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RESEARCH ARTICLE

Castleman Disease: A Wide Spectrum of Thoracic Manifestations

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ABSTRACT
Castleman's disease is a benign lymphoproliferative disorder affecting both lymph nodes and extranodal loci. Castleman's disease can occur in practically any part of the body, but it occurs mainly in the thorax (~70%) followed by the abdomen and pelvis, neck and axilla. Clinically, Castleman's disease can be classified into a unicentric or multicentric form, depending on the number of lymph nodes involved, and histologically into a hyaline vascular variant, plasma cell, mixed cellular or plasmablastic variant. In this mini-review we briefly report and focus on all clinical thoracic manifestations of Castleman's disease resuming for each of them the possible strategy of treatment.

Keywords: Castleman disease, mediastinal mass, lymphoproliferative disorder, thorax disease, mediastinal disease, surgical therapy

INTRODUCTION

Castleman's disease (CD) is a benign lymphoproliferative disorder affecting both lymph nodes and extranodal loci. [1-2] The first report of CD, also known as giant lymph node hyperplasia or angiofollicular lymphoid hyperplasia, is dated 1954.[3] Since the first report less than 300 have been reported in literature, more commonly as case report due to a low occurrence rate of 21 to 25 cases per million person-years.[4] Thus, its rarity makes it a diagnostic and therapeutic challenge.[5]

From a clinical point of view, two variants of CD are described: unicentric CD (UCD; ~75%) and multicentric CD (MCD; ~25%).[6] The UCD variant usually affects a single lymph node station while MCD affects more than one lymph node station and often manifests as generalized lymphadenopathy with an enlarged spleen or liver.[6] These two variants can be classified in the three main histological types: hyaline-vascular (HV), plasma cell (PC), or mixed.CD can occur in practically any part of the body, but arises predominantly in the thorax (~70%), followed by the abdomen and pelvis (~15%), neck (~12%) and axilla (4%).[7,8] Uncommonly, it can occur in extra-lymphatic tissues such as the larynx, lung, parotid gland, pancreas, and muscles.[9,10]

Concerning thoracic involvement, CD is found in most cases along the tracheobronchial tree in the mediastinum or pulmonary hilus, but it also can occur in the intrapulmonary fissure and thoracic wall.[11]

In this mini-review we briefly report the most common pathological pathway involved in CD and then we focus on all the possible clinical thoracic manifestations of CD resuming for each of them the suitable strategy of treatment.

CD PATHOGENESIS

The pathogenesis of CD remains unclear, but viral, inflammatory, and neoplastic mechanisms have been proposed as possible factors associated with the development of the disease.[12]

The distinction of CD between unicentric (UCD) and multicentric (MCD) diseases reflects major differences in the pathogenesis, clinical presentation and therapeutic options.

IL-6 dysregulation and viral infection (HIV, HHV-8) have been proposed to be the mechanism for developing of CD.[13] IL-6 is involved in the differentiation of lymphocytes, production of acute-phase reactants, angiogenesis, and tumorigenesis. Patients with CD may have elevated levels of IL-6 that normalize with the removal of the involved lymph nodes (13). However, patients affected by UCD are usually HIV-negative and no systemic symptoms or laboratory findings will reflect increased IL-6.[14] Patients with UCD usually present HV variant (90%) with a virological status negative for HHV-8. Therefore, the clinical presentation of UCD often relates to the localised mass effect on organ function. Systemic symptoms are uncommon with fever in <10% of cases and inflammatory markers including C-reactive protein (CRP), erythrocyte sedimentation rate and tests of organ function usually generally normal.[15]

On the other hand, patients affected by MCD are usually divided into HHV-8- positive MCD, idiopathic MCD (iMCD) and POEMS- related MCD (polyneuropathy, organomegaly, endocrinopathy, monoclonal proteins, and skin changes).[6]

The first variant of MCD is characterized by an excessive release of viral IL-6, while iMCD is driven by proinflammatory, mostly IL-6 hypercytokinemia. The latter one (POEMS-associated MCD) is believed to result from increased IL-6 and VEGF production by monoclonal plasma cells leading to clinical symptomatology. In particular, VEGF correlates with disease activity.[6]

MEDIASTINAL CD

Mediastinum is usually the main location of UCD (29%), followed by cervical (23%), abdominal (21%), retroperitoneal (16%) and also exceptionally axillary, inguinal or pelvic.[16]

Mediastinal UCD usually presents as solitary lymphoid tissue overgrowth or lymphadenopathy that is curable with a complete surgical resection. It might be asymptomatic or lead to compression-related symptoms. The anterior, middle and posterior mediastinum can be indifferently involved. In a recent study reporting 11 mediastinal UCD managed in 2 French thoracic surgery departments between 1988 and 2012 the most common mediastinal location was the middle mediastinum (73%), followed by anterior (18%) and posterior (9%) site.[16] For mediastinal UCD a resection surgery can be at the same time diagnostic and curative.

