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Наджла Салех Бен Гашир^{1,2}, Бабита Алинггал Мохамед¹, Ареф Чехаль³, Ашраф Алаккад⁴

¹ — Отделение патологии и лабораторной медицины, Медицинский центр Шейха Шахбута, Абу-Даби, ОАЭ

² — Академическая корпорация здравоохранения Дубая, Дубай, ОАЭ

³ — Отделение онкологии и гематоонкологии, Медицинский центр Шейха Шахбута, Абу-Даби, ОАЭ

⁴ — Отделение внутренних болезней, Госпиталь Мадинат Заед, Регион Эд-Дафра, ОАЭ

ЧИСТАЯ АДЕНОКАРЦИНОМА С КИШЕЧНОЙ ДИФФЕРЕНЦИРОВКОЙ ЯИЧКА КАК ПЕРВОЕ ПРОЯВЛЕНИЕ ТЕРАТОМЫ ЯИЧКА: КЛИНИЧЕСКИЙ СЛУЧАЙ И ОБЗОР ЛИТЕРАТУРЫ ПО ТАКТИКЕ ВЕДЕНИЯ

Najla Saleh Ben Ghashir^{1,2}, Babitha Alingal Mohamed¹, Aref Chehal³, Ashraf ALakkad⁴

¹ — Department of Pathology and Laboratory Medicine, Sheikh Shakhbout Medical City, Abu Dhabi, UAE

² — Dubai Academic Health Corporation, Dubai, UAE

³ — Department of Oncology and Hematooncology, Sheikh Shakhbout Medical City, Abu Dhabi, UAE

⁴ — Department of Internal Medicine, Madinat Zayed Hospital, AL Dhafra Region, UAE

Pure Adenocarcinoma with Intestinal Differentiation of The Testis as The First Presentation of a Testicular Teratoma: A Case Report with Literature Review of Management

Резюме

Чистая аденокарцинома — это соматический тип злокачественного новообразования, возникающего из герминогенной опухоли, встречается крайне редко, но такие случаи описаны. Обычно соматическая малигнизация проявляется как саркома, реже — как карцинома. Этот редкий феномен, как правило, объясняется развитием тератоматозного компонента. В большинстве случаев диагноз не вызывает затруднений благодаря смешению различных компонентов герминогенной опухоли и наличию герминогенной неоплазии in situ (GCNIS). Однако в некоторых редких случаях метастатическая карцинома в яичке может оказаться чем-то иным. В данном клиническом случае описывается 35-летний мужчина с опухолью яичка в виде аденокарциномы с кишечными чертами, напоминающей метастатическую колоректальную карциному. В окружающей ткани яичка была обнаружена GCNIS, а флуоресцентная гибридизация in situ на аномалии хромосомы 12p выявила наличие i(12p) в тестикулярной аденокарциноме, что подтверждает общее герминогенное происхождение. После забрюшинной лимфодиссекции были обнаружены метастатические отложения слизистой аденокарциномы. Обширное клиническое обследование помогло исключить метастазирование из другого первичного очага, в частности из желудочно-кишечного тракта. Наше наблюдение указывает на то, что аденокарцинома кишечного типа в препарате после орхиэктомии, хотя чаще и представляет собой метастаз из первичной опухоли желудочно-кишечного тракта, может быть первичной опухолью яичка герминогенного происхождения. Пациент был пролечен радикальной орхиэктомией с забрюшинной метастазэктомией с последующей химиотерапией, направленной на соматический тип злокачественной гистологии, по схеме для колоректальной аденокарциномы. В течение периода наблюдения 3,5 года у пациента сохранялась полная ремиссия.

Ключевые слова: Яичко, тератома, соматическая малигнизация, аденокарцинома, слизистая, кишечный фенотип

