Case report

Langerhans cell sarcoma: a case report and review of the literature

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We present the case of a 62-year-old male patient with a three-month history of pain in the left shoulder. Magnetic resonance imaging of the left scapula showed an osteo-destructive lesion. H&E stained sections revealed a Langerhans cell sarcoma, and immunohistochemistry was performed additionally; CD68, CD163, CD14, fascin, HLA-DR, lysozyme, S100 CD1a and langerin showed a positive reaction, while CD20, CD30, CD34, CD31, pan-cytokeratin, AE/1AE3, SMA, desmin, EMA, ERG, INI-1, CD21, CD4, PLAP, MPO and CD117c were negative. We suggested palliative treatment with chemotherapy and radiation. The patient refused any treatment and died 2 weeks later.

Key words: Langerhans cell sarcoma, histiocytic cell tumour, diagnosis, treatment, histology.

Introduction

Langerhans cell sarcoma (LCS) is an extremely rare neoplasm of Langerhans cells with malignant cytological features, which is highly aggressive with > 50% mortality from progressive disease [1]. According to the most recent WHO Classification of Tumours in 2008, it belongs to the category of histiocytic and dendritic cell neoplasms [1]. They can appear at any age, with a median age of 41 years. A female predominance (1 : 1.5) has been described in the literature [2].

Currently, only 46 cases (including the present case) of this neoplasm have been published (Table I). There are no universally accepted international guidelines available for the diagnosis and treatment of LCS patients [2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35]. To the best of the authors' knowledge, we report the first case of LCS appearing as an osteo-destructive expansion at the scapula.

Case presentation

In September 2014 a 62-year-old Caucasian male patient was admitted to our department with a three-month history of pain in the left shoulder. Plain x-rays showed destruction of the scapula with multiple osteolytic lesions. Magnetic resonance imaging (MRI) of the scapula showed an infiltration of the fossa subscapularis and the glenoid of 5 × 5 × 7 cm in diameter (Figs. 1 and 2). Bone scan and computed tomography revealed loco-regional metastases in the left shoulder, lymph nodes and multiple metastases in the liver. An incisional biopsy was performed.

Macroscopically the tumour showed a yellow to greyish colour tone. Microscopically, the tumour cells were epithelioid. The nuclei of the tumour cells were enlarged, vesicular as well as irregular with focal prominent nucleoli and they were surrounded by eosinophilic cytoplasm. Inflammatory cell infiltration, consisting of lymphocytes and eosinophil gran-

Table I. Summary of the reported cases of Langerhans cell sarcoma [2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35]

