Case Report

Challenges in the diagnosis of an unusual retroperitoneal pararenal well-differentiated Liposarcoma: A case report unraveling the complexities of genetic alterations and histomorphology

Shreeya Taresh Indulkar[1], B Vishal Rao[2], Sandhya Devi Gundimeda[3], Suseela Kodandapani[4], Rakesh Sharma[5], Subramanyeshwar Rao Thammineedi[6]

From [1]Fellow, [2]Senior Consultant Pathologist, [3]Consultant Cytogeneticist, [4]Head, Department of Pathology, [5]Consultant Urologist, [6]Medical Director and Chief of Surgery, Department of Surgical Oncology, Basavatarakam Indo American Cancer Hospital And Research Institute, Hyderabad, Telangana, India

ABSTRACT

Liposarcomas (LPS) presents diagnostic challenges, particularly when located in pararenal retroperitoneal regions near vital organs such as the kidneys and adrenal glands. Herein, we report a novel case of well-differentiated liposarcoma (WDLPS) with myxoid features and murine double minute 2 (MDM2)-DNA damage-inducible transcript 3 (DDIT3) co-amplification in a 50-year-old male, presented with right-sided abdominal pain and a palpable mass in the lumbar and hypogastric regions. Imaging revealed a large retroperitoneal mass displacing the adjacent organs. Histological examination showed a poorly circumscribed hypocellular mass comprised of stellate spindle cells in abundant myxoid stroma. Focal areas of increased cellularity and frequent mitoses were noted. Overall histological and immunohistochemistry suggested diagnosis of myxoid liposarcoma, however, Fluorescence *in situ* hybridization (FISH) test showed no fusion of FUS-DDIT3 fusion, typically seen in myxoid liposarcoma. Occasional lipoblasts were seen. FISH test for MDM2 and DDIT3 showed co-amplification of both genes. The overlap between WDLPS, MLS, and Dedifferentiated LPS highlights the necessity of thorough histological and genetic analyses for accurate diagnosis and treatment planning. This case underscores the recognition of DDIT3 amplifications and its association with ML-like changes in LPS, which further can aid in resolving diagnostic challenges, particularly in cases with atypical morphological features.

Key words: DNA damage-inducible transcript 3, Fluorescence *in situ* hybridization, Immunohistochemistry, Murine double minute 2, Myxoid liposarcoma, Pararenal liposarcoma, Well-Differentiated Liposarcoma

Liposarcomas (LPS), a type of slow-growing soft-tissue sarcoma, are predominantly observed in retroperitoneal locations [1]. The global incidence of Liposarcoma is approximately 1–2 cases/1,000,000 individuals annually. Although liposarcoma is relatively rare, it constitutes approximately 15–20% of all soft-tissue sarcomas, positioning it among the more prevalent subtypes within this heterogeneous category of malignancies. The incidence of liposarcoma exhibits minor variation influenced by geographic and demographic factors; however, the overall global incidence remains relatively stable. In the United States, liposarcoma is diagnosed at a slightly higher rate, with an estimated 2,000–2,500 new cases annually, predominantly affecting adults [2,3]. Atypical lipomatous tumors (ALT) most commonly arise in the deep soft tissues of the proximal extremities, particularly the thigh and buttock, as well as the trunk, including the back and shoulder. They can also involve the retroperitoneum and paratesticular regions [4]. For tumors located in areas such as the retroperitoneum, spermatic cord, or mediastinum, which are associated with a higher risk of disease progression, the designation "well-differentiated liposarcoma (WDLPS)" is often retained due to their aggressive potential [5].

Pararenal retroperitoneal LPS presents significant diagnostic challenges due to their close proximity to critical structures such as the kidneys and adrenal glands. WDLPS is characterized by the amplification of the q13–15 region of chromosome 12, whereas myxoid liposarcoma (MLPS) is primarily identified by FUS-DNA damage-inducible transcript 3 (DDIT3) gene fusion [6,7]. In this context, we present a case of liposarcoma arising in a pararenal

Access this article online	
Received - 06 September 2024 Initial Review - 21 September 2024 Accepted - 30 December 2024	Quick Response code
DOI: 10.32677/ijcr.v11i1.4808	

Correspondence to: Dr. B. Vishal Rao, MD Department of Pathology, Basavatarakam Indo-American Cancer Hospital and Research Institute, Banjara Hills, Road number 10, Hyderabad, 500034, Telangana, India. E-mail: vishalrao1980@gmail.com

retroperitoneal location, demonstrating the combined morphologic features of WDLPS and MLPS. This unique case underscores the importance of recognizing and understanding the morphological and genetic complexities of LPS and emphasizes the role of cytogenetic testing in achieving accurate subtype classification.