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Abstract

Pure adenocarcinoma is a somatic-type malignancy that comes from a germ cell tumor and is extremely rare but has been reported. It is usually seen as sarcoma, and less often as carcinoma. This rare phenomenon is generally attributed to the development of a teratomatous component. In most cases, the diagnosis remains straightforward due to the mixing of different germ cell tumor parts and the existence of germ cell neoplasia in situ (GCNIS). But, there are some rare instances where metastatic carcinoma to the testis could be something more. This case presentation discusses a 35-year-old man who had a testicular tumor of adenocarcinoma with enteric features, which looked like metastatic colorectal carcinoma. GCNIS was found in the background testicular tissue, and fluorescence in situ hybridization for chromosome 12p abnormalities showed the presence of i(12p) in the testicular adenocarcinoma, which supports a shared germ cell origin. After the retroperitoneal lymph node dissection, it was found that there were metastatic deposits made up of mucinous adenocarcinoma. Extensive clinical workup helped exclude metastasis from another primary, particularly the GI tract. Our report indicates that adenocarcinoma of intestinal type in an orchiectomy specimen, although usually strongly suggestive of metastasis from a gastrointestinal tract primary, maybe a primary testicular neoplasm of germ cell tumor origin. The patient was treated with radical orchidectomy with retroperitoneal metastasectomy followed by somatic-type malignant histology-directed chemotherapy for colorectal adenocarcinoma. The patient remained in complete remission for a 3.5-years follow-up period.

Key words: Testis, teratoma, somatic malignancy, adenocarcinoma, mucinous, intestinal phenotype

Conflict of interests

The authors declare no conflict of interests

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Conformity with the principles of ethics

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GCNIS — Germinogenic neoplasia in situ, TGCT — Germinogenic testicular tumor, CT — Computed tomography, SMT — Somatic malignant transformation, GCT — Germinogenic tumor, ADC — Adenocarcinoma, SCC — Squamous cell carcinoma, FOLFOX — Folinic acid, Oxaliplatin, 5-fluorouracil

Introduction

Testicular germ cell tumors are a type of solid neoplasms which account for the greatest incidence (> 90%) of testicular cancer among young adult men(1). TGCTs are histologically divided into 2 categories: non-seminomas and seminomas(2). Non-seminomas include embryonal carcinomas, choriocarcinomas, teratomas and yolk sac tumours. Testicular teratomas have further been subclassified as either prepubertal type or post-pubertal type. Prepubertal-type teratomas are generally not aggressive and do not have a link to germ cell neoplasia in situ (GCNIS), while post-pubertal-type teratomas can be malignant and are linked to GCNIS(3). Additionally, teratomas that occur after

puberty tend to spread to areas outside the gonads, such as the retroperitoneal lymph nodes. It is uncommon for testicular post-pubertal-type teratomas to develop into a somatic-type malignancy. We present our case of a testicular pure adenocarcinoma with background GCNIS. The primary tumor showed enteric differentiation while the retroperitoneal lymph node metastasis showed colloidal mucinous cystadenocarcinoma histomorphology. Although no other teratoma components were identified with the tumor, the presence of GCNIS in the adjacent testicular tubules supported germ cell origin. Molecular characterization of the primary tumor was undertaken supporting germ cell origin. Comprehensive clinical and imaging workup helped

further exclude primary gastrointestinal or pancreaticobiliary tract primaries.

Case Presentation:

A 35-year-old man, married with two children had a history of right orchiopexy for incidentally discovered undescended testis at the age of 7. The patient reported no family history of testicular cancer or other malignancies. Genetic counseling was offered, but no known hereditary cancer syndromes (e.g., Lynch syndrome, BRCA mutations) were identified in preliminary screening. He denied smoking or alcohol intake or recent scrotal trauma. There were no clinical signs of hormonal imbalance (e.g., gynecomastia, changes in libido). Serum testosterone and gonadotropin levels were within normal limits. He had 5 years history of on and off abdominal and lower right back pain accompanied with right testicular swelling, that did not respond to oral analgesics. Testicular ultrasound showed a large solid right testicular mass lesion. He was seen in a private Hospital where computerized tomography (CT) imaging was done in September 2020 and showed multiple bulky retroperitoneal/right para-aortic lymphadenopathy (figure 1) along with a testicular mass with central cavitation. CT scan findings were indicative of nodal metastasis from the clinically detected testicular mass. CT scan (with venous phase) of the chest and upper abdomen was negative for

lung and liver metastasis. The analysis of tumor markers indicated that there were no elevated levels of lactate dehydrogenase, beta human chorionic gonadotropin, alpha-fetoprotein.

