

Martyna Ziemińska<sup>1</sup>, Aldona Ząber<sup>1</sup>, Andrzej Mazurek<sup>2</sup>, Bartłomiej Grala<sup>3</sup>, Renata Duchnowska<sup>1</sup>

<sup>1</sup>Department of Oncology, Military Institute of Medicine, National Research Institute, Warsaw, Poland

<sup>2</sup>Department of Nuclear Medicine, Military Institute of Medicine, National Research Institute, Warsaw, Poland

<sup>3</sup>Department of Pathology, Military Institute of Medicine, National Research Institute, Warsaw, Poland

## Serous cystadenocarcinoma of the pancreas with synchronous breast cancer

**Key words:** breast cancer, pancreas, serous cystadenocarcinoma

A 77-year-old female was admitted to an acute surgical service for the management of suspected gastrointestinal bleeding. She was in moderately severe condition (Eastern Cooperative Oncology Group Performance Scale 3) with stable vital signs. Blood tests revealed a hemoglobin level of 5 g/dl (range 11.0–18.0 g/dl), hematocrit of 15% (range 35–55%), red blood cell count of  $1.66 \times 10^{12}/l$  (range  $3.5\text{--}5.5 \times 10^{12}/l$ ), platelet count of  $371 \times 10^9/l$  (range  $150\text{--}400 \times 10^9/l$ ), and white blood cell count of  $14.89 \times 10^9/l$  (range  $4.0\text{--}10.0 \times 10^9/l$ ). On physical exam, a tumor in the left breast and a non-moving mass in the epigastrium were palpated. Endoscopy showed moderate erythematous-exudative gastropathy and ulceration in the subcardiac area. Cold saline with adrenaline was locally administered, and red blood cell concentrates were transfused. Additionally, intravenous administration of tranexamic acid, ethamsylate, and pantoprazole was initiated. Mammography showed a tumor size of  $28 \times 27$  mm in the left breast classified as Breast Imaging-Reporting and Data System (BI-RADS) 5. The biopsy revealed an invasive carcinoma, intermediate grade G2 in immunohistochemistry (IHC) staining: estrogen receptor-positive with a strong reaction in more than 90% of the cells (Allred 8), progesterone receptor-negative, human epidermal growth factor type 2 negative (IHC 1+), and Ki67 15%.

Furthermore, contrast-enhanced computed tomography (CT) of the abdominal cavity demonstrated extensive solid-cystic hyperplasia with small calcifications ( $135 \times 92 \times 153$  mm) originating from the tail of the pancreas. The lesion extended from the fundus of the stomach to the left kidney and infiltrated the surrounding tissues of the spleen and stomach. One suspected lesion in segment 8 of the liver was detected. A biopsy of the lesion in the pancreas was conducted and demonstrated clear cell carcinoma with CKAE1/AE3+, vimentin +, RCC+/-, CD10 (-), CK7 (-), WT1 (-), PR (-), ER (-), mammoglobin (-), GCDFP15 (-), p53 (-). The IHC staining indicated that the origin of the disease is likely to be either the kidney or a reproductive organ.

Considering the advanced stage of the disease and unknown primary origin, the patient was initially qualified for induction chemotherapy with paclitaxel and carboplatin. However, the positron emission tomography (PET-CT) (separately from the breast tumor) showed a lesion with low metabolic activity originating from the pancreas or spleen (Fig. 1A–C). Based on the PET-CT result, the patient was qualified for upper midline laparotomy. A peripheral resection of the pancreas with splenectomy and segmental resection of the colon was performed. The histopathological examination revealed a spongy litho-cystic

Received: 25.01.2023 Accepted: 06.03.2023 Early publication date: 04.08.2023

This article is available in open access under Creative Common Attribution-Non-Commercial-No Derivatives 4.0 International (CC BY-NC-ND 4.0) license, allowing to download articles and share them with others as long as they credit the authors and the publisher, but without permission to change them in any way or use them commercially.

**Address for correspondence:** Prof. Renata Duchnowska, Department of Oncology, Military Institute of Medicine, National Research Institute, ul. Szaserów 128, 04-141 Warsaw, Poland, e-mail: rdt@wp.pl

Oncol Clin Pract, DOI: 10.5603/OCP.2023.0043, Copyright © 2023 Via Medica, ISSN 2450-1654, e-ISSN 2450-6478



