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Case report

Mediastinal Castleman disease presenting as a paraspinal mass causing back pain and shortness of breath in a young adult

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ABSTRACT

This case report details a rare presentation of unicentric Castleman disease (UCD), hyaline vascular type in a 22-year-old woman. The patient presented with a large, well-circumscribed mass in the paravertebral region causing back pain and shortness of breath. Diagnostic imaging and biopsy confirmed the diagnosis, and surgical excision led to a favorable outcome. This case underscores the critical need to include Castleman disease in the differential diagnosis for young adults presenting with mediastinal masses. Early recognition and surgical intervention are essential for a favorable prognosis in UCD cases.

1. Introduction

Castleman disease (CD) is a rare lymphoproliferative disorder often found incidentally on imaging studies in patients of any age, either with or without nonspecific symptoms. It typically presents as a well-defined, mildly hypodense or isodense, homogeneous lymph node enlargement on computed tomography (CT) imaging. This report highlights a case of UCD in a young woman, presenting with back pain, an uncommon manifestation of CD, demonstrating the clinical and diagnostic challenges, and underscores the significance of including Castleman disease in the differential diagnosis of mediastinal masses.

2. Case

A 22-year-old woman with no medical, surgical history and no cancer related family history presented to our outpatient department with a one-year history of back pain and tenderness over the paraspinal muscles. The discomfort was located in the lower thoracic area

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and was described as a deep aching and pressure-like sensation, occasionally accompanied by a sharp or stabbing component. Initially, the pain was tolerable but gradually worsened over several months. It was exacerbated by activities such as twisting or bending and by actions that increased intra-abdominal pressure, such as coughing, sneezing, or straining. Resting or applying heat provided some relief. Additionally, the patient reported experiencing shortness of breath few weeks preceding her visit, prompting her to seek medical attention.

Upon physical examination at our outpatient department, no specific findings were noted. The patient had no signs of B symptoms such as fever, night sweats and weight loss. There is also an absence of systemic inflammation signs, as well as redness or swelling in the affected area. There was no palpable mass in the chest or other node-bearing areas, and both liver span and spleen size were within normal ranges.

Laboratory evaluations were unremarkable, with the complete blood count and differential showing no significant abnormalities. While flow cytometry was not performed, other ancillary techniques for differential diagnosis such as an IHC panel for certain immune biomarkers(kappa/lambda, CD20, CD3, CD5, CD138, HHV8) was also negative. Serologic studies over infection and other inflammatory markers such as IL-6 or CRP were also unexceptional.

A chest radiograph revealed a well-defined mass in the lower thoracic paravertebral region on the left side, partially obscured by the cardiac shadow. The mass was located around the T9-10 vertebral level (Fig. 1A), causing some deviation or displacement of adjacent vertebrae. There was no evidence of calcification within the mass, and the lung fields were clear, without signs of infiltrates or pleural effusion.

Given these findings, further investigation with contrast-enhanced computed tomography (CT) of the chest was performed. The CT scan showed a lobulated mass measuring 5.8 cm in greatest diameter with heterogeneous enhancement in the left posterior mediastinum, consistent with the location seen on the chest X-ray. The mass compressed adjacent tissues but showed no evidence of invasion into surrounding structures, nor was there evidence of necrosis or rupture (Fig. 1B). Imaging characteristics raised concerns for a lymphoproliferative disorder, warranting further biopsy as part of the differential diagnosis.

A biopsy of the lesion revealed dense lymphoplasmacytic cell infiltration (arrow) along with areas of fibrosis and spindle cell proliferation (yellow arrow) (Fig. 2), suggesting a lymphoproliferative disorder. The absence of Reed-Sternberg cells and monoclonal B- or T-cell proliferation makes lymphoma less likely. Consequently, the patient underwent thoracoscopic excision for treatment and further pathological evaluation. The surgery proceeded smoothly, and the patient recovered without complications. The excised specimen was a well-circumscribed, lobulated mass measuring approximately 6–7 cm in diameter, with a reddish, hemorrhagic cut surface (Fig. 3A). Histopathological findings revealed a densely cellular lymphoid proliferation with an "onion skin" appearance, characterized by multiple concentric rings of mantle zone lymphocytes encircling an atretic germinal center (arrow) and a hyalinized or hypervascular interfollicular area (yellow arrow) (Fig. 3B). The histological features are consistent with typical unicentric Castleman Disease, hyaline vascular variant, thus the diagnosis was made.