In September 2020 the patient underwent a right inguinal radical orchiectomy. In October 2020 he had retroperitoneal lymph node dissection. His personal medical history was notable for the absence of prior significant injuries. There was no family history suggestive of hereditary cancer syndromes, such as Lynch syndrome or BRCA-related cancers, although genetic counseling was pursued for reassurance. He denied any history of gynecomastia, reduced libido, or other signs suggesting hormonal imbalance, with physical examination showing normal secondary sexual characteristics and stable serum testosterone and gonadotropin levels. During the initial evaluation, no symptoms typically indicative of systemic intoxication—such as persistent fever, generalized weakness, or significant appetite loss—were documented. His blood pressure readings during serial clinic visits remained within normal range. Additionally, his two children underwent clinical examination and were found to be healthy with no detectable abnormalities.

Histology of the right testes showed moderately differentiated adenocarcinoma (with intestinal differentiation) on a background of germ cell neoplasia in situ, indicative of a teratoma with somatic malignancy. Immunoperoxidase stains showed that the tumor was positive for the epithelial glycoprotein BER-EP4, CK20, CDX-2 and negative for the germ cell markers SALL-4, OCT4, AFP, CD30, PLAP. Seminiferous tubules showed germ cell neoplasia in situ (GCNIS) that is positive for PLAP and OCT4. (Fig. panel 1A-1H panel). Lymphovascular invasion was present. Tumor excision was locally complete. However, right para-aortic lymphadenectomy was performed showing metastatic deposits of an adenocarcinoma displaying mucinous differentiation, involving all four excised lymph nodes. (Fig. panel 2A-2D). Full medical and imaging follow-up with a body scan did not show evidence of another primary in the gastrointestinal and pancreaticobiliary tracts or elsewhere in the body.

Testicular tumor tissue was submitted for chromosomal microarray studies. Genomic alterations observed include a complex pattern of discontinuous gains (5 to 7 copies) and amplifications involving chromosome 12p (including CCND2 and KRAS) as well as lower level gain of chromosome 12q (3 copies). The abnormalities identified on chromosome 12p are structurally complex and are not typical of a classic isochromosome 12p, these findings are consistent with over-representation of most of the 12p relative to 12q. Such over-representation of chromosome 12p has been reported in association with a subset of testicular germ cell tumors, although these findings are not diagnostic of GCT.



Figure 1. September 2020 (multiple right para-aortic lymph node mass with the largest lymph node measures 5×5,4 cm compressing the IVC)



Figure 2. September 2021 (represent stable findings)

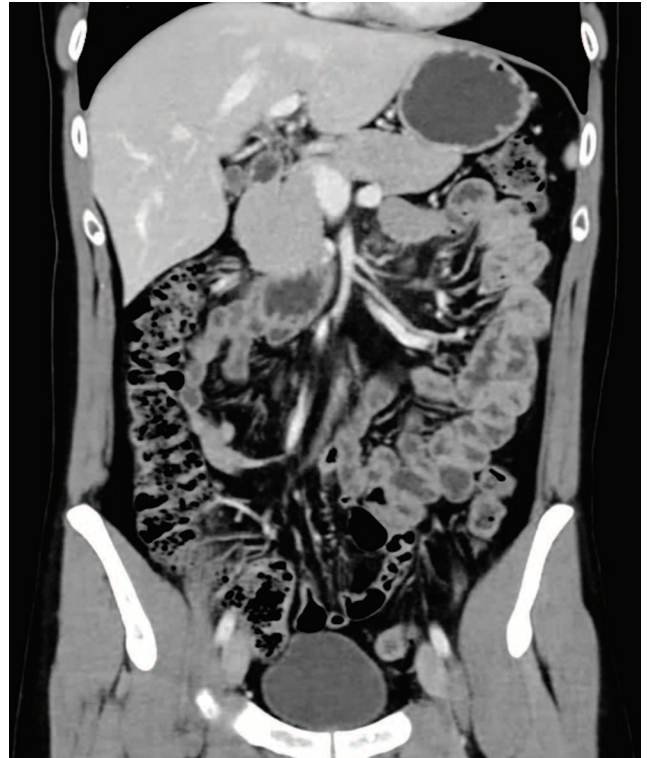


Figure 3. April 2025 (No metastasis in chest, abdomen and pelvis)

