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Hyperkalemia as a manifestation of paraneoplastic syndrome in esophageal cancer: a case report

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Abstract. Paraneoplastic syndromes are clinical manifestations associated with malignant neoplasms that are not directly related to local tumor growth or metastatic spread. They may significantly complicate diagnosis and treatment planning in cancer patients. Paraneoplastic syndromes are most commonly described in lung, breast, and pancreatic cancers, whereas such manifestations are extremely rare in esophageal cancer. This article presents a clinical case of esophageal adenocarcinoma complicated by persistent hyperkalemia, regarded as a manifestation of a paraneoplastic syndrome. The electrolyte disturbance was resistant to medical correction, which made standard chemotherapy and surgical treatment impossible. External beam radiotherapy to the primary tumor and regional lymphatic pathways was used as the first stage of treatment, resulting in normalization of serum potassium levels. This allowed subsequent neoadjuvant chemotherapy followed by radical surgical intervention, achieving a complete pathological response. The presented case highlights the importance of recognizing paraneoplastic electrolyte disorders in esophageal cancer and demonstrates the significance of an individualized treatment approach in patients with atypical disease course.

Keywords: paraneoplastic syndrome, hyperkalemia, esophageal cancer, adenocarcinoma, radiotherapy

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Introduction. Paraneoplastic syndromes (PNS) are systemic disorders in cancer patients triggered by the body's reaction to tumors, through the secretion of hormones, enzymes, peptides, or cytokines. Early mentions about PNS date back to 1865 [1], when A. Strauss described migratory thrombophlebitis in gastric carcinoma, and to 1888 [2], when H. Oppenheim described neurological symptoms associated with cancer that arose in three patients with gastric, breast, and uterine cancers. It should be noted that the term "paraneoplastic syndrome" was introduced only in 1949 by A. Guichard and G. Vignon [3]. Paraneoplastic syndromes commonly manifest with symptoms in the hematological, rheumatological, dermatological, and endocrine symptoms [4, 5], affecting 10–15% of cancer patients and causing 27% of deaths. Paraneoplastic syndromes are most commonly associated with lung, breast, thyroid (especially medullary), prostate, gynecological and hematological malignancies. These syndromes are equally common in both men and women [6].

Paraneoplastic syndromes arise from two main pathways: immunological, where the immune system attacks cancer cells but cross-reacts with normal tissues via antibodies or T-cells

targeting onconeural antigens, and non-immunological, where tumors secrete hormones, peptides, or cytokines [6]. Immunological PNS involve auto-antibodies or cell-mediated immunity, causing neurological damage, while non-immunological ones are hormonal/metabolic disorders (hypercalcemia, hyponatremia) [4].

Endocrine PNSs are divided into two large groups: those from peptide hormones, which are further divided into common and less common, and those from non-peptide hormones. Common syndromes include humoral hypercalcemia, syndrome of inappropriate antidiuretic hormone secretion (SIADH), Cushing's syndrome, and others. Less common syndromes include ectopic renin and β -human chorionic gonadotropin secretion [7].

Hyperkalemia in cancer patients stems from diverse causes, including excess renin secretion, causing hypertension and metabolic alkalosis [7], side effects of diuretic therapy in patients with SIADH syndrome [8], spontaneous tumor lysis syndrome in hematological and solid tumors, including blast crisis [9]. In addition to causes associated with PNS, hyperkalemia may occur in cancer patients with concomitant kidney disease and in those receiving nephrotoxic drugs (cisplatin,

gemcitabine, methotrexate, and others) [10,11].

Esophageal cancer (EC) is the eighth most common malignancy and the sixth leading cause of cancer-related deaths worldwide [12]. There are two histological types of esophageal cancer: adenocarcinoma, which is prevalent in many Western countries (USA, UK, Australia, Western Europe) and squamous cell carcinoma [13]. Esophageal cancer is often diagnosed at an advanced stage, with dysphagia being the most common symptom, leading to risky surgeries with high complication rates and early relapse. The optimal treatment strategy for stage II–III esophageal adenocarcinoma is perioperative chemotherapy with FLOT regimen, which increases the immediate treatment effectiveness and improves long-term results compared to CROSS chemoradiotherapy [14]. However, the FLOT regimen has side effects, which can be severe enough to prompt physicians to use FOLFOX or XELOX (oxaliplatin-based doublets with fluorouracil). Like other malignancies, EC can trigger PNSs, such as Bazex syndrome (paraneoplastic acrokeratosis), dermatomyositis, and syndrome of inappropriate secretion of antidiuretic hormone [13]. In esophageal adenocarcinoma, PNS is represented by a leukemoid reaction, acanthosis

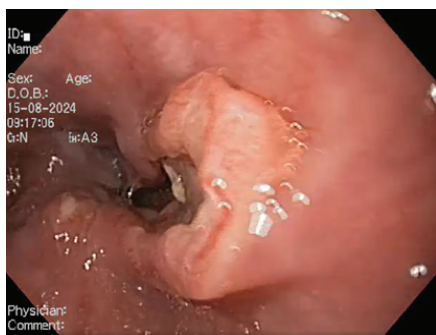


Fig. 1. Video esophagogastroscopy. Endoscopic image of an esophageal tumor: a flat infiltrative tumor is visualized at a level of 36 cm from the incisors, distorting the esophageal lumen and leading to stenosis of up to 0.5 cm.