**Figure 1.** Positron emission tomography with 2-deoxy-2-[fluorine-18]fluoro-D-glucose ( $^{18}\text{F}$ -FDG PET/CT); nodular lesion 141 × 76 mm; CC 115 mm with mediocre  $^{18}\text{F}$ -FDG metabolism; lesion involves the splenic hilum, tail of the pancreas, adjacent to the stomach, the descending colon and the left adrenal gland; focus of increased radiolabel accumulation in the left breast; **A.** Coronal scan; **B.** Sagittal scan; **C.** Axial scan

tumor, size of 128 × 135 × 68 mm, involving a portion of the pancreas, spleen, and subserosal tissues of the large intestine. The morphological characteristics were consistent with a microcystic serous cystadenocarcinoma (SCAC) of the pancreas, with IHC staining: CKAE1/AE3 (+), CK19 (+), CK7 (+) focal, inhibinA (+), vimentin (-), RCC (-), CD10 (-), CK5/6 (-), calretinin (-), WT-1 (-), CD117 (-), MelanA (-), HMB-45 (-), CD4 (-), CD31 (-), CD34 (-), ER (-), PR (-); Ki67 about 5%. Splenic infiltration, angioinvasion, and satellite nodules in the retroperitoneal space were present, while the surgical margin was free of cancer cells (R0 resection).

Approximately eight weeks later, a radical mastectomy with a sentinel node biopsy was performed. The histopathological examination showed invasive breast cancer of no special type, intermediate grade G2, with emboli in blood vessels, TNM staging pT2pN0(sn), L/V1, R0. Considering the patient's age, overall health condition, and preferences, hormone therapy with tamoxifen was started. There were no signs or evidence of disease recurrence during the 4-year follow-up.

## Discussion

Synchronous primaries are diagnosed in approximately 20% of cancer patients, mainly in the group over the age of 50. In patients with breast cancer (BC), the common synchronous neoplasms include contralateral BC and gynecologic cancers [1]. SCAC is a very rare tumor and usually occurs in women between the age of 50 and 70. The course of the disease is often asymptomatic. In the case of advanced SCAC, the most common symptoms are abdominal pain, upper gastrointestinal bleeding, weight loss, a palpable tumor or elevated transaminases, and rarely jaundice or pancreatitis [2, 3].

Diagnosis of SCAC by biopsy is difficult because of its similarity to the cells of benign serous cystic neoplasms. Malignant tumors are usually larger, locally invasive, and with distant metastases. Furthermore, they often infiltrate locally adjacent vessels, nerves, spleen, stomach, and duodenum. The differential diagnosis of SCAC should include clear cell carcinomas of the ovary or kidney [2].

The surgery is of crucial importance in SCAC, also in older patients [4]. The excellent prognosis associated with SCAC, even in the case of distant metastases, justifies an aggressive surgical approach. In turn, systemic therapy in SCAC has no proven effect [4]. The occurrence of synchronous primary tumors is always a challenge and often causes dilemmas in clinical practice. The therapeutic regimen should be decided by multidisciplinary teams, considering the patient's general condition, expectations, prognosis, and quality of life.

## Article Information and Declarations

### Ethics statement

Retrospective description. The patient gave consent. Data anonymized.

### Author contributions

M.Z., A.Z., R.D.: conception and design.

All authors: provision of study materials, collection and assembly of data, data analysis and interpretation, manuscript writing, final approval of manuscript.

### Acknowledgments

Jack Trepto, MD, PhD for his assistance in the diagnostic process.

### Conflict of interest

Authors declare no conflict of interest.

## Funding

None.

## Supplementary material

None.

## References

1. Tanjak P, Suktitipat B, Vorasan N, et al. Risks and cancer associations of metachronous and synchronous multiple primary cancers: a 25-year retrospective study. *BMC Cancer*. 2021; 21(1): 1045, doi: [10.1186/s12885-021-08766-9](https://doi.org/10.1186/s12885-021-08766-9), indexed in Pubmed: [34556087](https://pubmed.ncbi.nlm.nih.gov/34556087/).
2. King JC, Ng TT, White SC, et al. Pancreatic serous cystadenocarcinoma: a case report and review of the literature. *J Gastrointest Surg*. 2009; 13(10): 1864–1868, doi: [10.1007/s11605-009-0926-3](https://doi.org/10.1007/s11605-009-0926-3), indexed in Pubmed: [19459016](https://pubmed.ncbi.nlm.nih.gov/19459016/).
3. Massaras D, Pantiora EV, Koutalas J, et al. Serous Microcystic Cystadenocarcinoma of the Pancreas with Synchronous Liver Metastases: Clinical Characteristics and Management. *Cureus*. 2020; 12(4): e7707, doi: [10.7759/cureus.7707](https://doi.org/10.7759/cureus.7707), indexed in Pubmed: [32431986](https://pubmed.ncbi.nlm.nih.gov/32431986/).
4. Wasel BA, Keough V, Huang WY, et al. Histological percutaneous diagnosis of stage IV microcystic serous cystadenocarcinoma of the pancreas. *BMJ Case Rep*. 2013; 2013, doi: [10.1136/bcr-2012-007924](https://doi.org/10.1136/bcr-2012-007924), indexed in Pubmed: [23370947](https://pubmed.ncbi.nlm.nih.gov/23370947/).