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Case Report

Primary osseous Burkitt lymphoma with nodal and intracardiac metastases in a child

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ABSTRACT

Burkitt lymphoma (BL) is the most frequent non-Hodgkin lymphoma in pediatric patients, accounting for approximately 34% of the cases of lymphoma in children. This subtype of non-Hodgkin lymphoma was first described in 1958 as a monoclonal proliferation of B cell lymphocytes. Cardiac involvement of BL in association with osseous compromise and lymphadenopathy is rare and poorly documented. We report a case of femur primary BL in an 8-year-old boy with metastatic cardiac involvement, retroperitoneal and iliofemoral lymphadenopathy, and hepatosplenomegaly. We highlight the diagnostic challenge in a patient with clinical nonspecific findings and systemic disease.

Clinical case

An 8-year-old boy was referred to our hospital with a 1-month history of fever and a growing mass in the left inguinal region. Physicians first treated the mass as an infectious adenitis without satisfactory evolution. During the physical examination, a 10 × 5 cm, hard, mobile, and not very painful mass was found in the left inguinal region. In addition, the patient had a grade III/IV systolic heart murmur with inspiratory splitting of s2. Magnetic resonance (MR) of the left thigh, abdominal ultrasound, and echocardiography was ordered.

MR showed multiple lymphadenopathies forming conglomerates of 7.5 × 5.8 × 7 cm in size that surrounded the iliac vessels, the inguinal, and the left popliteal regions. The femoral lymph nodes were greatly increased in size with inflammatory changes. Incidentally, an aggressive soft tissue mass was identified in the left distal femoral metaphysis (Fig. 1). Complementary knee X-rays showed an eccentric lytic lesion involving the medial cortical border without associated fractures (Fig. 2). Abdominal ultrasound showed hepatosplenomegaly and enlarged retroperitoneal lymph nodes with sizes up to 3.7 cm (Figs. 3 and 4). The echocardiography

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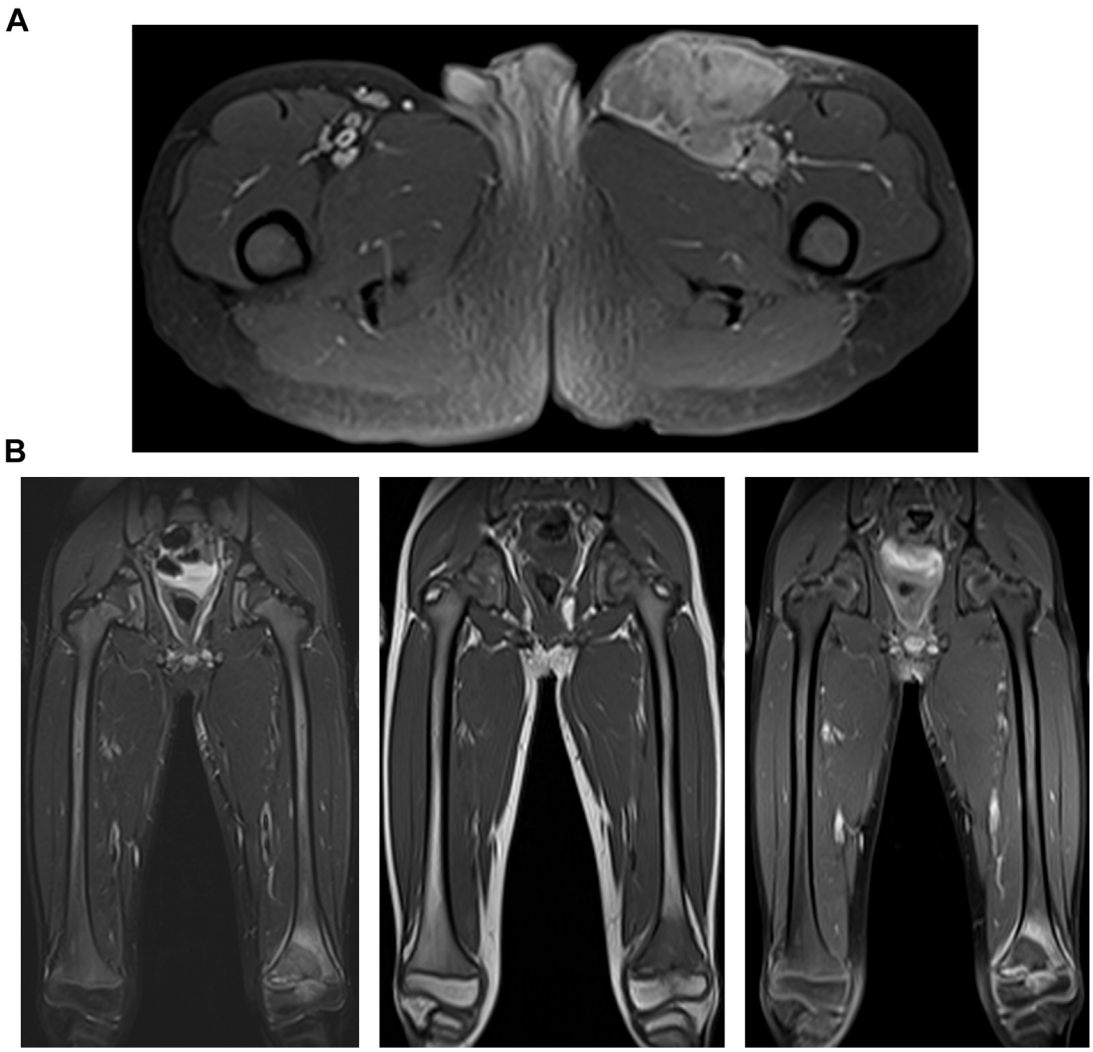