Case No.	Reference	Sex	Age (Year)	Involving	Treatment	Outcome
1	Pileri *et al*. [2]	F	17	lymph node	chemotherapy, radiation	RA CR
2	Pileri *et al*. [2]	M	48	lymph node	chemotherapy	AWD
3	Pileri *et al*. [2]	M	28	mediastinum, hepatoslenomegaly	none	DOD
4	Pileri *et al*. [2]	F	50	skin	NA	NA
5	Pileri *et al*. [2]	F	10	skin	surgery, radiation	CR
6	Pileri *et al*. [2]	F	23	lymph node, skin, lung	chemotherapy	DOD
7	Pileri *et al*. [2]	F	65	lymph node, lung, hepatosplenomegaly	chemotherapy	DOD
8	Pileri *et al*. [2]	M	72	lymph node, rib, central nervous system	chemotherapy	DOD
9	Pileri *et al*. [2]	F	50	bone	surgery	CR
10	Kawase *et al.* [3]	M	59	skin, lymph node, bone marrow, splenomegaly	chemotherapy	DOD
11	Kawase *et al.* [3]	M	35	bone, lymph node, pleura	chemotherapy	DOD
12	Kawase *et al.* [3]	F	62	lymph node, hepatosplenomegaly	chemotherapy	DOD
13	Kawase *et al.* [3]	M	60	bone	radiaton	AWD
14	Itoh *et al*. [4]	F	74	skin	surgery, radiation	DOD
15	Tani *et al*. [5]	F	49	skin, lymph node, lung	chemotherapy	DOD
16	Misery *et al.* [6]	F	38	skin	surgery	CR
17	Ferringer *et al.* [7]	M	33	skin, lymph node	chemotherapy	CR
18	Jülg *et al.* [8]	M	81	mediastinum, lung	chemotherapy	DOD
19	Lee *et al*. [9]	M	34	lung	surgery	CR
20	Bohn *et al.* [10]	M	47	skin, lymph node	surgery, chemotherapy	DOD
21	López-Ferrer *et al*. [11]	M	67	lymph node	NA	NA
22	Sumida *et al*. [12]	M	57	lymph node, tonsil, splenomegaly, bone marrow	chemotherapy	DOD
23	Deng *et al*. [13]	M	88	skin	surgery	CR
24	Diaz-Sarrio *et al*. [14]	M	58	skin, lymph node	surgery, chemotherapy, radiation	DOD
25	Zhao *et al.* [15]	F	74	galbladder, lymph node	surgery	CR
26	Uchida *et al.* [16]	M	72	skin	chemotherapy, surgery	CR
27	Yoshimi *et al*. [18]	F	53	lymph node, lung, hepatosplenomegaly, bone marrow, gaster	chemotherapy	DOD
28	Li *et al.* [19]	M	48	skin	surgery, chemotherapy	CR
29	Lakshmaiah *et al.* [20]	F	38	lymph node	chemotherapy	OFU
30	Chung *et al.* [21]	F	11-month	lymph node, hepatosplenomegaly	chemotherapy	OFU
31	Chung *et al.* [21]	F	1	lymph node, hepatosplenomegaly	chemotherapy	OFU
32	Kang *et al.* [22]	F	31	lymph node	surgery	OFU
33	Wang *et al*. [23]	F	77	lymph node	none	DOD
34	Chen *et al.* [24]	M	68	lymph node	chemotherapy	AWD
35	Wang *et al*. [25]	M	41	skin, liver, lung	surgery, chemotherapy, radiation	DOD

Table I. Cont.

Case No.	Reference	Sex	Age (Year)	Involving	Treatment	Outcome
36	Langfort *et al*. [26]	M	47	lung, lymph node	antibiotics and antifugal treatment	AWD
37	Yok-Lam Kwong [27]	M	24	skin, lymph node	surgery, chemotherapy	CR
38	Nakayama *et al*. [28]	M	62	lymph node	radiation	CR
39	Furmanczyk *et al*. [29]	M	75	skin, splenomegaly	surgery, radiation	DOD
40	Shimizu *et al.* [30]	F	67	lymph node	chemotherapy, radiation	CR
41	Keklik *et al*. [31]	M	39	nasopharynx	chemotherapy	DOD
42	Muslimani *et al*. [32]	F	66	hypopharynx	chemotherapy	AWD
43	Valentín-Nogueras *et al*. [33]	M	71	skin	surgery, chemotherapy	DOD
44	Xu *et al.* [34]	M	86	lymph node	radiation	DOD
45	Lucas *et al.* [35]	M	68	skin, lymoh node	surgery, chemotherapy, radiation	DOD
46	Current case	M	62	bone (scapula), lymph node, lung, liver	chemotherapy, radiation	DOD

F – female; M – male; DOD – dead of disease; AWD – alive with disease; CR – complete remission; RACR – relapse after complete remission; OFU – on follow-up; NA – not available