Following the surgery, the patient was satisfied with the treatment. Her postoperative recovery showed no complications, and according to her, she was discharged home in good condition without further discomfort. Follow-up visits confirmed her continued recovery, with no signs of recurrence or residual symptoms. The latest follow-up one year after the surgery indicates that the patient is doing well without any long-term complications.

3. Discussion

Castleman disease is a rare lymphoproliferative disorder that lacks a consensus definition. The variability in pathogenesis and

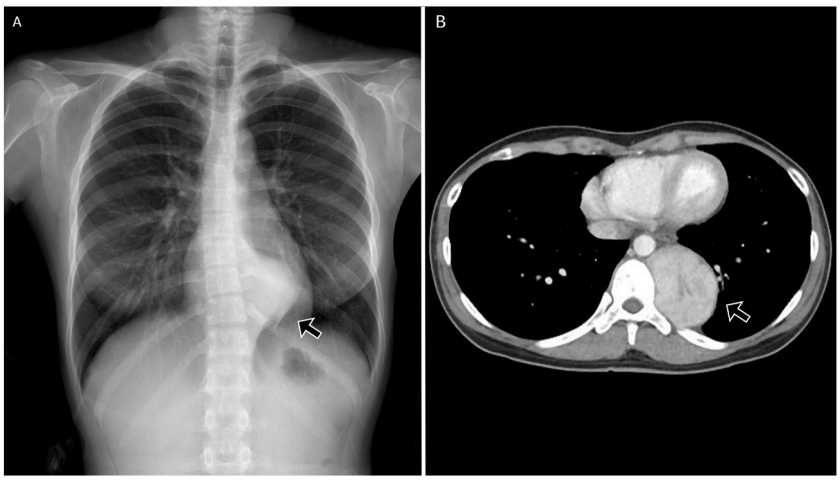

Fig. 1. (A) Chest radiograph showed a smoothly marginated mass in the left paravertebral region. **(B)** Contrast-enhanced computed tomography of the chest demonstrated a lobulated mass with a size of 5.8cm in greatest diameter and heterogeneous enhancement located in the left posterior mediastinum.

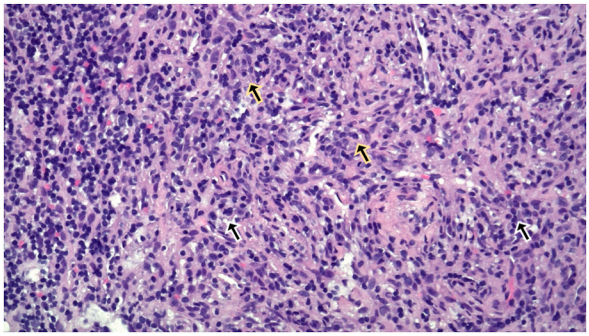

Fig. 2. Biopsy of the lesion revealed dense lymphoplasma cell infiltration **(arrow)** with areas of fibrosis and spindle cell proliferation **(yellow arrow)**.