Pre-operative diagnosis is difficult to obtain because the lesion can mimic other mediastinal tumors. Therefore, the limited amount of material recovered with percutaneous biopsy is often not sufficient to identify the typical features of CD that distinguish it from other benign or malignant lesions of the mediastinum.[17] A systematic review of 404 published cases of CD underlines the main objective of the chosen surgical approach: to achieve a complete excision of the lymph node or cluster of lymph nodes involved. This review confirms as surgery is the gold standard for treatment of UCD. Indeed, the lack of resection of

the primary involved lymph node with free margins is considered the only predictor for a fatal outcome resulting in in a significant mortality compared to debulking the surgical approach.[18]
Video- assisted thoracoscopy (VATS) and thoracotomy are the most common approaches chosen for the treatment of the UCD. Currently, VATS is the preferred technique to remove almost all mediastinal benign tumors as is UCD. However, several relevant features of CD should be considered when a mini-invasive approach is chosen for treating CD: the presence of adhesions with the surrounding tissues despite the existence of a capsule around the mass and the hypervascularity in the bulk, mostly in the hyaline vascular type. Probably the mediastinal site of UCD should be considered before to define surgical strategy. A review of the Japanese literature showed only few cases of bleeding or tight adhesions of UCD when it is located in the anterior mediastinum (amUCD). Conversely, these complications are more common when UCD is located in the posterior mediastinum (pmUCD). Thus, VATS is probably more indicated for amUCD while pmUCD should best be addressed directly with thoracotomy or quickly converted from VATS to thoracotomy in case of initial bleeding.[19]

LUNG CD

Parenchymal lung involvement in CD is extremely rare, its radiological and clinical non-specific signs may mimic a community-acquired pneumonia, tuberculosis or lung malignancies. A wide spectrum of lung presentation of CD is reported in literature: nodules or masses, patchy consolidation, ground glass opacities, peribronchovascular thickening and cysts.
Diffuse cystic lung diseases (DCLDs) are a group of different pulmonary disorders with variable pathophysiology characterized by the presence of thin-walled, air-filled spaces within the lung parenchyma. Although rarely, lung CD can occur as DCLD. It can cause irreversible lung remodeling including displacement, destruction and replacement of distal airways, alveolar septa and small vessels.[20] An interesting retrospective analysis performed in a tertiary Chinese hospital described 22 cases of CD-associated diffuse parenchymal lung disease (DPLD) between 1999 and 2015. The most frequently reported symptoms were cough (72.7%), fever (68.2%), and dyspnea (59.1%). In high-resolution chest Computed Tomography (CT) the most frequent finding was lymphadenopathy (81.8%), followed by multiple nodules of different sizes (72.7%), cysts (59.1%), and patches of ground-glass opacity (54.5%). In six patients, CT images showed a lymphocytic interstitial pneumonia (LIP)-like. To make the diagnosis mostly superficial lymph node biopsies (63.6%) were performed, followed by video-assisted thoracic surgery lung biopsies (27.3%), and in a fewer cases CT-guided percutaneous lung biopsies or endoscopic lymph node biopsies. The plasma cell variant was the most common pathological subtypes. Thirteen patients performed pulmonary function tests and only 4 showed airflow obstruction with no significant response to a bronchodilator test. The other 9 patients had normal values. Instead, it is interesting to note that most patients had impaired gas exchange, with a decreased diffusion capacity of the lung for carbon monoxide (DLCO).[21]
All patients enrolled in the study underwent chemotherapy except two patients, one because refused chemotherapy and the other because only with few symptoms (mostly slight fatigue). Fifteen patients received classic chemotherapy with CHOP (cyclophosphamide, adriamycin, vincristine, and corticosteroid) which is the most widely used therapeutic scheme for CD. One patient received CHOP associated to etoposide and another one performed CHOP with anti-CD20 receptor. Of these patients 14 improved, 2 were stable, 2 were refractory, 1 was lost during the follow-up and 3 died. Of the latter one patient died of several pulmonary infection after the CHOP treatment and one died of severe pulmonary hypertension associated with heart failure. The last one patient died of respiratory failure resulting from Bronchiolitis Obliterans (BO), despite the chemotherapy.[21]
In fact, BO is a rare and severe complication of CD. Bilateral lung transplantation may represent a possible therapeutic solution in patients with BO due to CD even if no reports on the safety or outcomes of bilateral lung transplantation are present in literature. In a recent, article Yue et al. have reported medical records of six consecutive patients who underwent bilateral lung transplantation between December 2012 and December 2020. The mean age of patients at lung transplantation was 33 $\pm$ 15 years, with a mean time from diagnosis to lung transplantation of 4.1 $\pm$ 2.7 years. Interestingly CD did not recur in any of the patients confirming that bilateral lung transplantation is a viable and safe treatment for selected patients with CD and BO, which can improve the quality of life and prolong survival.22
A case of a 44 years-old woman reported by Peng et al. is useful for understanding the importance of follow-up in patient affected by lung CD. Patient presented at the hospital for pulmonary opacities revealed on routine chest X-