CASE PRESENTATION

A 50-year-old male presented with right-sided abdominal pain for 8 months, prompting clinical examination that revealed a palpable mass in the right side of the abdomen.

On general examination, he was found to be moderately built and moderately nourished, with an ECOG performance status of I. His vitals were stable, and the following measurements were recorded: blood pressure – 130/70 mmHg, pulse – 72 bpm, SpO_2 – 96%, temperature – 98.6°F, and respiratory rate – 18/min. On local examination, a palpable mass was identified in the right lumbar and hypogastric regions. The mass measured approximately 12 cm in size and was smooth in consistency with well-defined upper margins. The deep margins were not palpable. The swelling was mobile to some extent with bimanual pushing, but not on simple palpation. Tenderness was present on palpation, and the temperature of the swelling was normal.

His complete blood count was within normal limits, and urine examination showed abnormal findings of 1–2 RBCs/hpf. Contrast-enhanced computed tomography (CECT) abdomen and pelvis revealed a huge mass, suspected to originate from the renal/pararenal area, causing displacement of the kidney anterolaterally and pushing the adjacent organs, that is, liver, duodenum, and pancreas (Fig. 1a and b). USG-guided biopsy confirmed a poorly differentiated neoplasm with atypical cells showing abundant clear to vacuolated cytoplasm.

Immunohistochemistry (IHC) was performed, which showed CD56 and CD99 positivity, and negative staining for PAX-8, AMACR, Melan-A, NKX2.2, desmin, WT1, chromogranin, and synaptophysin. Ki67 was 40%. Due to limited tissue, further investigations could not be performed. A biopsy diagnosis of poorly differentiated malignancy was rendered, possibly of adrenal origin. Subsequently, the patient underwent an open en bloc resection of the pararenal mass with right radical nephrectomy and adrenalectomy. Intraoperatively, the mass was found to be adherent to the right side of the diaphragm, encasing the right renal vessels and compressing the right kidney and adrenal gland. Enlarged nodes in the paracaval and interaortocaval regions were observed. The rest of the viscera was normal.

On gross examination, a multilobular gray-white soft-tissue mass with attached en bloc resected kidney and adrenal gland was observed. The mass measured 18.5×15×11 cm with a focal yellowish cut surface and necrotic, calcified areas on the cut surface. The mass was seen pushing the right kidney without any gross infiltration into the renal parenchyma (Fig. 1c and d). The adrenal gland was not involved. The cut surface of the kidney and adrenal gland did not reveal any gross abnormalities.

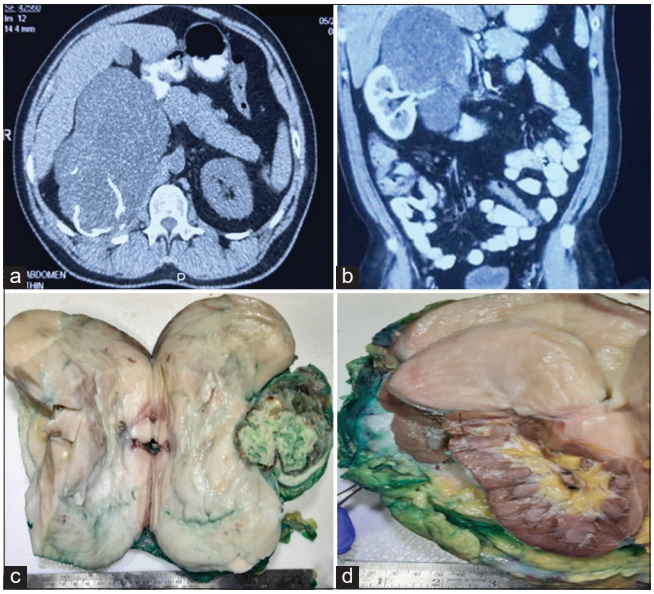

Figure 1: (a) CT abdomen coronal section showing pararenal mass. (b) Mass displacing the kidney laterally is seen in the sagittal section of contrast-enhanced CT abdomen. (c) En bloc resection shows a huge 18.5 cm mass with yellowish cut surface and focal necrotic and calcified areas. (d) Mass displacing the right kidney laterally without gross infiltration of the renal parenchyma