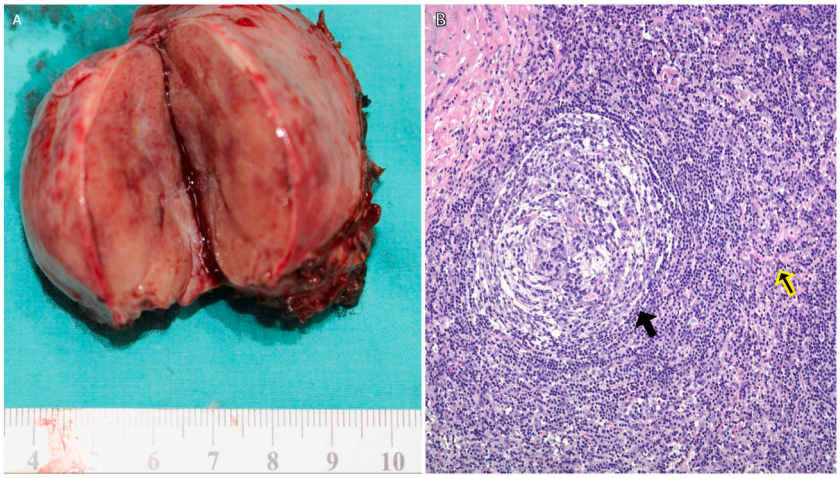

Fig. 3. The patient was referred for surgery, and thoracoscopic tumor excision was performed. **(A)** The gross specimen of mass depicted a well-circumscribed lesion with a reddish and hemorrhagic cut surface and the **(B)** histopathological evaluation of the resected segment revealed an onion skin appearance with multiple concentric rings of mantle zone lymphocytes encircling an atretic germinal center **(arrow)** and a hyalinized or hypervascular interfollicular area **(yellow arrow)**.

clinical features makes it hard to identify. A limited spectrum of histopathological features has been noticed over the years, and several subtypes have also been found, making the disease easier to detect. However, without typical symptoms and definitive criteria in clinical evaluation, it is still challenging to include the disease in the differential diagnosis during the initial evaluation.

Two main types have been identified over recent decades: unicentric and multicentric Castleman Disease (UCD and MCD). It is known to affect both genders across a wide age range, with most cases occurring in the 3rd to 5th decades of life. In most reviews so far, the median age of MCDs compared to UCDs is significantly higher, around 50 years compared to 30 years [1]. The incidence of each type remains debated [2]. The unicentric type is more common than the multicentric type in most areas, including the United States, China, Czechoslovakia, and New Zealand [1]. However, a Japanese study published in 2019 showed a subtype of MCD, known as iMCD, had more cases than UCD, with an incidence of 2.4–5.8 over 0.6–4.3, and the reason remains unclear [3].

In Unicentric Castleman Disease, the median age of diagnosis is relatively young, at about 34 years, but with a wide range from 2 to 84 years. The lack of known risk factors complicates its inclusion in differential diagnoses. Most cases are diagnosed in their 20s–30s, as in the case we described, but rare cases in young children and the elderly have also been reported over the years. There seems to be a slightly higher incidence in females than in males [1]. Study on MCD and its subtypes has shown some progress over the years. HHV-8 related and idiopathic MCD are considered to be complications of uncontrolled infection by several pathogens, overactive autoimmune processes, autoinflammatory diseases, as well as paraneoplastic somatic mutations in monoclonal lymph node cells [4]. On the other hand, the risk and etiology of UCD still remain unclear. Patients and cases reported with no known medical or other history and risk factors are common, just as in the case reported previously in this article. We only know that several rare diseases have been claimed to be related to UCD, including paraneoplastic pemphigus, bronchiolitis obliterans, amyloidosis, and neoplasms such as follicular dendritic cell (FDC) sarcoma and lymphoma [5].

The pathogenesis of CD is thought to be related to the overproduction of a cytokine, interleukin 6 (IL-6), but the mechanisms and triggers remain unclear so far. In UCD, stromal follicular dendritic cell hyperplasia is the main process of forming the disease, thus it is

considered to be a clonal neoplastic process [6]. A mutation of the somatic platelet-derived growth factor receptor-b (PDGFRB) was also found in about 20 % of patients in a study, and the mutation was specifically localized in stromal cells such as follicular dendritic cells [7]. Although the trigger of cytokine overproduction in idiopathic MCD is still unknown, viral-driven, inflammatory diseases, and paraneoplastic hypotheses are considered the most probable reasons. Elevation of VEGF was also detected in the TAFRO subtypes and POEMS syndrome. Comparatively, studies on HHV-8 associated MCD have made more progress. We now know that HIV infection causes immunosuppression of the host, allowing HHV-8 to evade immune control, resulting in excessive cytokine production [8].

