CLINICAL IMAGE

Tumor of the Turkish saddle with endocrine disorders as the first manifestation of gastric adenocarcinoma

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A 65-year-old woman was admitted to the department of endocrinology due to an abnormal intrasellar and suprasellar mass found a month earlier. A pathological mass (20 × 14 × 16 mm in diameter) was revealed by magnetic resonance imaging, which was performed because of a sudden onset of diplopia, ptosis of the left palpebra, and cranial nerve palsy. On admission, physical examination revealed cachexia and paresis of the oculomotor (III) and abducens (VI) nerves. Hormonal tests were performed (Supplementary material, *Table S1*). Because of weight loss of 10 kg in the preceding 3 months, tumor markers (CEA, CA 125, CA 19–9, and CA 15–3) were also measured. Owing to a significant elevation of these markers, we performed ^{18}F-fluorodeoxyglucose positron emission tomography integrated with computed tomography (^{18}F-FDG PET/CT) (FIGURE 1A and 1B), showing enlarged and metabolically active lymph nodes on both sides of the diaphragm, and focal lesions in the liver and pituitary gland. This raised a suspicion of the lympho- or myeloproliferative disease or malignancy arising from the gastrointestinal tract. A histological examination of a supraclavicular lymph node revealed metastases of adenocarcinoma. Gastroscopy showed neoplastic infiltration of the mucosa of the esophagus, cardia, and main body of the stomach. A histological examination confirmed adenocarcinoma G2. The patient was first referred for stereotactic radiotherapy of the pituitary tumor. Unfortunately, she died a few months later due to cancer dissemination.

Metastases to the sella turcica area and pituitary gland are rare and represent about 1% of

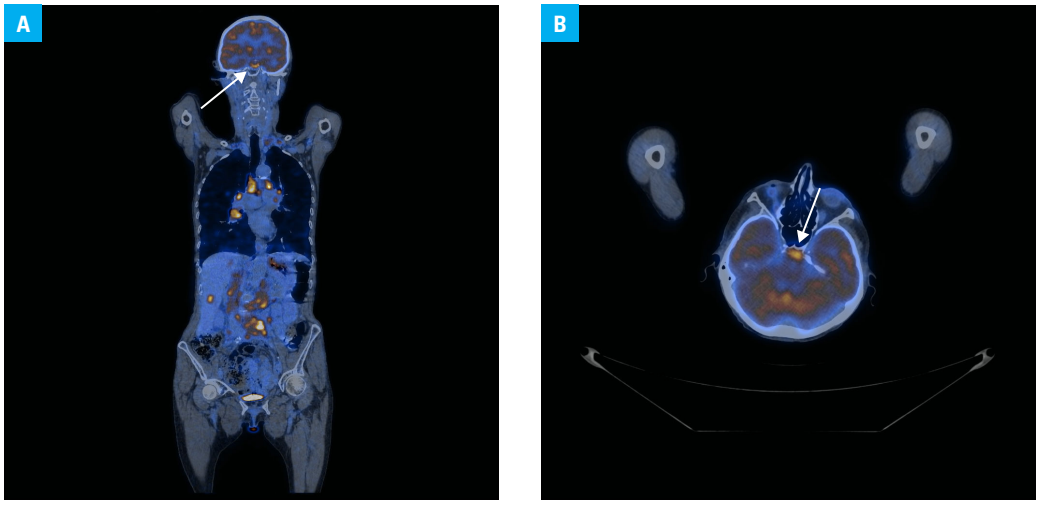

FIGURE 1 ^{18}F-fluorodeoxyglucose positron emission tomography integrated with computed tomography: **A** – enlarged and metabolically active lymph nodes on both sides of the diaphragm (arrow) and focal changes in the liver and pituitary gland (arrow); **B** – a metabolically active pathological mass in the pituitary gland

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all metastases to the brain. Pituitary metastases were found in 1.9%[1] and 5.1%[2] of the patients with cancer on autopsy. In the majority of cases, the primary sites were breast and lung cancers.[3] Gastrointestinal tumors constitute 6.4% of all pituitary metastases.[4] Most of these reports were accidental autopsy findings with no symptoms, although about 30% of pituitary metastases were the first manifestation of an unknown primary tumor, as in our patient.[1]

Generally, pituitary metastases are located in the posterior lobe, and the most commonly observed symptom is diabetes insipidus.[1,2] Anterior hypopituitarism, visual loss, paresis of the cranial nerves, or headaches are less frequently reported. A rare finding is Cushing syndrome or acromegaly as the result of hyperfunction. Also single cases of adrenocorticotropic hormone (ACTH)-dependent syndrome originating from primary tumors with ectopic ACTH secretion were reported.[5] Coexisting hyperprolactinemia (prolactin >200 ng/ml) is most likely caused by prolactinoma rather than a stalk compression, although Kalamnos et al[2] reported cases where high prolactin levels were due to compression,[2] as in our case.

Pituitary metastasis can mimic primary benign pituitary adenoma. He et al[1] reported that the involvement of the posterior lobe, presence of diabetes insipidus or cranial nerve paresis rather than hypopituitarism, older age, and fast onset of symptoms coexisting with systemic symptoms (weakness, weight loss) can indicate malignancy. Surgery, radiotherapy, and chemotherapy can diminish the size of pituitary metastasis and relieves symptoms. However, the outcomes are poor. Most patients have distant metastases at the time of diagnosis and most of them die within a year from diagnosis.[1,2,4,5] The main aim of the therapy is improvement of the quality of life.

SUPPLEMENTARY MATERIAL Supplementary material is available with the article at www.pamw.pl.

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