Ogilvie’s syndrome in a patient with multiple myeloma

Multipl myelomalı bir hastada Ogilvie’s sendromu

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Ogilvie’s syndrome is characterized by physical examination and radiologic findings indicative of mechanical obstruction but in which no physical obstructive process can be found. Many factors have been associated with this syndrome which include electrolyte imbalance, systemic infection, drugs, and occasionally, neurologic disease. A case of acute colonic pseudo-obstruction is presented which developed in a patient with multiple myeloma. The patient presented with severe thoracic pain, persistent and increasing abdominal distention and lack of bowel sounds. Plain radiography and ultrasonography revealed massive dilatation of the right and transverse colon. Nasogastric aspiration was initiated and all analgesic drugs were withdrawn. Erythromycin was given for nine days as prokinetic and a rectal tube was inserted for one day. Abdominal distention gradually disappeared within one day of nasogastric and rectal tube insertion and with multiple myeloma management.

Ogilvie’s syndrome is a very rare complication of multiple myeloma. Only one case of Ogilvie’s syndrome with multiple myeloma has been reported in the literature. This case report of Ogilvie’s syndrome in a patient with multiple myeloma is the second case report in the literature.

Key words: Ogilvie’s syndrome, Multiple myeloma.

INTRODUCTION

In 1948, Ogilvie described two patients with colonic pseudo-obstruction caused by malignant infiltration of the celiac plexus and attributed the syndrome to sympathetic deprivation (1). This severe form of adynamic ileus, also known as Ogilvie’s syndrome, is associated with a wide variety of medical and surgical conditions. Although the pathophysiology of acute colonic pseudo-obstruction is not fully understood, it is thought to result from an imbalance in the regulation of colonic motor activity by the autonomic nervous system. To our knowledge, there is only one previous report in the literature of Ogilvie’s syndrome associated with multiple myeloma and this is the second case report to be presented.

CASE REPORT

A 55-year-old male presented with severe thoracic pain, persistent and increasing abdominal distention and absence of bowel sounds. The diagnosis of multiple myeloma was made on the basis of bone marrow aspiration, mild hypercalcemia (10.4 mmol/l), hyperproteinemia, hyperglobulinemia and high sedimentation rate (108 mm/h). In the protein electropheresis, beta globulin level was high (37.3 % g/dl, n: 8.5-14.5 g/dl). Typically, the
myeloma cells were moderately large (15 to 30 mm), round or ovoid, and contained a nucleus about 5 to 7 mm in diameter. The bone marrow contained 35% myelomatous plasma cells. Plain radiography and ultrasonography revealed massive dilatation of the right and transverse colon, there were no air fluid levels or evidence of free air. In direct abdominal x-ray, the caecum diameter was 10.5 cm and ascending colon diameter was 11.5 cm. A thorough physical examination and rigid sigmoidoscopy revealed no mechanical cause for the colonic distention (Picture 1) and it was diagnosed as acute colonic pseudo-obstruction (Ogilvie’s syndrome). Bowel sounds were normoactive and the stool was negative for occult blood. Nasogastric aspiration was initiated, oral feeding discontinued and all analgesic drugs were withdrawn. Erythromycin was given for nine days as prokinetic and a rectal tube was inserted for one day. Abdominal distention gradually disappeared with nasogastric and rectal tube insertion and multiple myeloma management. He remained afebrile, had a normal white cell count, and was not acidic.

DISCUSSION

Ogilvie’s syndrome consists of massive dilatation of the colon in the absence of mechanical obstruction. Although the pathophysiology of acute colonic pseudo-obstruction is not fully understood, it is thought to result from an imbalance in the regulation of colonic motor activity by the autonomic nervous system. There is now a better understanding of the autonomic innervation of the colon. The parasympathetic nervous system increases contractility, whereas the sympathetic nerves decrease motility. Multiple pharmacologic and metabolic factors, as well as spinal and retroperitoneal trauma, can alter the autonomic regulation of colonic function, leading to excessive parasympathetic suppression, sympathetic stimulation, or both. This imbalance results in colonic atony or pseudo-obstruction and forms the rationale for pharmacologic manipulation of the autonomic innervation of the colon in patients with this condition.