The clinical findings of Castleman disease vary largely by its subtype and sites. MCD can present more severe symptoms involving different systems, including fever, weight loss, anemia, thrombocytopenia, or even organ failure. The National Comprehensive Cancer Network has already set criteria for the diagnosis of active disease, which include fever, abnormal serum CRP >20 mg/L indicating generalized inflammation, and ≥ 3 of the recorded symptoms [9], including peripheral lymphadenopathy, enlarged spleen, edema, pleural effusion, ascites, cough, nasal obstruction, xerostomia, rash, central neurologic symptoms, jaundice and autoimmune hemolytic anemia [10]. Severity should be assessed carefully due to its importance in management strategy [4].

On the contrary, UCD patients are often asymptomatic and diagnosed by incidental findings on imaging without significant discomfort. If lymphadenopathy compresses adjacent structures, symptomatic UCD might be observed, and the symptoms will always depend on the affected site, which is typically a single lymph node region. Over 70 % of reported UCD cases involve the thorax, mostly in the mediastinum. CT often reveals a single soft-tissue mass, averaging 5–7 cm in diameter, characterized by well-defined, smoothly lobulated borders. A systematic review in 2012 showed that the most common sites of UCD include the chest (29 %), neck (23 %), abdomen (21 %), and retroperitoneum (17 %). Rare sites such as the axilla (5 %), groin (3 %), and pelvis (2 %) were also noted [11]. Another systematic review recently showed similar results with a 29 % incidence in the head and neck area, retroperitoneum 20 %, abdomen 13 %, and a sum of chest and mediastinum at about 25 %. The pelvic and axillary areas are as low as the previous study [5].

Thoracic involvement may present symptoms caused by lymph node compression on surrounding organs, mimicking other chest neoplastic diseases, and may progress over time. Cough, hemoptysis, dyspnea, or chest discomfort are the most common findings in thoracic disease. Compression in the abdomen, pelvis, and retroperitoneum may cause abdominal or back pain, with rare manifestations such as bowel obstruction and ureteral intervention also recorded in the literature [12]. Masses confined to the head and neck area may present with nontender lymphadenopathy. A case report also showed dysphagia or dyspnea due to compression caused by the mass in the neck area [13]. Adjacent muscle or joint pain may also present due to compression caused by the mass effect [5]. Comparing a case report showing a rare large extrathoracic mass at the paraspinal area causing back pain [14], our case also presents CD in the paraspinal area, despite being in the thoracic cage, still causing paraspinal muscle strain and resulting in back pain.

Positive findings through clinical examination are rare in UCD compared to MCD, according to the systematic study mentioned above [5]. The most common finding is a palpable tumor mass, which is present in only 38 % of UCD patients compared to 65 % in MCD. The second and third most common findings are general illness, such as fever and weight loss, accounting for merely 10 % and 9 %, respectively, making the results unremarkable and thus hard to reach a diagnosis. While laboratory findings may show elevated IL-6 or anemia in MCD or UCD with the plasma-cell type, data shows anomalies are almost absent in the most common type of UCD, which is the hyaline-vascular type, accounting for 70–90 % of diagnosed UCD [5,15].

The symptoms and clinical manifestations described above are usually nonspecific, thus we can only consider a diagnosis of UCD or MCD when the patient presents isolated or multiple sites of lymphadenopathy. Imaging studies may be nonspecific, with localized mass or enlarged lymph nodes often seen. Radiographs are often the first-line examination for patients with thoracic clinical manifestations, which may show a round solitary mass in the thoracic area, including the lung, hilar, or paraspinal, mimicking thoracic tumors such as thymoma, lymphoma, or lung tumors. On the basis that UCD with the hyaline vascular variant has a considerable predilection for involvement of the thorax, it typically manifests as a solitary, well-circumscribed, and localized nodal mass on CT. The mass may show isodense or hypodense to musculoskeletal on non-contrast CT, while presenting hypervascularity with homogeneous intense contrast enhancement on contrast CT [15], as also shown in our case (Fig. 1B). Ultrasound, MRI, and PET may also be used for further evaluation. Alternative considerations other than UCD may be obtained when a PET evaluation shows a standardized uptake value max of >6, such as lymphoma [16].