Histological examination revealed a poorly circumscribed mass with variable cellularity, composed of atypical stellate cells arranged in loose fascicles and sheets against abundant lightly basophilic, myxoid stroma with a striking plexiform, delicately arborizing capillary network. Focal areas (5%) with hypercellularity and cells showing mild to moderately enlarged hyperchromatic nuclei were identified (Fig. 2a and 2b). Occasional lipoblasts were identified. Mitoses were observed (6–7/10 hpf), and small areas of osseous metaplasia were also noted. Lymphovascular invasion, necrosis, and evidence of heterologous elements/dedifferentiation were not identified. All resection margins were tumor-free. No infiltration of the renal parenchyma or adrenal gland was identified by microscopy. On IHC, tumor cells exhibited p16 strong nuclear positivity (Fig. 2c). There was patchy S100 positivity (Fig. 2d), and tumor cells were negative for CDK4, TLE1, BCL2 and nuclear beta-catenin. Murine double minute 2 (MDM2) IHC was not available, however, MDM2 Fluorescence *in situ* hybridization (FISH) test was suggested. A morphological diagnosis of MLPS was made, and FISH test was performed for further confirmation.

FISH examination was performed on 4-µm-thick sections of formalin-fixed paraffin-embedded tissue (FFPE). A representative FFPE block containing tumors with both areas of cellularity was selected. FISH for MDM2 was performed using a ZytoLight® SPEC MDM2/CEN12 dual color break-apart probe (Zytovision GmbH Bremerhaven Germany) with the target human MDM2 gene located at 12q13 labeled in green and chromosome 12 alpha satellites that served as the reference labeled in orange. The hybridization signals of 50 non-overlapping nuclei were visualized under an Olympus BX41 fluorescence microscope in oil immersion using appropriate filters, and the number of target and reference signals for all 50 cells were summed. The ratio of MDM2/CEN12 signals

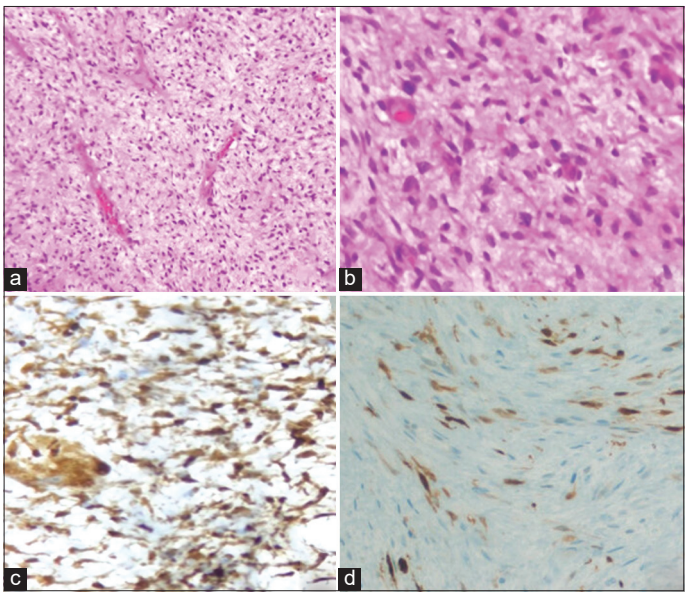

Figure 2: (a) hematoxylin and eosin stain-stained section of the mass showed atypical cells with plump oval to spindled nuclei arranged in loose fascicles and sheets. Intervening thin curvilinear vessels seen. (b) Tumor cells are mildly pleomorphic and show nuclear hyperchromasia (c) tumor cells showing P16 nuclear immunohistochemical(IHC) staining. (d) Tumor cells are negative for S100 IHC

was calculated. MDM2 gene amplification was detected with an MDM2:CEN12 ratio of 5.3 (Fig. 3a). FISH for FUS-DDIT3 gene fusion was performed using a ZytoLight® SPEC DDIT3 (CHOP) Dual color break-apart probe to detect translocations involving the DDIT3 gene at 12q13.3. No fusion of the FUS-DDIT3 genes was observed. However, amplification of the DDIT3 probe region (at 12q13) was observed in all the nuclei examined (Fig. 3b). FISH for FUS and EWSR1 gene rearrangement was also performed, and no gene fusions were observed.