Ogilvie’s syndrome is a rare condition usually arising in elderly patients (3), many of whom have concomitant medical or surgical problems. Several conditions have been associated with the development of Ogilvie’s syndrome; these include electrolyte abnormalities, myocardial infarction, respiratory failure (3), sepsis and burns (4, 5), obstetric or gynecologic procedures (3, 6), acute myeloid leukemia (7) orthopedic surgery (8, 9), urologic surgery, renal transplantation (10), pancreatitis, and certain drugs (11-13). However, the precise pathophysiology involved in these various conditions is yet to be clarified.

Multiple myeloma (MM) is a neoplasm of mature and immature plasma cells (14). The clinical manifestations of this disorder are a result of the proliferation and accumulation of these cells, the effect of marrow replacement by them, and the pathologic manifestations occasioned by their overproduction of certain proteins and their constituent polypeptide chains (M-components). The incidence of Ogilvie’s syndrome in patients with multiple myeloma is unknown. Only one case of Ogilvie’s syndrome with multiple myeloma has been reported in the literature (2). An elderly Spanish patient had been admitted to hospital with colonic obstruction due to Ogilvie’s syndrome.
and successful decompression had been achieved by colonoscopy. Our case is the second case report in the literature. The mechanism involved in Ogilvie’s syndrome associated with multiple myeloma, whether it was a manifestation of subtle neuro-hormonal imbalances, electrolyte disturbances, or altered functioning of the autonomic nervous system as originally suggested by Ogilvie, is unknown. The authors believe that some metabolic factors, immobilization and sympathetic stimulations can also play an important role in the pathophysiology of Ogilvie’s syndrome.

The treatment of colonic pseudo-obstruction varies with the condition of the patient and the severity of symptoms. The first step is to ascertain that there is no mechanical obstruction that requires operative correction. Most patients deserve a trial of conservative therapy first, since the vast majority of patients will respond to non-operative treatment. This includes strict cessation of oral intake, insertion of a nasogastric tube, intravenous fluid administration, correction of any underlying electrolyte abnormalities and possibly the insertion of a rectal tube. Other adjunctive measures that have been effective in certain clinical situations include endoscopically placing a long tube into the proximal colon and the use of certain medications such as neostigmine (16-19) and erythromycin (21) to stimulate colonic activity. Patients who do not respond to conservative measures or who develop signs of impending or actual bowel necrosis warrant immediate exploration. The signs and symptoms that warrant consideration of operative intervention include increasing white blood cell count, fever, worsening abdominal tenderness, and a cecal diameter equal to or greater than 12 cm (17). The approximate risk of spontaneous perforation is three percent, with an attendant mortality rate of 50 percent. Most cases respond to conservative management. Although its value is unproved, colonoscopic decompression is often performed to prevent ischemia and perforation of the bowel in patients who have no response to conservative management. Colonoscopy has been the principal tool for decompression in acute colonic pseudo-obstruction, known as Ogilvie’s syndrome (2,7,15), but is technically difficult and not always successful. Colonic distention may recur in up to 40 percent of patients despite initial decompression. The results of three uncontrolled studies suggest that the intravenous administration of neostigmine, an acetylcholinesterase inhibitor, produces rapid colonic decompression in patients with acute colonic pseudo-obstruction (18-21). This pharmacologic approach is based on the theory that acute colonic pseudo-obstruction is the result of inefficient colonic motility caused by excessive sympathetic stimulation, parasympathetic dysfunction, or both. Ogilvie’s syndrome may resolve with conservative treatment, but if the cecal diameter continues to increase, other treatments (neostigmine, colonoscopy or laparotomy) may be needed to prevent perforation of the colon. In our case, Ogilvie’s syndrome resolved with conservative treatment and multiple myeloma management.

In conclusion, Ogilvie’s syndrome, or acute colonic pseudo-obstruction, is a very rare complication of multiple myeloma. This association has previously been described only once to our knowledge and our case report of Ogilvie’s syndrome in a patient with multiple myeloma is the second such case report in the literature.

REFERENCES