ray and a Chest CT showed multiple bilateral nodules and mediastinal lymphadenopathies. A thoracoscopic biopsy was performed, and the histological examination revealed polyclonal characteristics, suggesting CD of the plasma cell subtype. She did not received treatment because asymptomatic. At 4-year follow-up, the CT revealed pulmonary nodules associated with clusters of cysts but the patient remained asymptomatic. Later the woman was not subjected to further examination until ten years after the first access to the hospital, she was admitted again with slight dyspnea. Chest CT showed diffuse cysts and a CT-guided percutaneous lung biopsy confirmed plasmacytic and lymphocytic infiltration, typical of MCD. The patient received a therapy with bortezomib, cyclophosphamide and dexamethasone obtaining regression of the symptoms. Chest CT performed after one year revealed considerably decrease in size of the lung nodules while the cysts remained stable but the pulmonary function tests showed a significant reduction of FEV1, FVC and DLCO. This suggests that lung involvement of MCD can, even if slowly, progressively damage the lung parenchyma. Close follow-up is therefore necessary to control disease progression and to start treatment on time.[23]

CHEST WALL CD

Chest wall involvement in Castleman's disease is a rare event. In 2006 Tanaka et al. reported a case of a 60-year-old woman with a right thoracic wall localization of CD and in their article performed a review of the literature. They found that in that during the previous 20 years, only 10 cases of thoracic wall CD involvement were described. This event was more frequent in woman, on the right side and, histologically, the hyaline-vascular type was the dominant variant.[24]

In 2009 a very unusual presentation of CD has been reported by Ueda et al. They reported a primary CD arising from a surgical wound (on the posterior portion of the previous posterolateral incision) in a 33-year-old female with a history of thoracic surgery at 5 years of age.[25]

Another curious presentation of CD was reported by Reynold et al. A 51 years old man native of West India but resident in England presented to the hospital with dyspnea and massive right pleural effusion with mediastinal shift to the left at chest X-ray. Chest CT performed after pleural effusion drainage showed a mass anteriorly, at the cardiophrenic angle with adhesion to the right pericardium and involving the phrenic nerve. The mass had spread posteriorly over the right hemidiaphragm and to the chest wall. An explorative thoracotomy was performed with an incomplete excision of the mass due to the proximity to the phrenic nerve. A parietal pleurectomy was carried out. A diagnosis of Castleman's disease of mixed or transitional type was made. The patient was quickly discharged home in good condition and at the 9-month check-up the patient was fine with no systemic complaints.[26]

In 2003 Wilkinson et al reported a very strange case of a 35-year-old man with axillary lymphadenopathy, hepatosplenomegaly and skin erythema associated with a lesion of a 10th right rib. The biopsy of axillary lymph nodes was performed with a diagnosis of CD. Then a right thoracotomy was carried out over the 10th rib. The mass was palpable in the 10th rib extending up on to the 9th rib. Three quarters of the 9th and 10th ribs were resected en bloc with the surrounding tissue. However, histologic examination of the mass showed typical characteristics of a plasmacytoma. Plasmacytoma is infrequently associated with CD but we should consider it when we find bone lesions in a patient suffering from this disease.[27]

RARE THORACIC PRESENTATION OF CD

Other rare thoracic presentations of Castleman's disease are the lesions of the soft tissues of the chest wall such as intercostal muscles or diaphragm.