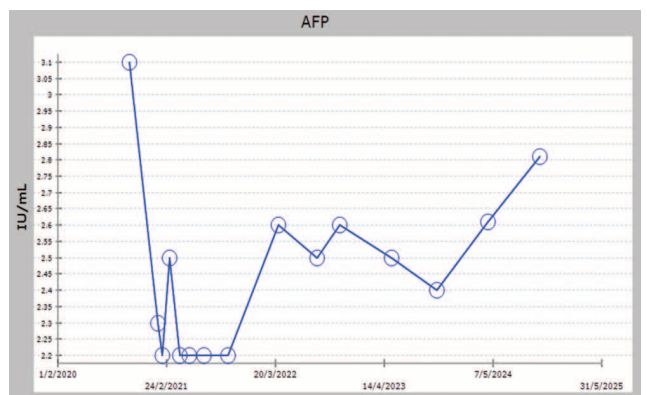
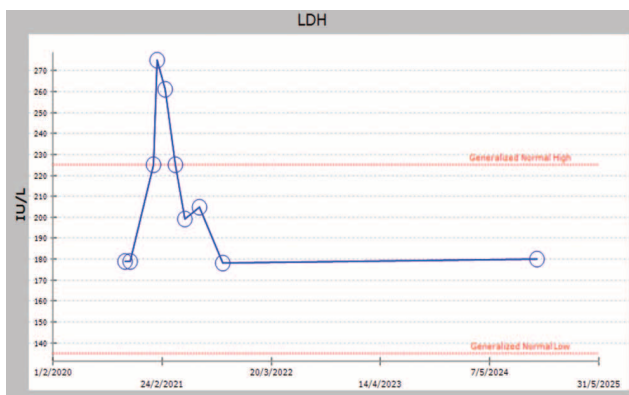


Figure 4. Tumor markers

Because of metastatic disease that was surgically debulked, the patient was subjected to somatic-type malignant histology-directed chemotherapy, in this case colorectal carcinoma regime was applied. Surgical resection was immediately followed by 12 cycles of chemo/adjuvant (FOLFOX) completed over 6 months from October 2020 to March 2021. CT scan in April 2021 and laboratory results were normal. The latest clinic follow-up was in April 2025 when he had no clinical, radiologic (Fig. 2,3), or biochemical (fig. 4) evidence of relapse or recurrence. He was planned to continue 3-month follow-ups and to repeat CT scans in 6 months.

Discussion

Our case presents several unique and clinically significant features that distinguish it from typical testicular germ cell tumors. The 35-year-old patient demonstrated pure adenocarcinoma with intestinal differentiation arising from a germ cell tumor, confirmed by the presence of GCNIS in adjacent testicular tissue and characteristic chromosome 12p abnormalities. The absence of elevated tumor markers (LDH, β -hCG, AFP) at presentation was particularly noteworthy, as this combination is unusual in typical testicular germ cell tumors but can occur in somatic-type malignancies.

Histologically, our case showed moderately differentiated adenocarcinoma with enteric features in the primary tumor, while metastatic deposits in retroperitoneal lymph nodes displayed mucinous adenocarcinoma characteristics. The molecular analysis revealed complex chromosome 12p gains (5-7 copies) involving CCND2 and KRAS genes, along with lower-level chromosome 12q gains, supporting the germ cell origin despite the absence of typical *i(12p)*. Importantly, comprehensive clinical workup successfully excluded primary gastrointestinal or pancreaticobiliary sources, confirming the testicular origin.

The clinical presentation was also distinctive, with a 5-year history of intermittent symptoms, absence of systemic intoxication signs, normal hormonal parameters, and notably, a history of cryptorchidism requiring orchiopexy at age 7. The patient's excellent response to histology-directed FOLFOX chemotherapy, resulting in 42 months of complete remission, contrasts sharply with the generally poor prognosis reported in the literature for carcinomatous somatic-type malignancies.

Teratoma is a common type of TGCT that includes components from two or more germ-cell layers, which are endoderm, mesoderm and ectoderm(4). Teratoma with malignant transformation (SMT) is a rare type of teratoma that has somatic-type malignant elements found in different organs and tissues(5). This entity includes a range of tumors such as carcinomas like squamous cell carcinoma, adenocarcinoma and sarcomas such as rhabdomyosarcoma and malignant nerve sheath tumor, hematopoietic malignancies like leukaemia, and other types like nephroblastoma, carcinoid and primitive neuroectodermal tumors(6).