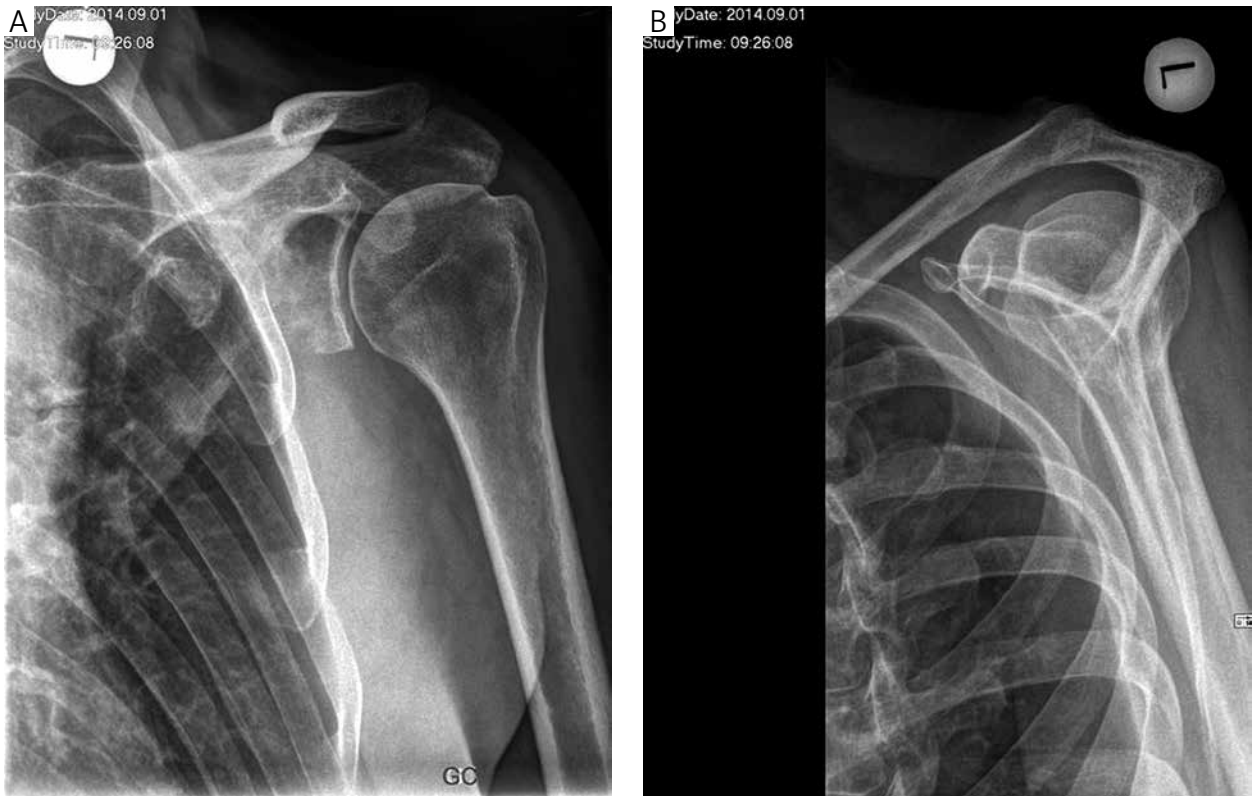

Fig. 1. A) Anterior-posterior and B) lateral plain radiographs of the left shoulder showing multiple osteolytic lesions

ulocytes, was also found, and isolated mitotic figures were identified (Fig. 3A and 3B).

Immunohistochemically, the tumour cells were multifocal reactive for CD68, CD163, CD14, fascin, HLA-DR and lysozyme. The tumour cells were also positive for S100 protein (Fig. 4). Antibodies against CD1a (Fig. 5) and langerin (Fig. 6) showed a focal and diffuse positive reaction. The following were negative: CD20, CD30, CD34, CD31, pan-cytokeratin (AE/1AE3), SMA, desmin, EMA, ERG, INI-1,

