Massive saddle pulmonary embolism during radiochemotherapy of head and neck cancer

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Thromboembolic complications (TC) are common in the course of some cancers, such as lung, gastrointestinal, and gynecologic cancers, as well as brain tumors.[1] Surgical treatment of patients with head and neck cancer is an important risk factor for TC.[2,3] However, data on the incidence of TC in nonsurgically treated patients with head and neck cancer are scarce.

A 65-year-old patient with low-differentiated carcinoma (FIGURE 1A–1C) of the head and neck region, with multiple bilateral metastatic lymph nodes in the neck (TxN3M0 according to the TNM classification; FIGURE 1D–1F), was hospitalized to undergo megavoltage 3-dimensional radiotherapy (70 Gy/35 fractions delivered in 7 weeks) with 3 courses of concomitant chemotherapy. The comorbidities included type 2 diabetes, arterial hypertension, and obesity. From the beginning of the therapy, the patient showed fluctuating glucose levels; therefore, insulin therapy along with the regulation and close monitoring of blood glucose levels were introduced. Because of persistent vomiting, the patient was administered antiemetic agents, parenteral hydration, and electrolyte supplementation. He remained mobile. After 2 weeks of radiotherapy, he developed a progressive acute inflammatory radiation reaction in the mucous and skin of the head and neck

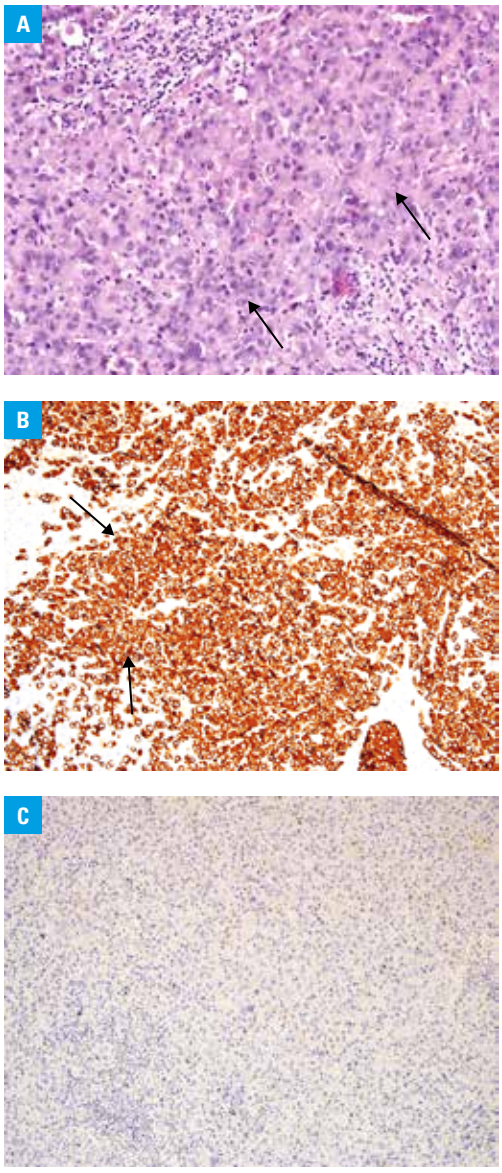

FIGURE 1 A 65-year-old man with unknown primary cancer of the head and neck (TxN3M0) treated nonsurgically; **A** – hematoxylin and eosin staining indicating cancer cells (arrows); magnification, ~400×; **B** – strong staining (brown color) for cytokeratin 7, indicating the presence of carcinoma (arrows); magnification, ~200×; **C** – negative staining for p63, excluding squamous cell carcinoma; magnification, ~200×