SMT can show up as either primary or metastatic GCT and can also develop in places outside the gonads, like the intracranial cavity retroperitoneum and mediastinum(4). SMT makes up less than 5% of metastatic testicular tumors and usually impacts younger men(7-9).

Hwang MJ and colleagues examined the clinicopathologic characteristics of 63 GCTs, which included 22 in the testis and 41 with metastases(10). The patients with SMT in the testis had a median age of 26 years, which is younger compared to those with metastatic SMT, who had a median age of 38.5 years. Sarcoma was the most common type of testicular tumors, while carcinoma was the most common type of tumors in metastases, with most carcinomas being adenocarcinomas.

Several theories have been discussed by people in regards to how malignant transformation occurs in a GCT. Malignant transformation may occur either by the differentiation of totipotent germ cell elements into somatic tissues, which then transform into malignancy, or by malignant transformation of already existing

teratomatous components(9). According to Oosterhuis et al., mature teratoma of metastases derives from primary tumors with mature components. These authors claim that the observed differentiation in metastases is due to the selective destruction of non-teratomatous elements by chemotherapy, rather than differentiation of totipotent germ cells(11), which allows for the selective growth of the chemo resistant teratomatous elements.

This process allows for the selective growth of the chemo-resistant teratomatous elements. Additionally, the occurrence of chromosome 12p abnormalities in these tumors, particularly the isochromosome 12 seen in most instances, indicates a shared clonality in GCTs(12, 13). Both adenocarcinoma with enteric differentiation and mucinous adenocarcinomas have occurred as variants of SMT. However, in our case, mucinous differentiation was only manifest in the retroperitoneal lymph node metastasis which may argue for Green's theory(13).

The first pathogenetic event of GCT happens during embryonal development, impacting a gonocyte or primordial germ cell. Even though this starts in the uterus, the tumor won't show up clinically until after puberty, with carcinoma in situ (CIS) being the precursor. All invasive TGCT, including both nonseminomas and seminomas, along with CIS cells, are aneuploid. Invasive TGCT primarily shows consistent structural chromosomal abnormalities, particularly gains on the short arm of chromosome 12, which are mostly caused by the formation of isochromosome (*i(12p)*). This indicates that having more copies of a gene or genes on 12p is linked to the occurrence of a clinically evident TGCT(14, 15).

SMTs are identified histologically by the invasion of nearby germ-cell elements by very atypical somatic cells(16). According to the authors, the key characteristic for diagnosing SMT is the growth of somatic malignant elements. Clinically significant SMT is identified when the somatic-type component occupies a field of view at low magnification, specifically with a 4× lens. Carcinomas like SCC, ADCs, neuroendocrine carcinomas are a rare group among SMT patients (16). Some tumors show staining for carcinoembryonic antigen and cytokeratins but they test negative for GCT markers like human chorionic gonadotropin, alpha-fetoprotein and placental alkaline phosphatase(17).

One of the most challenging aspects of our case was distinguishing primary testicular adenocarcinoma from metastatic colorectal carcinoma to the testis. Our comprehensive approach, including negative gastrointestinal workup, presence of GCNIS, and molecular confirmation of germ cell origin, was crucial for accurate diagnosis.

Metastatic carcinoma to the testes is rare and most commonly incidentally found at autopsy(18). The most common tumor to metastasize to the testes excluding leukemia and lymphomas is kidney (9%), prostate

(35%), melanoma (18%), lung (18) (18) and colorectal less than 8%(19). From 1950 to 2017, 75 cases of colorectal metastasis in testis have been reported(20). The testicular mass is even rarer as the first sign of a primary tumor(21). Our case adds to the limited literature on primary testicular adenocarcinomas that can mimic metastatic colorectal cancer, emphasizing the importance of thorough molecular and histologic evaluation. Ouellette says that there are fewer than 25 documented cases of colorectal cancer that have metastasized to the testis. The rarity of testicular metastases may be explained by low scrotum temperature limits metastases dissemination to the testes through the blood.

Treatment

There isn't a lot of research out there, so we don't have a set standard for SMT care. Instead, we rely on management advice from centers that handle a lot of these cases. For localized SMT disease, the usual treatment is radical orchiectomy. The importance of adjuvant chemotherapy is still a topic of debate. In the past, TGCTs that have somatic-type malignancy haven't really responded well to radiation and the usual platinum-based chemotherapy treatments(22, 23).