Fig. 2. Contrast-enhanced abdominal CT. Axial section: a circular thickening of the wall of the lower thoracic esophagus due to tumor measuring up to 12 mm, with stenosis of the lumen.

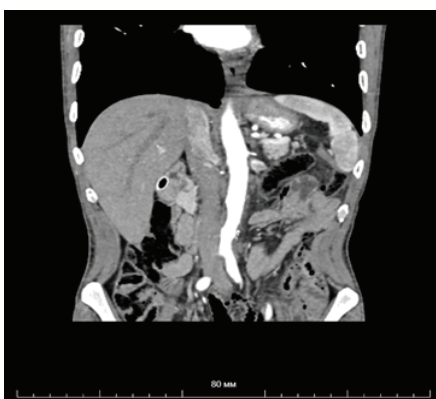


Fig. 3. Contrast-enhanced abdominal CT. Sagittal section: a tumor in the lower thoracic esophagus extending up to 52 mm is visualized.

nigricans, Sweet's syndrome (febrile neutrophilic dermatosis), thrombocytosis, and atypical Cushing's syndrome [13, 15, 16, 17].

Our review of the literature revealed insufficient data on the incidence of hyperkalemia in EC patients and the choice of treatment strategy for these patients. Here, we report a rare case of EC pre-

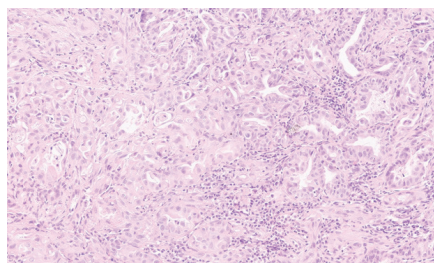


Fig. 4. Esophageal adenocarcinoma before treatment. The tumor consists of atypical glandular structures with pronounced cellular and nuclear atypia, disorganized architecture, and invasive growth. The tumor stroma is minimally expressed with lymphoid infiltration. Hematoxylin and eosin staining, magnification ×400.

senting with hyperkalemia. Written informed voluntary consent to participate in the study, publication of the description of the clinical case and photographs in a medical journal, including its electronic version, was obtained from the patient (01.12.2025).

Case presentation. A 64-year-old male patient presented to the Outpatient Department of the Cancer Research Institute with complaints of grade III dysphagia combined with 13 kg weight loss in 3 month. A video esophagogastroscopy with biopsy performed on August 15, 2024, revealed a flat, infiltrative tumor at 36 cm from the incisors, causing significant narrowing of the esophageal lumen (stenosis up to 0.5 cm) (Fig. 1). A contrast-enhanced computed tomography of the chest and abdomen from August, 2024 showed irregular thickening (12–23 mm) of the stomach's upper part (cardia/fundus) and lower esophagus (thoracic/abdominal), extending about 52mm. (Figs. 2, 3).

Histological examination of the biopsy specimen confirmed esophageal adenocarcinoma (Fig. 4).

Based on clinical and instrumental data, the patient was scheduled for perioperative chemotherapy using the FLOT regimen (4 courses of neoadjuvant and 4 courses of adjuvant chemotherapy) followed by Lewis-type surgery. However, hyperkalemia (6.15 mmol/L) was detected during hospitalization prior to treatment. Electrocardiographic examination revealed no rhythm disturbances. Furthermore, the patient's medical history revealed that he was not taking diuretics or potassium supplements. A nephrologist consultation ruled out kidney disease, and renin levels were within reference limits. Attempts to treat hyperkalemia with medication were unsuccessful.