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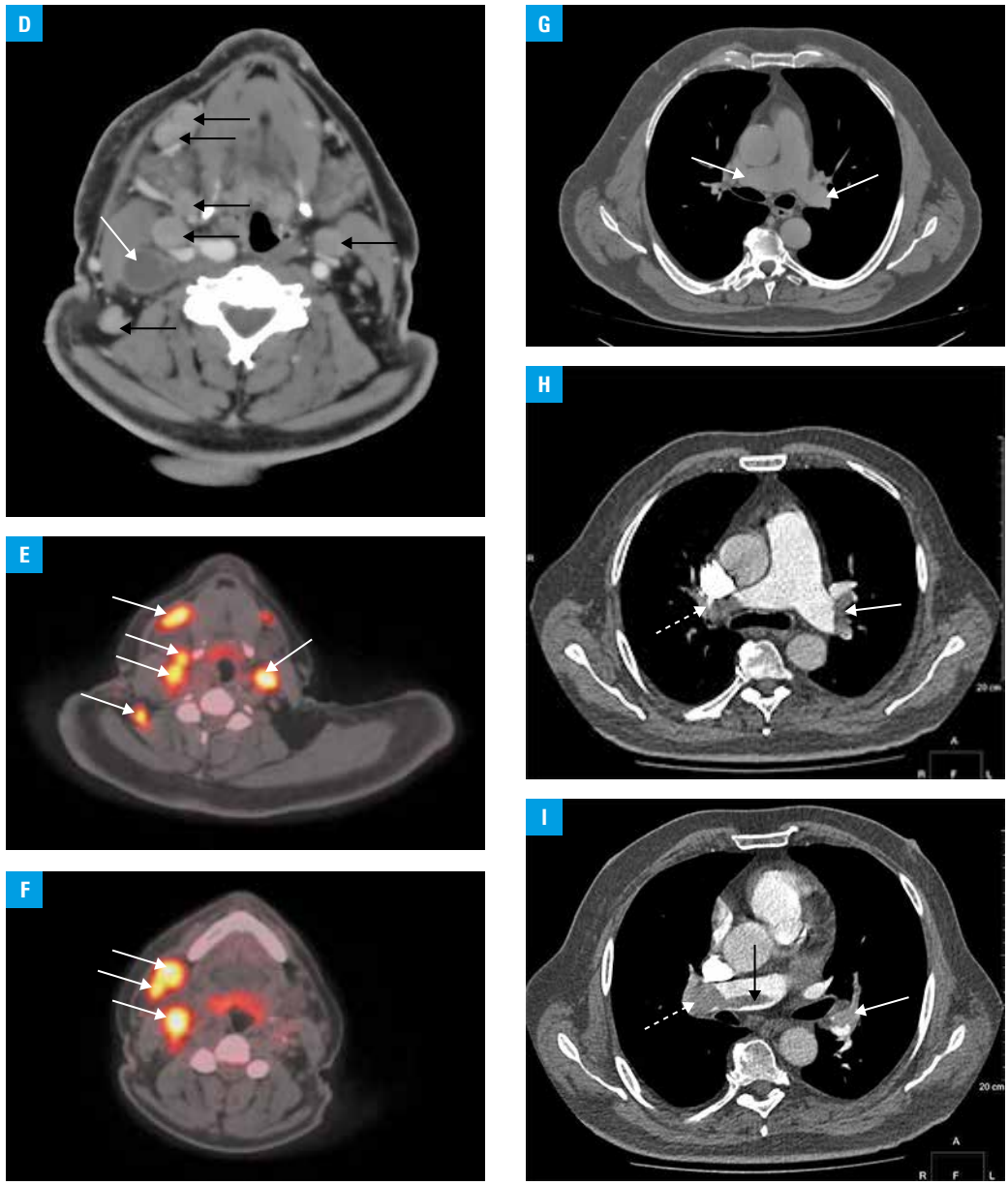

FIGURE 1 A 65-year-old man with unknown primary cancer of the head and neck (TxN3M0; TNM classification) treated nonsurgically; **D** – neck computed tomography (CT) scan; multiple bilateral metastatic lymph nodes (black arrows); necrosis in a metastatic lymph node (white arrow); **E**, **F** – 18-fluorodeoxyglucose positron emission tomography–computed tomography showing high glucose uptake (active metabolic process) in metastatic lymph nodes in the neck (arrows); **G** – a pretreatment chest CT scan with normal pulmonary arteries (white color indicated by arrows); **H**, **I** – a chest CT angiography scan with iodine contrast agent, showing multiple filling defects (dark grey color in contrast to normal white) both at the bifurcation of the pulmonary trunk and in the pulmonary arteries (saddle pulmonary embolism [PE]); full white arrow, PE in the left pulmonary artery; dotted white arrow, PE in the right pulmonary artery; black arrow, PE in the bifurcation of the pulmonary trunk.

region. He received antibiotics, analgesics, and antifungal agents. After the fourth week of radiotherapy and the second course of chemotherapy, he was weak, sleepy, and spent most of the time in bed. After the fifth week of radiotherapy, he fainted with a short-term loss of consciousness. Neutropenia, thrombocytopenia, and hypoalbuminemia were observed, and granulocyte colony-stimulating factor, cyclonamine, and protein supplementation were administered. Ten days later, after the third course of chemotherapy, the patient fainted again, with a short loss of contact, involuntary urination, low arterial pressure, and tachycardia. Cardiac enzyme levels and the results of brain computed tomography were normal. Because of weakness, acute inflammatory reaction, and immobility, dalteparin at a prophylactic dose of 5000 IU subcutaneously (SC) once daily was introduced. Six days later, the patient fainted for the third time, with tachycardia, slightly increased blood pressure, increased D-dimer levels, and a decline in arterial oxygen saturation. Chest computed tomography angiography showed a massive saddle pulmonary embolism (PE; FIGURE 1G–1I). The patient was transferred to a cardiology unit and received a therapeutic dose

of enoxaparin (120 mg SC once daily). His clinical status improved, and radiotherapy was discontinued. A month later, he fainted again and was hospitalized due to heart insufficiency. A full dose of low-molecular-weight heparin (LMWH) was continued. After 2 months, cancer progression was observed, and the patient underwent systemic palliative treatment. Six months after PE, he developed severe neutropenia and his general condition deteriorated. He was referred for supportive care, while still receiving therapeutic doses of LMWH.

Although the patient suffered from cancer characterized by low risk of TC and was not treated surgically, and despite thromboprophylaxis, he developed saddle PE. Radiotherapy and chemotherapy cause damage to cancer cells with the release of the main procoagulant, tissue factor. This process, together with comorbidities and risk factors such as radiochemotherapy, unstable glucose levels, inflammatory reaction, infection, neutropenia, and immobility, may lead to PE.[1,4] Physicians caring for nonsurgically treated cancer patients should be aware of the risk of TC and remember that the risk is reduced, but not eliminated, by thromboprophylaxis.

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