The differential diagnosis for the various subtypes of Castleman disease (CD) is extensive, even after a lymph node biopsy confirms consistency with CD. nicentric CD (UCD) tends to have a more limited differential diagnosis, as it is typically localized, requiring the exclusion of fewer conditions compared to multicentric CD (MCD), which involves systemic symptoms and mimics a broader range of diseases. Aside from lymphomas, few other diseases present with a solitary enlarged lymph node exhibiting histopathological features similar to those seen in UCD patients, though reactive lymphadenopathies and benign conditions may still overlap histologically [17].

The presence of B symptoms—fever, night sweats, and weight loss—often raises suspicion for both Non-Hodgkin lymphoma (NHL) and Hodgkin lymphoma (HL), but it's important to know that these systemic symptoms can also occur in MCD. UCD patients, by contrast, typically do not present with systemic symptoms. A comprehensive serological study, including complete blood count (CBC) with differential, a metabolic panel, serum lactate dehydrogenase (LDH), CRP, and ESR should be measured in the initial workup [9]. Immunophenotyping by flow cytometry and immunohistochemistry of the surgical specimen are usually sufficient to distinguish NHL from UCD. In some cases, however, further molecular studies or additional biopsies might be necessary if the differentiation is difficult. An excisional biopsy is more reliable for diagnosing HL, as classical HL is characterized by the presence of Hodgkin and Reed-Sternberg (HRS) cells. It is worth noting that UCD may sometimes exhibit cells that mimic Reed-Sternberg cells, emphasizing the importance of immunohistochemistry in these cases [18,19].

While infectious causes are less likely to affect a solitary lymph node, they should still be considered. Certain infections like tuberculosis, localized bacterial or viral infections can present with isolated lymphadenopathy. Serological tests, imaging, biopsy and occasionally needle aspiration may be used to confirm the pathogen. For toxoplasma lymphadenitis, ELISA testing for IgM and IgG

antibodies is commonly used to differentiate it from UCD, while detection of parasites by microscopy or culture of body fluids and tissues can also help exclude infectious causes [20].

If autoimmune diseases, such as rheumatoid arthritis or systemic lupus erythematosus (SLE), are suspected, additional blood tests including autoantibody panels (e.g., ANA, anti-dsDNA) and flow cytometry should be performed to confirm the diagnosis, as these conditions can cause lymphadenopathy [21].

According to NCCN guidelines, the gold standard for UCD diagnosis is excisional biopsy; other biopsy methods usually lack accuracy. The gross specimen of the mass in Castleman Disease often depicts a well-circumscribed lesion with a reddish and hemorrhagic cut surface (Fig. 3A) [22]. Histopathological evaluation of the resected segment in the UCD hyaline vascular variant may reveal an onion skin appearance with multiple concentric rings of mantle zone lymphocytes encircling an atretic germinal center (arrow) and a hyalinized or hypervascular interfollicular area (yellow arrow) (Fig. 3B) [22]. On the other hand, the plasma cell variant does not present follicular dysplasia or atypia; instead, variable follicular hyperplasia with paracortical plasmacytosis is usually seen [4].

Complete surgical resection is often curative and may eliminate any clinical abnormality [22,23]. If the mass involves vital organs and structures such as main vessels or the trachea, debulking surgery should be considered as an alternative. Long-term follow-up with or without neoadjuvant therapy after debulking or partial resection may also help reduce the recurrence rate and remaining size [24]. If the patient is not surgically available, radiotherapy, chemotherapy, and targeted therapy used in MCD may also be considered, such as rituximab or anti-IL-6 therapy. Although the optimal treatment other than surgery has not been well defined, radiotherapy is considered a reasonable choice to treat UCD alone [4].

UCD is generally associated with a favorable prognosis compared to its multicentric counterpart. Patients rarely die from the disease itself, and the mortality associated with UCD is relatively low. A large series showed that the overall mortality rate of UCD was about 5 % after surgery, while patients who did not receive probable resection therapy, such as incomplete resection, were reported to have a higher fatality rate of 17.6 % [11]. A retrospective analysis also showed a cure rate of 95 % after surgery, which is consistent with the series mentioned previously [24]. The only exceptions are the rare development of lymphoma or paraneoplastic pemphigus (PNP) in UCD patients, which can be deadly complications [25]. In summary, surgical resection remains the gold standard treatment for UCD patients, as demonstrated in the case described, and the patient was discharged without complications and presented no other discomfort during follow-up.