Evidence is in favor of aggressive resection as having negative margins is essential for long term remission and better oncological results(24). A study from 1998 involving 46 SMT patients who underwent complete resection showed that they had better oncological outcomes during follow-up in comparison with those people who had positive margins and incomplete resection ($P=0.003$) (24). Patients who have clinical stage I disease should definitely be considered for primary retroperitoneal lymph node dissection (RPLND). Conversely, patients with advanced but resectable disease typically undergo post-chemotherapy RPLND. This procedure requires a collaborative surgical effort and the complete removal of any essential vascular and visceral structures.

Several authors have suggested that histology-specific systemic chemotherapy regimens could be a more effective way to manage SMT. Efforts to direct chemotherapy at the transformed histology in metastatic teratomas have produced varied results, with certain studies indicating lasting positive responses while others report no response at all (13, 25). Atwi and colleagues showed responses in patients who had a specific type of cancer(25).

In a study, seven of the 10 SMT patients with a response to regimens tailored to the histology of the somatic malignancy achieved a partial response and three had a long term response (13, 26). In a similar study in Europe, 8 SMT patients who received chemotherapy directed against the non-GCT component at relapse had a 50% partial response(12).

The effectiveness of chemotherapy directed at TGCT and somatic-type malignant histology in metastatic cases, especially for patients with various histologic subtypes, is still mostly unknown. Patients with SMT can show systemic progressive disease and have normal serum tumor markers even when they are receiving proper treatment with cisplatin-based regimens because of their chemoresistant characteristics. Patients with SMT do not respond well to standard GCT treatments and tend to experience late systemic failure(27). Therefore, the best approach for managing SMT should include removing all areas affected by the disease along with systemic therapy focused on malignant transformation. Even so, dealing with SMT is still tough, and there aren't really good treatment options available for advanced cases(28, 29). Gene expression profiling is a new way to gain insights into molecular mechanisms and find possible targets that could be acted upon in difficult SMT cases. At the first diagnosis of a testicular tumor, our patient had retroperitoneal lymph node metastasis. Since we used FOLFOX every 3 weeks as adjuvant chemotherapy for colorectal cancer, we selected FOLFOX every 3 weeks as adjuvant chemotherapy. After surgery, the patient has been relapse or recurrence free for 42 months.

Prognosis

The outcomes of carcinomatous SMT depend on the stage and whether the disease can be surgically removed (30). When looking at different SMT histologies, carcinomas tend to have a delayed relapse, often occurring 5 years or more after the initial GCT diagnosis. They also rarely show a response to fluorouracil-based chemotherapy treatments or radiation(24). There isn't much information about the results of SMT, and what we do have mostly comes from small case series, primarily from large cancer centers(24, 30-32). Patients who are in stage I of the disease usually have a favorable prognosis, while those with metastatic disease experience worse outcomes in terms of cancer treatment, even when they undergo aggressive surgery and receive standard cisplatin-based systemic therapy for germ cell tumors, which has historically resulted in cancer-specific survival rates of about 50%(33).The largest single-institution SMT series included patients with carcinoma, nephroblastoma, and sarcoma, sarcomatoid yolk sac tumor(10).

Approximately 75 percent of the patients had stage II-III disease, and the total estimated 5-year cancer-specific survival rate was 64%, with a median follow-up of 71 months. No differences were found in outcomes between patients with sarcoma and those with carcinoma, although the patterns of recurrence varied. Patients with carcinomas tended to relapse several years later than those with sarcomas after the initial GCT diagnosis. A study conducted by Hwang MJ and colleagues

involving 63 patients found that those with metastatic SMTs had a suggestively lower overall survival rate compared to patients with SMTs in the testis, with a five-year survival rate of 35 % versus 87 % ($P=0.011$)(10). Additionally, patients with carcinomatous SMTs showed a significantly poorer prognosis compared to those with sarcomatous or PNET SMs, with 5-year survival rates of seventeen percent, 77 percent, and 73 percent, respectively ($P=0.002$), when analyzing the entire cohort, which included testicular and metastatic SMTs. The histologic subtype of SMT really impacts the clinical outcome, and it turns out that the carcinomatous SMT has an elevated risk for mortality.

