of precipitating medication, placement of a nasogastric and/or rectal tube, a tap-water enema, and optimal body positioning. These can be continued for 24-48 hours. The success rate of conservative treatment is 20-92%.⁶ If conservative therapy fails or the cecal diameter is longer than 12 cm, pharmacological therapy such as neostigmine can be used. Neostigmine (2 mg over 3-5 min), an anticholinesterase parasympathomimetic agent, is the best-documented pharmacological therapy for Ogilvie syndrome. The long-term response rate is 79-100%, but close monitoring during administration is necessary due to the risk of bradycardia, asystole, hypotension, seizure, and bronchoconstriction.^{2,3} In cases of medical treatment failure, the next step is endoscopic treatment. Although the efficacy of endoscopic treatment has not been established in randomized trials, colonoscopic decompression is the preferred method. One study revealed initial decompression was successful in 69% of 292 patients, but the success rate varied from 61% to 95%.⁶ The complication rate of decompression colonoscopy is approximately 3%, including perforation (2%).³ Surgical therapy is the last resort, and the mortality rate can be higher than in other conditions.¹ Because of this increased risk, percutaneous endoscopic colostomy has been attempted as an alternative treatment.⁷ However, an insufficient number of cases have accumulated and this procedure is usually performed to treat chronic intestinal pseudo-obstruction; thus, more clinical experience is required.⁸ Edelman et al.² reported that 83% of patients with herpes zoster and Ogilvie syndrome were observed and treated conservatively. Three (10%) patients underwent nonsurgical decompression and five (17%) underwent surgery. Similarly, although our patient started antiviral therapy before the appearance of colonic pseudo-obstruction, such therapy did not prevent its occurrence. However, because the pathogenesis of herpes zoster with colonic involvement is associated with the interaction between VZV and the nervous system, antiviral therapy may affect the progress of colonic pseudo-obstruction. Further investigations and more clinical experience are needed to fully elucidate the relationship between VZV and colonic pseudo-obstruction.

We present here a rare case of herpes zoster associated with colonic pseudo-obstruction; to our knowledge, this is the first such report from Korea. Rare causes of colonic pseudo-obstruction should not be overlooked because recognition of the correct comorbidity at the right time may help to avoid

unnecessary treatment and shorten the period of hospitalization.

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