In 2012 Eloueriachi et al. reported a case of a 47-year-old woman with a right paracardiac basal opacity giving an appearance of localized elevation of the right diaphragmatic coupola on chest X-ray. The thoraco-abdominal computed tomography showed the presence of a mass measuring 55 mm long axis, of mixed structure well limited, which was enhanced after injection of contrast product, projecting onto the anterior part of the diaphragm. An exploratory thoracotomy passing through the 6th right intercostal space was performed, revealing a firm, richly vascularized mass approximately 6 cm long, continuous with the diaphragm and located at the level of the right anterior mediastinal sinus. This mass was totally excised from the diaphragm without breaking it, then was extracted after ligature-section of its pedicle. The diagnosis of Castleman's disease of the hyalino-vascular type was performed and clinical and radiological follow-up spread over a period of 10 months revealed no recurrence of pathology.[28]

A case of CD originating from the intercostal space was reported by the group of Rena et al. They described the case of a 47-year-old woman arrived at the hospital after an incidental finding of left para-aortic opacity of the chest in radiographs carried out for routine occupational

control. On CT images, the lesion appeared as well-marginated soft-tissue mass in the left costovertebral sulcus with minimal quantity of pleural fluid associated. Fine-needle aspiration biopsy was performed, but it resulted not diagnostic. Left lateral thoracotomy was carried out and 200 ml of pleural fluid were aspirated and sent for microbiology and cytology. The mass located in the costovertebral sulcus, from the third to fifth intercostal space was capsulated and covered by the parietal pleura and tenaciously adherent to the chest-wall. Partial parietal pleurectomy was achieved and the excision of the mass with soft surrounding tissues was carried out totally extrapleural. The woman was discharged on day six.[29]

To complete the scenario of rare clinical presentation, in 2018 we published a case of a 67-year-old patient came to our Institute for a persistent cough. The CT scan images revealed a right lower lung opacity with mediastinal adenopathy miming a metastatic lung cancer.

We performed an invasive staging of mediastinal lymph-nodes 4R and 4L but these resulted negative for nodal metastasis. Then the patient underwent an explorative thoracotomy that showed mediastinal, hilar and interscissural adenopathies. Fine needle aspiration biopsy revealed a nonsmall cell in the context of lymphoid tissue, a not conclusive diagnosis for lymphoproliferative disease or metastatic lung carcinoma. Thus, a right lower lobectomy with extended lymphadenectomy was performed. At the definitive histological exam, the neoplastic elements expressed the immunophenotypic profile typical of plasmablastic variant CD. A classic chemotherapy associated with rituximab for six cycles was successfully performed. At 5-year follow-up the patient was in good general condition with no evidence of relapse of the disease.[30]

In the table 1 are reported all clinical thoracic manifestation of CD mentioned in our work.

Table1

Thoracic district	Author	Kind of article	Year	Variant of CD described	Radiological findings	Clinical presentation	Treatment
MEDIASTINUM	Legras[16]	Retrospective analysis	2018	**UCD**	**SML MM: 73% AM: 18% PM: 9%**	**No symptoms or CRS**	**CE of the LA in: PL TCT (55%) pSTM (18%) pSTM (18%) tSTM (18%) tSTM (18%) VATS (9%)**
	Bıçakçıoğlu[17]	Retrospective analysis	2014	**UCD: 95%** MCD: 5%	**SML MM:37%AM:16%PM:42% SML + LI: 5%**	**No symptoms or CRS(SML), Dyspnea+ weight loss (SML+LI)**	**SML: CE of the LA in TCT(78%) MPY ->STM(5%) VATS (16%) SML+ LI: cCT + antiCD20R**
	Talat[18]	Systematic review	2012	**UCD: 68% MCD: 32%**	**SML or SVL in UCD ML +/- fever, dyspnea, weight loss, sweating in MCD**	**No symptoms or CRS in UCD**	**CE of the LA in UCD HT+CT in MCD**
	Iyoda[19]	Case report + Review	2000	**UCD**	**SML in PM**	**No symptoms or CRS**	**CE of the LA in: TCT Only 1 case: VATS -> TCT**
LUNG	Ma[20]	Case report	2020	**MCD**	**DCLD**	**Dyspnea, cough, anorexia, night sweats, and fatigue**	**Irreversible condition. Anti-inflammatory therapy to stop the progression.**
	Huang[21]	Retrospectve analysis	2017	**MCD**	**Lung multiple nodules, Cysts, GGO, Air trapping , Interlobular septal and bronchovascular bundles thickening, Localized consolidation, LIP-like patterns**	**Coughing, fever, and dyspnea**	**cCT +/- antiCD-20R**
	Peng[23]	Case report	2020	**MCD**	**Pulmonary nodules, Mediastinal lymphadenopathy, Cluster of pulmonary cysts**	**Dyspnea**	**cCT**
CHEST WALL	Tanaka[24]	Case report	2006	**UCD**	**Right upper posterior tumor on the chest wall (IRF)**	**No symptoms**	**Only radiologic FU**

