

DIAGNOSIS

The large pleomorphic cells are positive for CD30, CD2, CD3, CD4, CD5, CD43, granzyme B, EMA and TIA1. They are negative for ALK-1, CD8, CD20, CD79a, PAX5 and CD68. The combined histological, immune phenotypical and clinical features are most consistent with the diagnosis of breast implant-associated anaplastic large-cell lymphoma (BI-ALCL), without invasion of the breast tissue. Additional positron emission tomography/computed tomography-scan and bone marrow examination showed no signs of additional lymphoma localisations.

The prevalence of women with breast implants is increasing worldwide. In 2016 the World Health Organization recognized BI-ALCL as a separate entity.¹ Median time of diagnosis after implantation is 9 to 11 years. The relative risk for women with breast implants to develop BI-ALCL is 421.8 and the absolute risk is 1 per 7000 at age 75.² Cytological analysis is the cornerstone for diagnosing BI-ALCL. The smears demonstrate large pleomorphic lymphoid cells, often with an epithelioid appearance with plenty cytoplasm and large pleomorphic, occasionally kidney-shaped nuclei with a usually prominent nucleolus. After surgical removal of the implants and capsules, histopathologic examination is mandatory to estimate infiltrative growth. Lymphoma cells are mostly confined to the seroma fluid, without invasion of the capsule and the adjacent tissues.² BI-ALCL cells are CD30 positive and ALK-1 negative whereas ALK-1 is expressed in more than half of systemic ALCL cases. In addition, tumor cells frequently express EMA, CD2, CD3, CD4, CD43 and CD45 and rarely express CD5, CD7 or CD8. The pathogenic mechanism leading to the development of BI-ALCL remains to be elucidated. Some studies suggest a higher change on BI-ALCL with textured implants than with smooth implants, but they also note that the use of smooth implants is very limited.^{2,3}

Otherwise, chronic inflammation in the biofilm on the implant surface, immune response, repeated trauma of the rough implant surface and the patient's genetic makeup are thought to be associated with the development of BI-ALCL.

After diagnosing BI-ALCL, standard lymphoma workup is recommended. Without capsule invasion, conservative management is advised, with removal of the implant and capsule, as was done in this patient.³ In advanced cases, with invasion through the capsule, systemic chemotherapy is advised, where CHOP (cyclophosphamide, doxorubicin, vincristine and prednisone) and CHOP-like treatment are the most commonly used protocols. One study showed a 100% overall survival after 18 months of follow-up in cases of non-invasive, in-situ BI-ALCL, in contrast to 52.5% in advanced, infiltrative cases.⁴ Our patient underwent uncomplicated surgery and will be followed regularly at the outpatient clinic. In retrospect it is likely that the swelling after the first implants was an indication for the development of BI-ALCL, although the cytological puncture showed no abnormalities.

In conclusion, BI-ALCL is recognised as new entity and with an increasing incidence of breast implants BI-ALCL should be considered when swelling of the breast occurs.

REFERENCES

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