Fig. 1 – Left thigh contrast-enhanced magnetic resonance (MR). (A) Axial T1-weighted postcontrast fat saturation images. Note the enlarged femoral lymph nodes forming a mass with inflammatory changes. (B) coronal short TI inversion recovery (STIR), T1-weighted precontrast and postcontrast with fat suppression images. Aggressive soft tissue lesion in the distal metaphysis of the left femur, with marked bone edema, heterogeneous enhancement, and fine periosteal reaction. There is no joint effusion and no soft tissue mass.

revealed a mobile mass in the right atrium adhered to the tricuspid valve (Fig. 5).

With the described findings, the orthopedic oncology group had in mind a bacterial endocarditis with secondary distal femoral osteomyelitis vs a soft tissue sarcoma with intracardiac involvement. A cardiac MR was ordered for better evaluation of the cardiac compromise (Fig. 6).

Laboratory tests showed hypochromic microcytic anemia, C-reactive protein of 3.96, and a lactate dehydrogenase of 312 U/L. Bone marrow aspiration was normal.

With the above findings, the possibility of bacterial endocarditis with osteomyelitis was remote. Other entities were considered, mainly lymphoproliferative disease and Ewing sarcoma. Incisional biopsies of the left thigh mass and distal femur bone lesion were performed. The results of the bone and lymph node pathology showed a malignant lymphoid infiltrate with intermediate size cells and extensive necrosis. Lymphoid cells expressed CD20, bcl-6, and CD10 markers, and they were negative for BCL-2, MUM1, and TdT (Fig. 7). The diagnosis of IVA-stage Burkitt lymphoma (BL) with nodal and extranodal compromise was made.

The patient underwent a karyotype test for 46 XY translocation (4; 11) (q21; q23), which was negative and was treated with AA chemotherapy (cytarabine, etoposide, ifosfamide) with a satisfactory clinical evolution.

Discussion

Lymphoma is a form of cancer that affects the immune system, specifically lymphocytes. There are two broad types of lymphoma: Hodgkin's or non-Hodgkin's. BL is a form of non-Hodgkin's lymphoma in which it starts in immune cells called B-cells [1,2].

Denis Burkitt described BL for the first time in Uganda in 1958. The World Health Organization classifies it in three