findings consistent with CD multicentric, plasmablastic type. A vigorous search for monoclonal immunoglobulin was negative. Skeletal x-rays showed osteopenia. There was no infection with HHV-8 or HIV.

After 6 cycles of CHOP regimen there was dramatic improvement of the systemic symptoms, the lymphadenopathy and, surprisingly, the dermatopathy. He remained symptom-free for 2 years and then he presented with the same signs, symptoms and laboratory findings. A new biopsy confirmed the initial diagnosis of CD. Repeated virology tests were negative for HHV-8 and HIV and still no M-protein was detected. He was re-treated with 6 courses of R-CHOP but while most symptoms resolved the patient complained of extreme fatigue. Two months after the last course of chemotherapy physical examination revealed a small hard inguinal lymph node. Histological examination showed adenocarcinoma and despite a thorough search the primary site was not found. The patient's condition deteriorated and he deceased in a rapidly debilitating condition.

This case is exceptional in several ways: The patient presented with massive lymphadenopathy which is usually attributed to malignant lymphoproliferative disorders. The diagnosis of CD was a kind of – pleasant- surprise, although in a few cases CD can behave in a malignant way and it has to be treated with conventional chemotherapy. He had a chronic dermatopathy, i.e. a chronic antigenic stimulus, which could be associated with the development of CD. Also, both at initial diagnosis and during the relapse he presented several signs of POEMS syndrome such as polyneuropathy which resolved after chemotherapy. However, despite an elaborate search no monoclonal protein could be detected and the diagnosis of POEMS had to be rejected. Finally, the patient died from an unrelated cause, a solid tumor. There are case reports of solid tumors arising in patients with CD, such as sarcomas, vascular neoplasms, Kaposi's sarcoma, pheochromocytoma, and carcinoma of the rectum, kidney and thyroid gland, which presented either simultaneously or long after the initial diagnosis [5]. In this case the tumor probably developed while the patient was treated for CD and it was highly aggressive.

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Synchronous presentation of breast cancer and hepatocellular carcinoma in a postmenopausal woman: Dual action of aromatase inhibitor on breast and hepatic cancer cells?

Dear Editor,

A 75-year-old woman presented with a lump 2 cm in size in the outer-upper quadrant of the right breast. Tru-cut biopsy revealed infiltrating ductal carcinoma, grade I, estrogen (ER) and progesterone receptor (PR) positive and HER-2 negative. At the same time abdominal ultrasonography revealed a 2 cm mass in the right lobe of liver. Her liver function tests were within normal limits. Viral hepatitis markers and tumor markers including CA 15-3 and alpha fetoprotein (AFP) were within normal range. She was treated with breast-conserving surgery and axillary nodal dissection. Tumor stage was T2N0M0. At the same session of operation, the liver mass was excised with clear surgical margin and diagnosed as hepatocellular carcinoma (HCC). She was put on adjuvant aromatase inhibitor and had an appointment for adjuvant radiotherapy to the breast.

Current randomized studies showed that aromatase inhibitors have demonstrated superiority over tamoxifen in terms of disease-free survival and recurrence as initial therapy in postmenopausal women with hormone receptor positive breast cancer [1]. HCC represents the sixth leading cancer and the third most common cause of death from cancer worldwide. Experimentally, estrogens are involved in stimulating hepatocyte proliferation *in vitro* and may act as liver tumor inducers or promoters *in vivo* [2].

Inefficacy of the anti-estrogen tamoxifen in the management of HCC, however, could be partly attributable to the presence of variant ER forms, or to alternative, non-receptor mechanisms potentially involved in growth regulation of both normal and cancer human liver cells by estrogen [3]. One study has investigated the activity and expression of aromatase enzyme in non-tumoral, cirrhotic, and malignant human liver tissues and cells using both chromatographic and reverse transcription-PCR analyses. The authors reported that human HCC tissues showed elevated aromatase activity with increased estrogen formation rates, as opposed to non-tumoral hepatic tissues [4]. Therefore, aromatase inhibitors may also be considered in the management of HCC [5]. In our case aromatase inhibitor was given as adjuvant treatment for breast cancer. Given the above information, aromatase inhibitor may also be effective as an adjuvant treatment for HCC. This assumption needs to be clarified in clinical trials.

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