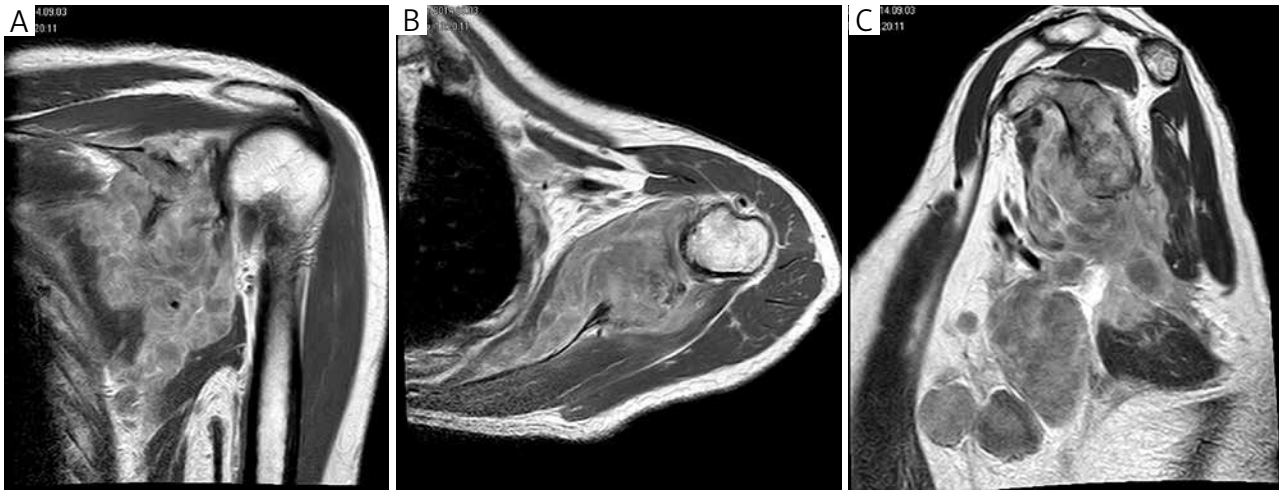

Fig. 2. A–C) MRI of the left shoulder demonstrating the whole involvement of the scapula as well as loco-regional soft tissue metastases

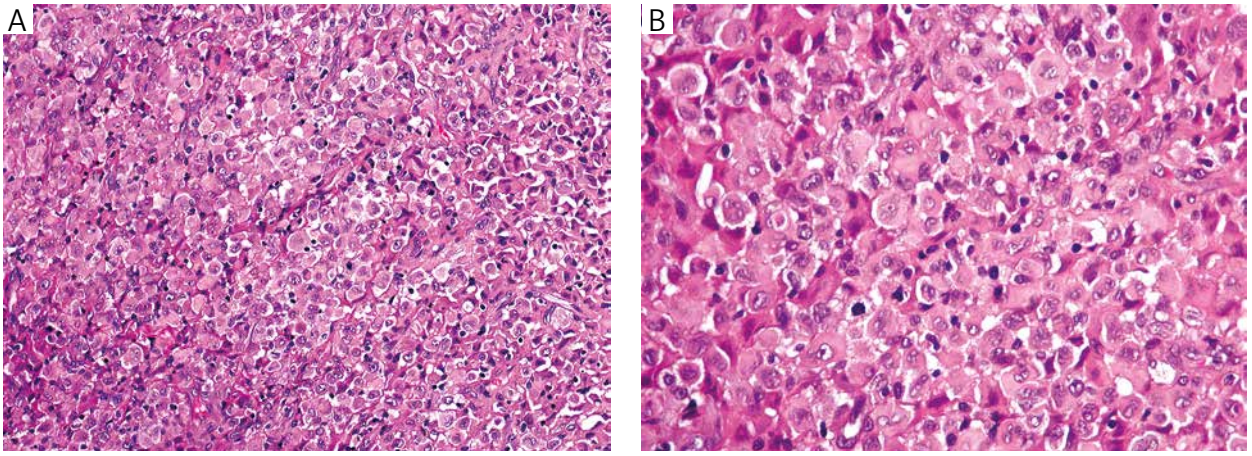

Fig. 3. A) Epithelioid tumour cells with enlarged, vesicular and irregular nuclei (HE, magnification 20×). B) Tumour cells with eosinophilic cytoplasm inflammatory infiltration consisting of lymphocytes as well as eosinophil granulocytes were found within the tumour cells (HE, magnification 40×)

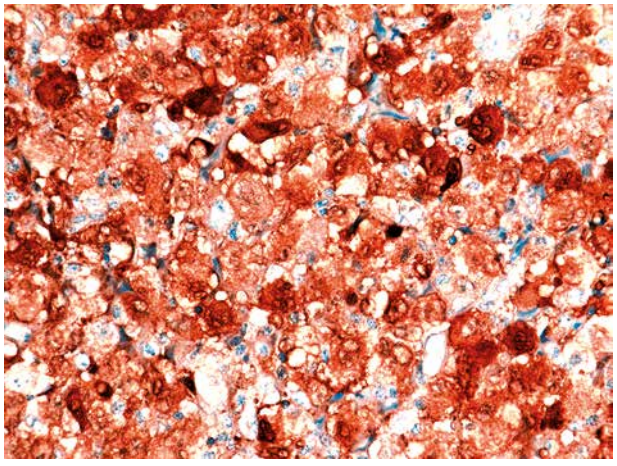

Fig. 4. Tumour cells positive for S100 protein (magnification 40×)