A final diagnosis of WDLPS exhibiting MLPS -like morphology with MDM2 and DDIT3 co-amplification was made. After surgery, the patient underwent a planned course of six cycles of Ifosfamide and carboplatin chemotherapy, achieving good health. Ongoing follow-up with routine investigations was done to monitor treatment response and overall well-being. With a further follow-up of 1.5 years, the patient remains healthy and alive.

DISCUSSION

LPS is the most common histological type of retroperitoneal sarcoma, accounting for approximately 15–20% of all sarcomas in adults [1,8]. In the pararenal retroperitoneal location, differential diagnoses include various entities ranging from benign to malignant, such as perivascular epithelioid cell tumor, angiomyolipoma, adrenal cortical neoplasms, clear cell sarcoma, and renal cell carcinoma (RCC). The recent 2022 WHO classification of soft-tissue neoplasm includes WDLPS, Dedifferentiated LPS (DDLPS), MLPS, pleomorphic LPS (PLPS), and myxoid pleomorphic LPS (MPLPS) as the malignant neoplasm of adipocytic origin [9]. The unique histological and molecular profile of different malignant soft-tissue neoplasms is described in Table 1. Diagnostic challenge

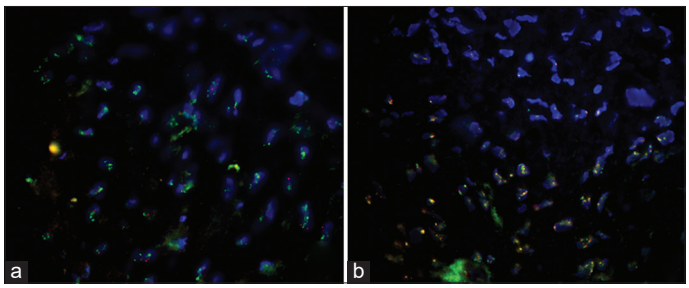

Figure 3: (a) DNA damage-inducible transcript three amplification and (b) murine double minute amplification on Fluorescence *in situ* hybridization

is further heightened by the morphological heterogeneity often observed in retroperitoneal tumors, where multiple patterns may coexist within the same lesion.

ALT/WDLPS is characterized by supernumerary ring and giant marker chromosomes, usually alone or with other numerical or structural abnormalities [4]. Telomeric associations are common and may give ALT/WDLPS karyotypes a complex appearance [4,10]. Both supernumerary rings and giant markers contain amplified sequences originating in the 12q14-q15 region, MDM2 (12q15) being the main driver gene [4]. Many genes in the 12q14-q15 region, including TSPAN31, CDK4, HMGA2, YEATS4, CPM, and FRS2, are coamplified with MDM2 [4,11-13]. They always coamplify at least one genomic segment in addition to 12q14-q15 [4]. Ancillary techniques such as IHC and FISH, or reverse transcription polymerase chain reaction, can be very helpful in supporting the distinction of these malignant soft-tissue neoplasms of adipocytic origin.

In our case, a small biopsy revealed a poorly differentiated malignant neoplasm, necessitating an extensive IHC panel to exclude various differentials. However, no conclusive diagnosis was reached due to the limited amount of tissue. Sioletic *et al*., first observed the occurrence of MLPS-like characteristics in MDM2-amplified liposarcoma [14]. They identified myxoid stroma in 56 cases, which included 22 well-differentiated and 34 DDLPS. In addition, they noted a MLPS-like vascular pattern in 11 out of the 22 WDLPSs [14]. Nevertheless, our case did not exhibit the characteristic FUS-DDIT3 fusion commonly observed in MLPS.

