Pseudocirrhosis Following Great Response to CDK4/6 Inhibitor and Endocrine Therapy in a Premenopausal Patient With HR+/HER2- Metastatic Breast Cancer

Mariana Carvalho Gouveia[1], Rafaela Melo Campos Borges[1], Pedro Zanuncio[2], Bruna Migliavacca Zucchetti[1]

1- Hospital 9 de Julho- Américas Oncologia, São Paulo, Brazil; 2- Hospital Beneficência Portuguesa, São Paulo, Brazil.

Introduction

CDK4/6 inhibitors have revolutionized the management of hormone receptor–positive, HER2-negative (HR+/HER2–) metastatic breast cancer (MBC), improving survival across pre- and postmenopausal populations. In premenopausal women, their combination with ovarian suppression and endocrine therapy is the standard first-line regimen.

Liver metastases occur in up to 50% of patients with MBC, and pseudocirrhosis — a radiologic mimic of cirrhosis — has been reported in approximately 5–15% of those with hepatic involvement. While traditionally associated with chemotherapy, pseudocirrhosis is rarely reported with CDK4/6 inhibitors. Recognizing this entity is crucial to avoid misdiagnosis with disease progression and unnecessary treatment changes.

Methods

We report a case of pseudocirrhosis in a 43-year-old premenopausal woman with HR+/HER2– MBC and high hepatic tumor burden at diagnosis who achieved a robust response to first-line abemaciclib plus endocrine therapy. Data were collected retrospectively from electronic records with patient consent for publication.

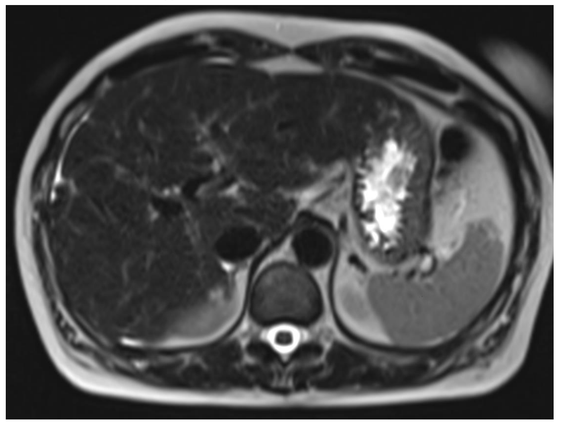
Case description

She was diagnosed in August 2022 with grade 3 invasive breast carcinoma of no special type, ER 98%, PR 2%, HER2-negative, Ki-67 85%, and extensive liver involvement. Treatment was initiated with tamoxifen plus abemaciclib, transitioning to letrozole following GnRH agonist. Molecular testing revealed no somatic or germline mutations. She experienced radiologic complete response, with only a solitary liver lesion progression in February 2025, which was treated with stereotatic body radiation therapy.

In July 2025, routine labs revealed elevated liver enzymes without clinical symptoms. Laboratory tests showed: ALT 60 (Reference value – RV: 13-35); AST 43 (RV:10-49), GGT 510 (RV<38), ALP 232 (RV: 46-116). Comprehensive evaluation excluded viral, autoimmune, or drug-induced hepatitis, and imaging suggested morphologic features of cirrhosis, without any evidence of disease progression. Despite imaging findings, the patient had no signs of portal hypertension or liver failure. The diagnosis of pseudocirrhosis has been done and started with ursodeoxycholic acid 300 mg BID and vitamin E 800 mg daily to avoid its progression. She continues on first-line treatment, with stable disease and improvement in liver enzymes.



August/2022: Liver metastases



July/2025: Liver with complete response, irregular contour and widened fissures

Discussion

Pseudocirrhosis refers to radiologic changes in the liver—such as capsular retraction, nodularity, and caudate lobe hypertrophy—mimicking true cirrhosis, but without histologic confirmation of fibrosis. It has been reported most frequently in breast cancer, particularly in patients with hepatic metastases undergoing chemotherapy. While the exact mechanism is unclear, proposed explanations include extensive tumor regression leading to capsular retraction, nodular regenerative hyperplasia, desmoplastic reaction, and hepatocellular injury due to oxidative stress from rapid tumor lysis. Despite resembling chronic liver disease radiographically, pseudocirrhosis often lacks clinical features of decompensated cirrhosis, making the correct diagnosis crucial. Importantly, up to 75% of women with metastatic breast cancer may exhibit some degree of liver contour irregularity on imaging, underscoring the need for oncologists to correlate radiologic changes with treatment history and clinical context.

Conclusion

This case highlights pseudocirrhosis as a rare but important differential diagnosis in patients with extensive liver metastases responding to systemic therapy. Awareness of this entity can prevent misinterpretation of imaging as disease progression or cirrhosis and help guide appropriate management without unnecessary treatment changes.

Contact

Bruna M. Zucchetti
Hospital 9 de Julho- Rede Américas Oncologia , São Paulo, Brazil
Email: brunamzucchetti@gmail.com
Phone: +55 11 910615584

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