Acute colonic pseudo-obstruction (Ogilvie's syndrome) with myxedema coma and palmoplantar keratoderma

Miksödem koması ve palmoplantar keratodermanın eşlik ettiği akut kolonik psödo-obstsrüksiyon (Ogilvie sendromu)

To the editor,

Although myxedema coma is rarely seen, mortality rate is above 50% even with modern treatment. Rarely, palmoplantar keratoderma and type-2 respiratory failure are observed (1,2). To the best of our knowledge, no study on the coexistence of acute colonic pseudo-obstruction (Ogilvie' s syndrome) with myxedema coma and palmoplantar keratoderma has been previously reported in the literature.

A 51-year-old female patient applied with fatigue and non-flowing desquamous lesions on the perioral area and hands (Figure-1A). The general situation was bad with blood pressure: 90/50 mmHg, pulse: 96/min, fever: 36.8 °C, and respiratory muscle dysfunction. The patient's laboratory findings displayed hemoglobin: 6.9 g/dL, leukocyte count: 3.39.10³/mcL, Na: 129 mmol/L, albumin: 2.5 g/dL, f T4: 0.3 ng/dL (N: 0.8-1.9), TSH: 49.9 microI- U/mL (N: 0.4-4), anti-Tg: 90.01 IU/mL (N:1- 4.11). Physical examination and laboratory findings revealed that the patient had clinical myxedema coma. Medication with thyroxin sodium (0.05 mg/day) was planned. During the follow-up period, ileus developed. Gas-fluid levels and dilatations on intestines and colons were observed upon the abdominal spiral computed tomography. Pneumonitis intestinalis was present in the sigmoid colon and cecum (Figure-1B). With these findings, Ogilvie's syndrome was considered. The patient developed respiratory distress, hypotension and cold intolerance and was transferred to the intensive care unit. Her arterial blood gas result was as follows: pH: 7.204, pO₂: 63.4 mmHg, pCO₂: 68.2 mmHg, HCO3: 18.4 mmol/ L, oxygen saturation: 86.3%. With mentioned analysis results, hypoventilation and respiratory distress, type-2 respiratory failure



Figure 1. (A). Palmoplantar keratoderma (B) Abdominal spiral computed tomography

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was thought. Later the patient was intubated and cardiac arrest developed. The patient could not respond to the cardiopulmonary resuscitation.

The reasons underlying acute colonic pseudo-obstruction are either surgical or medical, which might be associated with intra-, retro-, extraperitoneal surgery, trauma, sepsis, malignancy, systemic diseases, fluid-electrolyte imbalances. The pathogenesis generally cannot be clarified. The most common mechanism is the sympathetic-parasympathetic neurostimulatory imbalance (3). The myxedematous respiratory failure is rarely seen in elders. A series consisting of seventeen patients was presented by Guo et al. (4). Alopecia,

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hyperhidrosis, xanthomas and palmoplantar keratoderma may be seen in thyroid diseases (1). Palmoplantar keratoderma co-occurring with myxedema has been reported in the literature (1, 5); however, the pathogenesis cannot be clearly identified. Keratin-1 and keratin-2 mutations are suspected. Hypothyroidism with myxedematous respiratory failure in elders is vitally important in terms of prognosis (6).

As a consequence, Ogilvie's syndrome may present with many varying clinical situations. Coexisting with acute colonic pseudo-obstruction and myxedema coma, palmoplantar keratoderma should also be kept in mind.

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Solitary rectal ulcer syndrome presenting as polypoid mass lesions in a female patient

Bir kadın hastada polipoid kitle lezyonları şeklinde görülen soliter rektal ülser sendromu

To the editor,

Solitary rectal ulcer syndrome (SRUS) is a rare benign disease of the rectum, which predominately affects young adults aged between 30 and 50 years with a prevalence of 1 in 100.000 people per year (1). SRUS usually presents with a symptom complex of rectal bleeding, passage of mucus and straining on defecation, tenesmus, perineal and abdo-

Address for correspondence: Ümit Bilge DOĞAN Adana Numune Training and Research Hospital, Department of Gastroenterology, Adana, Turkey E-mail: ubdogan@hotmail.com minal pain, sensation of incomplete defecation, constipation and rectal prolapse (2). The underlying etiology of SRUS is not fully understood, but it is likely to be secondary to ischemic changes in the rectum associated with paradoxical contraction of the pelvic floor and external anal sphincter muscles and with rectal prolapse (3). The macros-

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