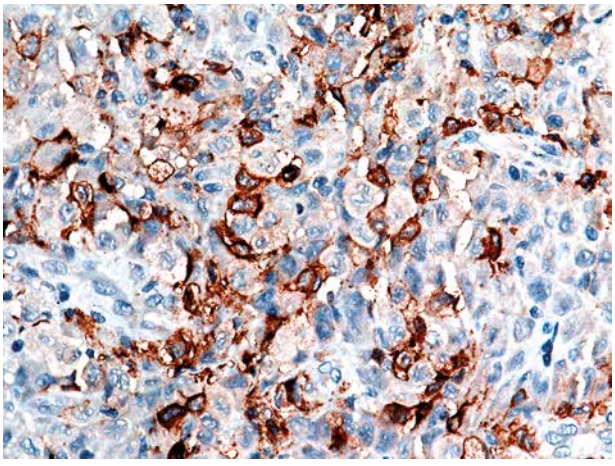

Fig. 5. Focal and diffuse positive reaction for antibodies against CD1 (magnification 40×)

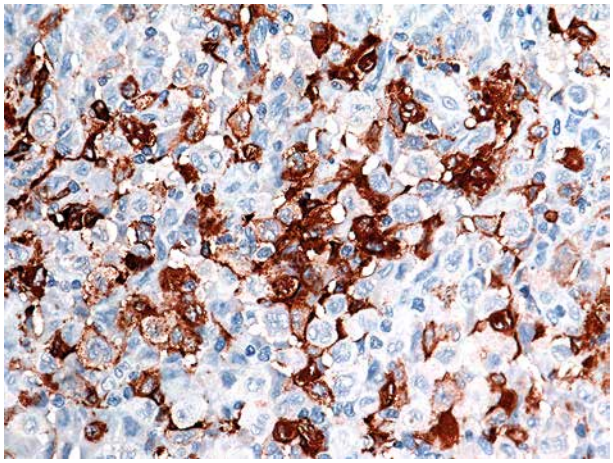

Fig. 6. Focal and diffuse positive reaction for antibodies against langerin (magnification 40×)

CD21, CD4, PLAP, MPO and CD117c. Based on histopathological and immunohistochemical findings, Langerhans cell sarcoma was diagnosed.

Further radiological staging investigations including computed tomography (CT) with contrast medium of the chest, abdomen and pelvis revealed loco-regional metastases in the left shoulder, lymph nodes in the dorsal part of the scapula and the supraclavicular region, and multiple metastases in both lungs as well as in the liver. Therefore, we suggested palliative treatment with chemotherapy and radiation. The patient refused any kind of treatment and died 2 weeks later.

Discussion

In 1984, Wood gave the first report of a clinically aggressive and infiltrating Langerhans cell tumour. He recognized the tumour as malignant histiocytosis X [36]. The International Lymphoma Study Group first defined LCS in 2002 [2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36]. Since its definition in 2002, only 46 cases of LCS have been published [3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35] (Table I).

Langerhans cell sarcoma may develop de novo or by progression from previous Langerhans cell histiocytosis (LCH) [1, 9]. Furthermore, a case of LCS occurring in a patient who underwent liver transplantation was also reported [14]. As per definition of the WHO, LCS is a clonal neoplastic proliferation of Langerhans cells with malignant cytological features. The cells are oval and about 10-15 μm in size. Grooved, folded, indented or lobulated nuclei with fine chromatin, inconspicuous nucleoli and thin nuclear membranes are typical characteristics. They also show nuclear atypia, but mitotic activity is variable and can be high without atypical forms. An ultrastructural hallmark is the cytoplasmic Birbeck granules, whose presence can be confirmed by Langerin expression. They have a tennis racquet shape, and are 200-400 nanometres long and 33 wide, with a zipper-like appearance [1].

The pathologic diagnosis of LCS is difficult, because of the rarity of this entity and morphological similarity to other neoplasms. Other tumours, such as metastatic melanoma and metastatic undifferentiated large-cell carcinoma, have also been confused with LCS [37, 39].

LCS shows highly aggressive clinical behaviour and is characterized by local destruction and multi-organ involvement in terms of metastatic disease, such as lymph nodes, liver, lung and bone [1]. Using immunohistochemical techniques is an invaluable tool for diagnosis of LCS. The immunophenotype of LCS is identical to that of LCH and shows consistent expression of S-100 and CD1a [1].