	Ueda[25]	Case report	2009	**UCD**	**Tumor arising from surgical wound (previous postero-lalteral incision)**	**Left-shoulder pain**	**CE of tumor in TCT+ Resection of 3rd +4th ribs**
	Reynolds[26]	Case report	1992	**UCD**	**Massive pleural effusion + Tumor of right chest wall**	**Dyspnea + Weight loss**	**CE of tumor in TCT + PPI**
	Wilkinson[27]	Case report	2003	**MCD + Plasmacytoma**	**Axillary lymphadenopathy+ 10th rib lesion**	**Axillary lymphadenopathy, hepatosplenomegaly, skin erythema**	**CE of lesion in TCT + Resection of 10th +3/4 ot the 9th ribs**
OTHER RARE THORACIC CD	Eloueriachi[28]	Case report	2012	**UCD**	**Mass of anterior diaphragm (IRF)**	**No symptoms**	**CE of lesion in TCT**
	Rena[29]	Case report	2001	**UCD**	**Mass of left costovertebral sulcus + Pleural effusion (IRF)**	**No symptoms**	**CexplE of the mass in TCT + pPPI**
	Testori[30]	Case report	2018	**MCD**	**Right lower lung opacity + Mediastinal Lymphadenopathy**	**Persistent cough**	**RLL + LAct -> cCT + antiCD-20R**

Table1. CD: Unicentric Castleman Disease; MCD: Multicentric Castleman; SML: Solitary Mediastinal Lymphadenopathy; SVL: Solitary Visceral Lymphadenopathy; ML: Multiple Lymphadenopathy; MM: Middle Mediastinum; AM: anterior mediastinum; PM: Posterior mediastinum CE: Complete Excision; TCT: Thoracotomy; LA: Lymphadenopathy; PL TCT: Postero-Lateral Thoracotomy;; MPY: Mediastinoscopy; STM: Sternotomy; p/t STM: partial/total sternotomy; VATS: Video-Assisted Thoracoscopic Surgery; PPI: Parietal Pleurectomy; pPPI: partial Parietal Pleurectomy; LI: lung Involvement; HT: Immunotherapy IRF: Incidental Radiological Finding; CRS: Compress Related Symptoms; Disease; DLCD: Diffuse Lung Cysts Disease; cCT: classical chemotherapy;anti-CD20R: anti-CD20 receptor therapy; FU: follow-up; CexplE: Complete Extrapleural Excision; RLL: Right Lower Lobectomy; LAct: LymphoAdenectomy

CONCLUSION

In this mini-review we have reported all clinical thoracic manifestations of the CD. They can be very different ranging from solitary or multiple lymphadenopathies, mainly located in the mediastinum to lung manifestation in form of nodules, cystis, ground glass opacities, interstitial pneumonia. Moreover, rare forms of CD involving the chest wall and the soft tissues of chest wall, occasionally associated with pleural effusion have been described.

CD diagnosis is often difficult to obtain with an only fine-needle biopsy and in the most of the cases it is necessary performing a complete surgical excision of the lesion. Therefore, in UCD surgical excision can result in both a diagnostic and therapeutic tool. Conversely, if the localization of CD is multiple (MCD), after obtaining the diagnosis, chemotherapy is the treatment of choice. One of the most effective therapeutic regimens for MCD is CHOP (cyclophosphamide, doxorubicin, vincristine, and prednisone). In the last few years, immunotherapy has also proved very useful for MCD. Indeed, the humanized monoclonal antibody to CD20 (Rituximab) used either as monotherapy or in combination with chemotherapy gave great results, particularly in patients with MCD and HIV and/or HHV+.[31]

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CONFLICT OF INTEREST

The authors declare no potential conflict of interest with respect to research, authorship and/or publication of this article.

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