DDIT3 also known as CHOP or GADD153 plays a complex role as a phenotype-directing factor in lipomatous tumors [15]. DDIT3 is typically activated in response to cellular stress and inhibits the process of adipocytic terminal differentiation. In WDLS/DDLS tumors, DDIT3 is expressed in a substantial proportion of cells, ranging from 40% to 94%, likely due to recurrent gene amplicons carrying DDIT3, resulting in constitutive expression [15]. In MLS, DDIT3 expression is a component of fusion oncoproteins FUS-DDIT3 or EWSR1-DDIT3, detectable in a subset of tumor cells, indicating post-transcriptional regulation, with similar proportions across cases [15-17]. Further, Kåbjörn Gustafsson *et al*. found cytoplasmic DDIT3 expression, with occasional nuclear staining in abnormal nuclei, in PLS, possibly induced by stress-related factors such as DNA damage and hypoxia [15]. Notably, benign lipomas also show DDIT3 expression in 15–81% of cells, despite lacking amplification or

Table 1: Clinicopathological features and molecular profile of malignant adipocytic neoplasm.[10,19-20]

Differential diagnosis	ALT/WDLPS	MLPS	DDLPS	PLPS	MPLPS
Clinical features					
Age (peak)	4th–5th decade	4th–5th decade	6th decade	7th decade	<3rd decade
Gender predilection	No sex predilection	No sex predilection	Male	Male	Female
Typical site of origin	Extremities Retroperitoneum Rarely at paratesticular and mediastinum	Thigh; other proximal extremities	Extremities, retroperitoneum, paratesticular, mediastinum, head and neck	Proximal and distal extremities	Mediastinum Rarely at thigh, head and neck, perineum, abdomen, and back
Typical histology	Lipoma-like ALT/WDLPS: variation in adipocytic size with nuclear atypia in stromal and/or adipocytic cells; sclerosing: hyperchromatic bizarre stromal cells in a fibrillary sclerotic background; inflammatory: scattered atypical stromal cells in a chronic inflammatory background; lipoblasts are not needed for diagnosis.	Myxoid matrix containing delicately arborizing capillaries; bland round to ovoid cells; variable number of small non-pleomorphic lipoblasts,	Transition (abrupt or gradual) from WDLPS (of any type) to spindle cell and pleomorphic non-lipogenic (rarely lipogenic) tumor (of low grade or high grade).	Pleomorphic spindle cell sarcoma containing a variable number of pleomorphic lipoblasts; myxofibrosarcoma-like morphology with pleomorphic lipoblasts; epithelioid subtype with sheets of carcinoma-like epithelioid cells with pleomorphic lipoblasts;	Distinctive admixture of relatively bland zones resembling conventional myxoid liposarcoma and much more cellular and atypical areas, resembling pleomorphic liposarcoma
IHC					
CDK4	Positive	Negative	Positive	Negative	Negative
MDM2	Positive	Negative	Positive	Negative	Negative
Other positive IHC stains	S100, CD34	S100, DDIT3	p16	Keratins (epithelioid type)	p16, S100, CD34
Molecular features					
MDM2/CDK4 amplification	Yes	No	Yes	No	No
FUS-DDIT3 fusion	No	Yes (Rare cases show EWSR1-DDIT3 fusion)	No	No	No
Complex karyotype	No	No	Yes	Yes	Yes
TP53 mutation	In rare pediatric cases associated with Li–Fraumeni syndrome	No	Yes	Yes	Yes
RB1 loss	No	No	Variable	Yes	Yes
TERT promoter mutation	No	Yes (50%)	Yes	No	No
Patterns of recurrence	Local recurrence Little to no metastatic potential	Local and/or metastatic (bone, soft tissue, serosa) in up to 40%	Local recurrence – approximately 40% Metastasis (lung) – approximately 20-30%	Local recurrence – approximately 30–40% Metastasis (lung) – approximately 30–50%	Local recurrence and distant metastasis (lung)– approximately 50%
Response to chemotherapy	Poor	Typically, sensitive to chemotherapy and radiotherapy	Poor	Variable chemosensitivity	Limited data, Variable chemosensitivity

DDLPS: Dedifferentiated liposarcomas, MLPS: Myxoid liposarcomas, PLPS: Pleomorphic liposarcomas, MPLPS: Myxoid pleomorphic Liposarcomas, DDIT3: DNA damage-inducible transcript 3

rearrangement of the DDIT3 gene, suggesting a link to terminal adipocyte differentiation [15]. The presence of DDIT3 may be linked to a terminal differentiation program, as lipomas undergo adipocyte differentiation. The abnormal expression of DDIT3 in liposarcoma may hinder adipocyte differentiation, potentially explaining the presence of lipoblasts [15].