An interdisciplinary consultation was held at the Abdominal Oncology Depart-

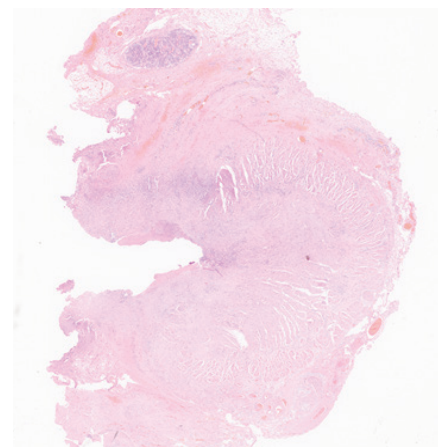
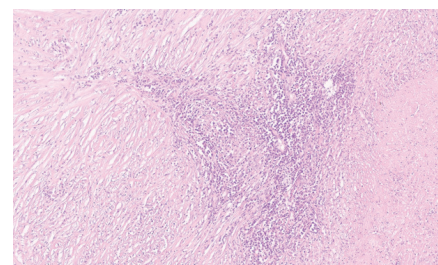
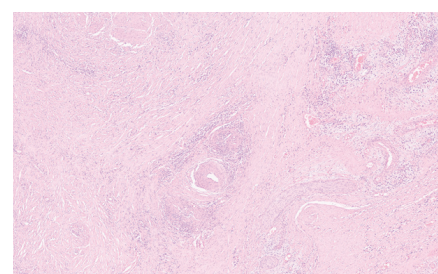


Fig. 5. Morphological changes in esophageal adenocarcinoma after treatment. Note. A – Image of the tumor bed after completion of treatment. Severe fibrosis, ulcerative mucosal defect, and areas of necrosis are visible. The absence of viable tumor tissue indicates complete pathological regression. Hematoxylin and eosin staining, whole slide imaging (Aperio), magnification ×40.



Note. B – Fragment of the tumor bed at higher magnification. Dense fibrous tissue, inflammatory infiltration, and foci of mucosal necrosis without signs of tumor cells are revealed, confirming complete pathological tumor regression (Mandard TRG 1). Hematoxylin and eosin staining, magnification ×200.



Note. C – Muscle layer of the gastric wall in the area of complete pathological tumor regression. Tumor cells are not detected. Angiomatosis, pronounced interstitial and perivascular fibrosis, fibrosis of the vessel walls, and lymphohistiocytic infiltration are seen. Hematoxylin and eosin staining, magnification ×100.

ment of the Cancer Research Institute with the participation of a surgeon, anesthesiologist, therapist, radiation therapist, and chemotherapist. Given the persistent increase in blood potassium levels (6.15

mmol/L from August 26, 2024; 6.25 mmol/L from September 4, 2024; 6.18 mmol/L from September 12, 2024; 6.40 mmol/L from September 18, 2024; 6.17 mmol/L from September 27, 2024), it was deemed impossible to administer either systemic chemotherapy or surgery. In this regard, palliative external beam radiation therapy was started on September 30, 2024 after inserting a gastrostomy tube.

External beam radiation therapy at a total dose of 50Gy resulted in normalization of blood potassium levels to 4.25 mmol/l (15.10.2024). After repeated consultation with the chemotherapist and evaluation of the performance status of the patient (ECOG 2), neoadjuvant FOLFOX chemotherapy with a 15% dose reduction was administered. Two courses of chemotherapy were complicated by grade II leukopenia and grade III hepatotoxicity; therefore, further treatment was discontinued.

Partial tumor regression was observed 8 weeks after completing external beam radiotherapy. On November, 2024, Lewis esophagectomy with esophagoplasty using a gastric graft and two-field lymph node dissection was performed. The postoperative period was uneventful; oral feeding was initiated on postoperative day 2, and the patient was discharged from the hospital on day 11. Pathological examination revealed complete regression according to Mandard (TRG 1). Post-therapy morphological changes in the tumor bed and the muscular layer of the gastric wall are shown in Fig. 5A–C.

Given the severe toxicity of neoadjuvant chemotherapy, the patient refused adjuvant treatment. A follow-up examination in May 2025 revealed no signs of recurrence or disease progression.

Discussion. Paraneoplastic syndromes are rare cancer-related disorders causing diverse symptoms in patients with lung, gynecological, and blood cancers [2, 4]. Paraneoplastic syndromes affect 15% of patients with cancer [2]. However, data on the incidence of PNSs in EC are scarce, making it hard to detect them early and choose optimal treatments.

The case report and the analysis of literary data demonstrate that hyperkalemia detected in the patient with EC can

be a PNS manifestation. Despite a comprehensive examination, the exact pathogenetic mechanism for the development of this condition was not established. One possible cause of hyperkalemia may be ectopic renin secretion by tumor cells, which was not confirmed by standard laboratory methods. However, there are reports on the disturbances in the regulation of the renin-angiotensin system in cancer patients, including those with rare tumor locations, suggesting the presence of similar mechanisms in EC [18].

Indirect confirmation of the paraneoplastic nature of hyperkalemia in our case is its resistance to drug therapy and subsequent normalization of potassium levels during external beam radiation therapy. This indicates a possible direct relationship between the tumor activity and the development of electrolyte disturbances, and emphasizes the role of local antitumor therapy in relieving systemic paraneoplastic manifestations.