The literature concerning expression of lysozyme and CD168 is sparse [1, 42]. Langerhans cells, once neoplastic, should lack lysozyme and CD168 [42]. However, according to the recent WHO Classification of Tumours of Haematopoietic and Lymphoid Tissue, the IHC profile is consistent with lysozyme, which is also multifocal reactive in our case. Interestingly, in our analyses, CD168 was also multifocal reactive. We interpreted this result by the fact that this gene encodes a protein which is a member of the scavenger receptor cysteine-rich (SRCR) superfamily and is exclusively expressed in monocytes and macrophages. In addition, CD68 also highlights monocytes and macrophages. As a reaction with CD68 is described in LCS, it is not so unusual that also CD163 is focal positive. We also found controversy about the expression of fascin. While the 12 cases of LCH analyzed by Jaffe *et al*. expressed fascin rarely (no more than 1% of the lesion cells), Pinkus *et al.* evaluated 34 cases of LCH and observed fascin reactivity in all cases [40, 41]. They noted that the lack of antigen-retrieval techniques may be a factor that leads to diverse results [40]. Their findings correlate with our case, where fascin is multifocal reactive. We interpreted this result by the fact that LCS originates from Langerhans cells, which are a special type of dendritic cells. Negative staining for PLAP can be explained due to the fact that we only had a tumour biopsy and not the whole specimen. PLAP can partially get lost in sarcomas. LCS can be distinguished from LCH by malignant cytological features such as Langerhans cells with high atypical and high mitotic rates. According to the WHO, the mitotic rate is high and greater than 50 per 10 high power fields [1]. Malignant cytological features are normally linked to an aggressive clinical course and poor outcome. Due to that reason, Pileri *et al.* discussed the clinical outcome between LCH and LCS [2]. They suggested a possi-

ble correlation between cytological malignancy and poor clinical outcome. Ben-Ezra *et al*., however, suggest that in the case of LCH, morphology is only an imperfect predicator of the clinical outcome. They described cases of benign cytological findings and aggressive clinical course and cases of malignant cytological findings and benign clinical outcome [37].

Newton and Hamoudi described two morphologic subtypes: type 1 and type 2 Langerhans cells [42]. Type 1 Langerhans cells can be found as single, non-coherent cells with few eosinophils, whereas type II Langerhans cells are arranged in sheets and many eosinophils can be found. Clinically, type I patients had an aggressive course and type II patients had a benign outcome [42].

LCS occurs in a wide age range, in the reported literature from 1 to 88 years, with mainly elderly patients and only in 4 cases younger than 18 years (Table I). The calculated male: female ratio is, including our present case, 1.42 : 1, although a female predominance of 1 : 1.5 has been described in the literature [2]. Surgery, chemotherapy, radiation or a combined version of these three methods was used as treatment. About half of the patients experienced multi-organ involvement, despite the intensive therapy. Multi-organ involvement is a predictor of poor prognosis and short survival time, and nearly all of the above-mentioned patients had a poor clinic outcome (Table I). On the other hand, solitary lesions in the skin, lung, and lymph nodes showed a favourable clinical outcome (Table I).

There are no universally accepted international guidelines available for the diagnosis and treatment of adult LCS patients. Researchers and clinicians have treated patients with LCS in accordance with the common lymphoma treatment protocols using the CHOP (cyclophosphamide, prednisolone, doxorubicin, and vincristine) [21], MAID (mesna, doxorubicin, ifosfamide and dacarbazine) [16], a modified ESHAP (etoposide, carboplatin, cytarabine, and methylprednisolone), EPIG (etoposide, cisplatin, ifos famide, mesna and gemcitabine) or AIM regimen (doxorubicin, ifosfamide and mesna) [30].

Lucas *et al.* reported that radiation therapy also has the potential to shrink tumour lesions [35]. They found that LCS responds well to radiotherapy and believe that irradiation is a good option in selected cases with symptomatic or bulky lesions.

To achieve the correct diagnosis of this rare, high-grade entity and thereby the adequate treatment, a multidisciplinary approach is essential. Treatment options vary depending on disease extent and severity at onset. A uniform diagnostic work-up is necessary. One of the main problems of LCS is the variety of potentially involved organs, resulting in several physicians being consulted.

The authors declare no conflict of interest.

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