Conclusion

In summary, this case highlights the rare occurrence of pure testicular adenocarcinoma with intestinal differentiation as a primary germ cell tumor, emphasizing the critical role of histopathological and molecular characterization in confirming its origin. The patient's detailed clinical history, imaging workup, and tailored treatment approach—involving radical orchiectomy, retroperitoneal metastasectomy, and histology-specific chemotherapy—led to a durable remission over a follow-up period exceeding 3.5 years. This case underscores the necessity of individualized treatment strategies and long-term surveillance in managing somatic-type malignancies arising from germ cell tumors.

SMT is a rare kind of GCT. Managing SMT patients effectively requires a team-based approach that includes proactive surgery and systemic therapy tailored to the specific histology. Surgery can really help patients with early-stage malignant TSCST, but for those with a lot of metastatic disease, the results aren't great since these tumors don't respond well to chemoradiation. More future studies are really important to help clarify how these rare malignant tumors develop. This will help us find targets we can act on, new ways to predict outcomes, and new treatment methods.

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Ашраф Алаккад: клиническое ведение пациента, сбор данных, утверждение окончательного варианта рукописи

Наджла С. Бен Гашир: концепция и дизайн исследования, сбор и интерпретация данных, подготовка рукописи

Бабита А. Мохамед: гистопатологический анализ, интерпретация данных, редактирование рукописи

Ареф Чехаль: клиническое ведение пациента, интерпретация данных, критический пересмотр рукописи

Author Contribution:

All the authors contributed significantly to the study and the article, read and approved the final version of the article before publication

Ashraf Alakkad: made a major contribution to the development of the concept of the article with writing and editing the case report

Najla Saleh Ben Ghashir: contributed to the development of concept and writing up of the manuscript, the collection and interpretation of the clinical data, and critically reviewed the final version of publication

Babitha Alingal Mohamed: contributed to the interpretation of clinical data and critically reviewed the manuscript

Aref Chehal: contributed to the interpretation of clinical data and critically reviewed the manuscript

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Информация об авторах

Наджла Салех Бен Гашир — FRCPath (Великобритания), MBCh. Врач-консультант, гистопатолог, отделение патологии / отделение патологии и генетики, Госпиталь Дубая, Дейра. ORCID ID: 0000-0001-9345-4148, E-mail: Najla_dr2005@yahoo.com

Ашраф АЛаккад — MD, врач-интернист, заведующий программой рационального использования противомикробных препаратов, отделение внутренних болезней, Госпиталь Мадинаат Заед. ORCID ID: 0000-0002-4083-2800, E-mail: ashraf.alaqqad@gmail.com

Ареф Чехаль — MD, консультант, отделение онкологии и гематологии, Медицинский центр Шейха Шахбута; адъюнкт-профессор медицины и онкологии, Медицинский университет залива. ORCID ID: 0009-0000-3753-2076

Д-р Бабита Алинал Мохамед — M.D., D.N.B., M.N.A.M.S., F.R.C.Path. (Великобритания) — специалист в области патологической анатомии и клинической патологии в Медицинском центре Шейха Шахбута (SSMC) в Абу-Даби. ORCID ID: 0009-0003-3234-0384

About the authors

Najla Saleh Ben Ghashir — FRCPath(UK), MBCh, Consultant Histopathologist, Pathology Section or Pathology and Genetics Department, Dubai Hospital, Deira, ORCID ID 0000-0001-9345-4148, Najla_dr2005@yahoo.com

Ashraf ALakkad — MD, Internist, Department of Internal Medicine, Chair of Antimicrobial Stewardship Program Madinat Zayed Hospital. ORCID ID: 0000-0002-4083-2800, Scopus ID: 60052817400, Web Of Science Researcher ID: AEW-9201-2022, E-mail: ashraf.alaqqad@gmail.com

Aref Chehal — MD, Consultant, Oncology and Hematology Department, Sheikh Shakhbout Medical City; Adjunct Professor of Medicine and Oncology, Gulf Medical University, ORCID: 0009-0000-3753-2076

Dr. Babitha Alingal Mohammed — M.D., D.N.B., M.N.A.M.S., F.R.C.Path. (UK), is an anatomic and clinical pathology specialist at Sheikh Shakhbout Medical City (SSMC) in Abu Dhabi, ORCID: 0009-0003-3234-0384

✉ Автор, ответственный за переписку / Corresponding author