DDLPS shares a genetic overlap with ALT/WDLPS. Both conditions are distinguished by the frequent amplification of MDM2 genes located at 12q14-q15. DDIT3 is found to be amplified in 33% of DDLPS cases and is strongly correlated with the presence of MLPS-like characteristics, as compared to cases without amplification [18]. Even after extensive tumor sampling, our case did not exhibit genuine pleomorphic lipoblasts, as seen in PLPS; or a sudden or gradual transformation into a non-lipogenic low- or high-grade sarcoma, which is a crucial diagnostic characteristic of DDLPS. The differentiation between WDLPS and DDLPS is significant because DDLPS exhibits a higher incidence of local recurrences, approximately 40% [9]. The recently described Myxoid pleomorphic LPS 'MPLPS' do not show MDM2, DDIT3 amplifications, or FUS-DDIT3 fusion [9,19,20].

A recent study by Murshed *et al*. reported the first documented case of WDLPS with co-amplification of MDM2 and DDIT3 in the hypopharyngeal space [21]. A submucosal tumor comprised of lobules of atypical oval to spindle non-lipogenic cells in a prominent myxoid stroma with arborizing chicken-wire vasculature was observed on microscopic examination. Lobules of adipose tissue, surrounded by thick fibrous septa that contained atypical hyperchromatic spindle cells, were observed in the vicinity. No definite lipoblasts/necrosis was found and the mitosis was 2/10 HPFs. Both tumor cell components showed CDK4 reactivity, but no MDM2 IHC positivity [21].

CONCLUSION

WDL of the pararenal or retroperitoneal region is common, and some cases can have prominent myxoid stroma and features resembling ML. Due to its close proximity to the kidney and adrenal gland, this tumor can be misdiagnosed as either a clear cell RCC or an adrenal tumor based on radiological findings. The scarcity of tissue on biopsy is a significant barrier to accurately diagnosing and identifying the transitioning areas observed in DDLPS. IHC and cytogenetic studies are essential for a definitive diagnosis in such cases. Awareness of the fact that DDIT3 gene amplification is associated with ML-like changes and can be seen in a WDLPS or DDLPS is essential, which may also help to solve diagnostically challenging cases.