4. Conclusion

This case report underscores the importance of considering Castleman disease in the differential diagnosis of well-circumscribed mediastinal masses in young adults. The patient's typical clinical manifestations and successful treatment highlight the significance of early recognition and intervention in UCD cases, contributing to improved clinical practice and patient outcomes.

CRediT authorship contribution statement

Huan Yang Chen: Writing – review & editing, Writing – original draft, Conceptualization. **Shou-Hsin Wu:** Writing – original draft, Supervision, Resources, Investigation, Conceptualization. **Fatt Yang Chew:** Writing – original draft, Supervision, Investigation, Data curation, Conceptualization. **Suat Yee Lee:** Writing – review & editing, Writing – original draft, Supervision, Resources, Data curation, Conceptualization.

Informed consent statement

Written and signed consent was obtained from the patient.

Availability of data and materials

The data mentioned in the article or supplementary material is cited within the text.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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References

[1] D. Simpson, Epidemiology of castleman disease, Hematol. Oncol. Clin. N. Am. 32 (1) (2018) 1–10, https://doi.org/10.1016/j.hoc.2017.09.001.
[2] C. Hoffmann, M. Hentrich, M. Tiemann, A. Rosenwald, F. Weber, W. Willenbacher, K. Hübel, Recent advances in castleman disease, Oncol. Res. Treat. 45 (11) (2022) 693–704, https://doi.org/10.1159/000526640.