In our case, the use of radiation therapy as an initial treatment and deviation from standard clinical guidelines should be considered a necessary, clinically justified strategy. Severe and persistent hyperkalemia was a serious limitation for both systemic chemotherapy and surgery, making alternative treatments the only viable options.

K.A. Goodman [19] reported on encouraging results of using FOLFOX chemotherapy combined with radiation therapy for EC patients, with the rate of pathological complete regression (pCR) of 40.3%. However, it should be noted that this treatment was administered to young patients with ECOG 0–1 performance status. Case reports and studies confirm that the combination of FOLFOX chemotherapy and targeted therapy/immunotherapy shows significant promise for achieving high objective response rate, even complete tumor regression. However, this treatment strategy should not automatically apply to patients with severe concomitant metabolic disorders [20]. In this context, the achievement of complete pathological tumor regression in our patient with ECOG 2 and severe hyperkalemia emphasizes the importance of a personalized treatment approach in complex clinical situations.

Conclusion. We described a rare case of paraneoplastic syndrome (PNS) in which esophageal adenocarcinoma triggered persistently elevated potassium levels (hyperkalemia), which seriously complicated cancer treatment by destabilizing the patient's condition. The use of external beam radiation therapy as a primary treatment resulted in stabilization of electrolyte imbalance and created the conditions for subsequent systemic therapy and radical surgery, ultimately leading to the patient's recovery.

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Difficulties in diagnosing peripartum cardiomyopathy: a case report

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Abstract. Peripartum cardiomyopathy is a rare idiopathic form of heart failure that occurs during late pregnancy or within a few months postpartum. This article presents a clinical case of a 39-year-old female patient with peripartum cardiomyopathy, including an analysis of the clinical presentation, diagnosis, and treatment. Objective: To describe a clinical case of peripartum cardiomyopathy in a 39-year-old female patient, to analyze the diagnostic difficulties, and to assess the patient's progression during therapy. Materials and methods: This study is based on an analysis of a clinical case of a female patient who was treated in a general hospital. Results and discussion: a previously healthy woman presented with heart failure (shortness of breath, edema, hydrothorax, and ascites) and a significant decrease in left ventricular ejection fraction (to 30-34%) with dilation of the left ventricular cavity. After excluding possible rheumatic, endocrine, and infectious causes of this condition, a diagnosis of PPCM was made. Follow-up observation revealed incomplete symptomatic resolution despite treatment. Positive dynamics were noted, including resolution of edema and an increase in left ventricular ejection fraction from 34% to 50%. Signs of heart failure (shortness of breath with moderate physical exertion, palpitations) persisted in the clinical picture, and cardiac arrhythmia developed. Despite the positive dynamics, the patient continued to have significant myocardial dysfunction, demonstrating the variability of the prognosis in PPCM and confirming the need for a multidisciplinary approach to improve outcomes and reduce maternal mortality.

Key words: peripartum cardiomyopathy, heart failure, left ventricular dysfunction, pregnancy, postpartum period.

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Peripartum cardiomyopathy (PPCM) is a form of systolic heart failure characterized by a decrease in left ventricular ejection fraction below 45% [12]. This condition occurs in young women of reproductive age during pregnancy or immediately after childbirth [7,10,11]. The disease is most common in South Africa and Haiti, where the incidence is 1 case in every 100 and 300 births, respectively. In Japan, this figure is significantly lower—only 1 in 20,000 births. In the United States, rates range from 1:1000 to 1:4000 births. South Korea has a rate of 1 in 1741 births, and Germany has a rate of 1 in 1500 births. Statistical data on the prevalence of PPCM in the Russian Federation are currently unavailable;

only isolated cases have been described [1,4,9].

To date, the risk factors and causes of PPCM remain poorly understood. However, the risk of developing PPCM is known to increase in women over 30 years of age or during early pregnancy, with repeated births, the development of hypertension during pregnancy and pre-eclampsia, anemia, the use of tocolytic therapy, and in vitro fertilization [8, 13].

The clinical presentation of PPCM is varied and can result in complete recovery, persistent myocardial dysfunction, heart failure, and rapid deterioration, leading to an acute need for temporary or long-term mechanical circulatory support and heart transplantation [9].

It is noted that the prognosis for PPCM is better than for other types of cardiomyopathy. Complete recovery of myocardial function occurs in approximately 23–54% of patients with PPCM [1]. The process of restoration of cardiac function typically lasts from three months to two years. The mortality rate among women with PPCM varies depending on the duration of observation: six months after the onset of the disease, mortality is 2–12.6%, after one year – 4–14%, and after 19 months – approximately 7% [2].

Regardless of the diagnostic criteria, PPCM often remains under the radar of medical professionals or is misdiagnosed. This disease is characterized by a rarity of manifestation, as a result