REFERENCES

1. Horowitz J, Singhal M, Marrero D, Bashjawish F, Leto D, Winters M, *et al*. A multi-modality treatment of retroperitoneal de-differentiated liposarcoma. Am J Case Rep 2020;21:e919245.
2. Bock S, Hoffmann DG, Jiang Y, Chen H, Il'yasova D. Increasing incidence of liposarcoma: A population-based study of national surveillance databases, 2001-2016. Int J Environ Res Public Health 2020;17:2710.
3. de Pinieux G, Karanian M, Le Loarer F, Le Guellec S, Chabaud S, Terrier P, *et al*. Nationwide incidence of sarcomas and connective tissue tumors of intermediate malignancy over four years using an expert pathology review network. PLoS One 2021;16:e0246958.
4. Lee AT, Thway K, Huang PH, Jones RL. Clinical and molecular spectrum of liposarcoma. J Clin Oncol 2018;36:151-9.
5. Ameloot E, Cordier F, Van Dorpe J, Creytens D. Update of pediatric lipomatous lesions: A clinicopathological, immunohistochemical and molecular overview. J Clin Med 2022;11:1938.
6. Sandberg AA, Bridge JA. Updates on the cytogenetics and molecular genetics of bone and soft tissue tumors: Chondrosarcoma and other cartilaginous neoplasms. Cancer Genet Cytogenet 2003;143:1-31.
7. Powers MP, Wang WL, Hernandez VS, Patel KS, Lev DC, Lazar AJ, *et al*. Detection of myxoid liposarcoma-associated FUS-DDIT3 rearrangement variants including a newly identified breakpoint using an optimized RT-PCR assay. Mod Pathol 2010;23:1307-15.
8. Keung EZ, Hornick JL, Bertagnolli MM, Baldini EH, Raut CP. Predictors of outcomes in patients with primary retroperitoneal dedifferentiated liposarcoma undergoing surgery. J Am Coll Surg 2014;218:206-17.
9. WHO Classification of Tumours Editorial Board. Soft Tissue and Bone Tumours. WHO Classification of Tumours Series. 5th ed., Vol. 3. Lyon (France): International Agency for Research on Cancer; 2020.
10. Mandahl N, Mertens F, Willén H, Rydholm A, Kreicbergs A, Mitelman F. Nonrandom pattern of telomeric associations in atypical lipomatous tumors with ring and giant marker chromosomes. Cancer Genet Cytogenet 1998;103:25-34.
11. Barretina J, Taylor BS, Banerji S, Ramos AH, Lagos-Quintana M, Decarolis PL, *et al*. Subtype-specific genomic alterations define new targets for soft-tissue sarcoma therapy. Nat Genet 2010;42:715-21.
12. Erickson-Johnson MR, Seys AR, Roth CW, King AA, Hulshizer RL, Wang X, *et al*. Carboxypeptidase M: A biomarker for the discrimination of well-differentiated liposarcoma from lipoma. Mod Pathol 2009;22:1541-7.
13. Wang X, Asmann YW, Erickson-Johnson MR, Oliveira JL, Zhang H, Moura RD, *et al*. High-resolution genomic mapping reveals consistent amplification of the fibroblast growth factor receptor substrate 2 gene in well-differentiated and dedifferentiated liposarcoma. Genes Chromosomes Cancer 2011;50:849-58.
14. Sioletic S, Dal Cin P, Fletcher CD, Hornick JL. Well-differentiated and dedifferentiated liposarcomas with prominent myxoid stroma: Analysis of 56 cases. Histopathology 2013;62:287-93.
15. Kåbjörn Gustafsson C, Engström K, Aman P. DDIT3 expression in liposarcoma development. Sarcoma 2014;2014:954671.
16. Panagopoulos I, Höglund M, Mertens F, Mandahl N, Mitelman F, Aman P. Fusion of the EWS and CHOP genes in myxoid liposarcoma. Oncogene 1996;12:489-94.
17. Crozat A, Aman P, Mandahl N, Ron D. Fusion of CHOP to a novel RNA-binding protein in human myxoid liposarcoma. Nature 1993;363:640-4.
18. Mantilla JG, Ricciotti RW, Chen EY, Liu YJ, Hoch BL. Amplification of DNA damage-inducible transcript 3 (DDIT3) is associated with myxoid liposarcoma-like morphology and homologous lipoblastic differentiation in dedifferentiated liposarcoma. Mod Pathol 2019;32:585-92.
19. Dermawan JK, Hwang S, Wexler L, Tap WD, Singer S, Vanderbilt CM, *et al*. Myxoid pleomorphic liposarcoma is distinguished from other liposarcomas by widespread loss of heterozygosity and significantly worse overall survival: A genomic and clinicopathologic study. Mod Pathol 2022;35:1644-55.
20. Fadaei S, Cordier F, Ferdinande L, Van Dorpe J, Creytens D. Myxoid pleomorphic liposarcoma. Histol Histopathol 2024;39:1101-8.
21. Murshed KA, Abo Samra H, Ammar A. Well-differentiated liposarcoma of the hypopharynx exhibiting myxoid liposarcoma-like morphology with MDM2 and DDIT3 co-amplification. Head Neck Pathol 2022;16:288-93.

Funding: Nil; Conflicts of interest: Nil.

How to cite this article: Indulkar ST, Rao BV, Gundimeda SD, Kodandapani S, Manilal R, Thammineedi SR. Challenges in the diagnosis of an unusual retroperitoneal pararenal well-differentiated Liposarcoma: A case report unraveling the complexities of genetic alterations and histomorphology. Indian J Case Reports. 2025; 11(1):31-35.