[3] Y. Masaki, H. Kawabata, S. Fujimoto, M. Kawano, N. Iwaki, T. Kotani, A. Nakashima, et al., Epidemiological analysis of multicentric and unicentric Castleman disease and TAFRO syndrome in Japan, J. Clin. Exp. Hematop. 59 (4) (2019) 175–178, https://doi.org/10.3960/jslrt.19021.
[4] K.L. Chan, S. Lade, H.M. Prince, S.J. Harrison, Update and new approaches in the treatment of Castleman disease, J Blood Med 7 (2016) 145–158, https://doi.org/10.2147/jbm.S60514.
[5] M. Haap, J. Wiefels, M. Horger, A. Hoyer, K. Müssig, Clinical, laboratory and imaging findings in Castleman's disease - the subtype decides, Blood Rev. 32 (3) (2018) 225–234, https://doi.org/10.1016/j.blre.2017.11.005.
[6] K.C. Chang, Y.C. Wang, L.Y. Hung, W.T. Huang, J.H. Tsou, M.J. D, H.L. Song, Y.M. Yeh, L.Y. Kao, L.J. Medeiros, Monoclonality and cytogenetic abnormalities in hyaline vascular Castleman disease, Mod. Pathol. 27 (6) (2014) 823–831, https://doi.org/10.1038/modpathol.2013.202.
[7] Z. Li, X. Lan, C. Li, Y. Zhang, Y. Wang, W. Xue, L. Lu, et al., Recurrent PDGFRB mutations in unicentric Castleman disease, Leukemia 33 (4) (2019) 1035–1038, https://doi.org/10.1038/s41375-018-0323-6.
[8] D.C. Fajgenbaum, F. van Rhee, C.S. Nabel, HHV-8-negative, idiopathic multicentric Castleman disease: novel insights into biology, pathogenesis, and therapy, Blood 123 (19) (2014) 2924–2933, https://doi.org/10.1182/blood-2013-12-545087.
[9] J.S. Abramson, Diagnosis and management of castleman disease, J Natl Compr Canc Netw 17 (11.5) (2019) 1417–1419, https://doi.org/10.6004/jnccn.2019.5037.
[10] N.C.C. Network, Castleman disease (version 1.2024), Cited October 17, https://www.nccn.org/professionals/physician_gls/pdf/castleman.pdf, 2024.
[11] N. Talat, A.P. Belgaumkar, K.M. Schulte, Surgery in Castleman's disease: a systematic review of 404 published cases, Ann. Surg. 255 (4) (2012) 677–684, https://doi.org/10.1097/SLA.0b013e318249dcdc.
[12] J.D. Soumerai, A.R. Sohani, J.S. Abramson, Diagnosis and management of Castleman disease, Cancer Control 21 (4) (2014) 266–278, https://doi.org/10.1177/107327481402100403.
[13] E. Raboso, R. Barberá, L. Guimaraens, J.L. Solorzano, B. Delgado-Vargas, Castleman's disease as an unusual cause for dysphagia and dyspnea in a pediatric patient, Int. J. Pediatr. Otorhinolaryngol. 125 (2019) 164–167, https://doi.org/10.1016/j.ijporl.2019.07.017.
[14] N. Buntic, T. Brown, U.S. Nisar, C. Yeary, Unicentric castleman's disease presenting as back pain, Cureus (2022), https://doi.org/10.7759/cureus.32219.
[15] R.S.M. Wong, Unicentric castleman disease, Hematol. Oncol. Clin. N. Am. 32 (1) (2018) 65–73, https://doi.org/10.1016/j.hoc.2017.09.006.
[16] F. van Rhee, P. Voorhees, A. Dispenzieri, A. Fosså, G. Srkalovic, M. Ide, N. Munshi, et al., International, evidence-based consensus treatment guidelines for idiopathic multicentric Castleman disease, Blood 132 (20) (2018) 2115–2124, https://doi.org/10.1182/blood-2018-07-862334.
[17] A. Dispenzieri, D.C. Fajgenbaum, Overview of castleman disease, Blood 135 (16) (2020) 1353–1364, https://doi.org/10.1182/blood.2019000931.
[18] R.T. Hoppe, R.H. Advani, R.F. Ambinder, e al, Hodgkin Lymphoma. National Comprehensive Cancer Network (NCCN) Clinical Practice Guidelines in Oncology, 2024.
[19] A. Bowzyk Al-Naeeb, T. Ajithkumar, S. Behan, D.J. Hodson, Non-Hodgkin lymphoma, Bmj (2018), https://doi.org/10.1136/bmj.k3204.
[20] G. Saadatnia, M. Golkar, A review on human toxoplasmosis, Scand. J. Infect. Dis. 44 (11) (2012) 805–814, https://doi.org/10.3109/00365548.2012.693197.
[21] D.C. Fajgenbaum, T.S. Uldrick, A. Bagg, D. Frank, D. Wu, G. Srkalovic, D. Simpson, et al., International, evidence-based consensus diagnostic criteria for HHV-8-negative/idiopathic multicentric Castleman disease, Blood 129 (12) (2017) 1646–1657, https://doi.org/10.1182/blood-2016-10-746933.
[22] S.J. Kligerman, A. Auerbach, T.J. Franks, J.R. Galvin, Castleman disease of the thorax: clinical, radiologic, and pathologic correlation: from the radiologic pathology archives, Radiographics 36 (5) (2016) 1309–1332, https://doi.org/10.1148/rg.2016160076.
[23] M.Y. Zhang, M.N. Jia, J. Chen, J. Feng, X.X. Cao, D.B. Zhou, D.C. Fajgenbaum, L. Zhang, J. Li, UCD with MCD-like inflammatory state: surgical excision is highly effective, Blood Adv 5 (1) (2021) 122–128, https://doi.org/10.1182/bloodadvances.2020003607.
[24] D. Boutboul, J. Fadlallah, S. Chawki, C. Fieschi, M. Malphettes, A. Dossier, L. Gérard, et al., Treatment and outcome of Unicentric Castleman Disease: a retrospective analysis of 71 cases, Br. J. Haematol. 186 (2) (2019) 269–273, https://doi.org/10.1111/bjh.15921.
[25] Y. Dong, M. Wang, L. Nong, L. Wang, X. Cen, W. Liu, S. Zhu, et al., Clinical and laboratory characterization of 114 cases of Castleman disease patients from a single centre: paraneoplastic pemphigus is an unfavourable prognostic factor, Br. J. Haematol. 169 (6) (2015) 834–842, https://doi.org/10.1111/bjh.13378.