International Journal of Research in Medical Sciences
Kumar SV et al. Int J Res Med Sci. 2015 Jan;3(1):327-330
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: 10.5455/2320-6012.ijrms20150163

Case Report

Mixed germ cell tumour of testis: a case report with review of literature

S. V. Kumar[1], M. Bhagavan Naik[1], M. S. R. Harshvardan[1], A. Bhagya Lakshmi[2]*

[1]Department of Surgery, Andhra Medical College/King George Hospital, Visakhapatnam, A.P., India
[2]Department of Pathology, Andhra Medical College/King George Hospital, Visakhapatnam, A.P., India

Received: 7 November 2014
Accepted: 8 December 2014

***Correspondence:**
Dr. A. Bhagya Lakshmi,
E-mail: dr.a.bhagyalaxmi@gmail.com

ABSTRACT

Testicular tumours are rare neoplasm. Mixed germ cell tumour is the most common histological variant. Essentially, any admixture of the germ cell tumours as seen in pure form may be seen, one of the most common admixtures being embryonal carcinoma and teratoma. Unfortunately many of these patients present late usually with some complications. We present a rare case of mixed germ cell tumour with predominant embryonal carcinoma and yolk sac tumour in adolescent patient with multiple metastatic foci at the time of presentation.

Keywords: Mixed germ cell tumour, Embryonal carcinoma, Virchow's node

INTRODUCTION

Testicular tumours are respectively rare neoplasm; they make up for approximately 2% of all malignant tumours in men and account for up to 10% of all malignant diseases occurring within the male genitourinary system. Approximately 95% of all testicular neoplasms are of germ cell origin. WHO classification divides germ cell tumours into two categories. About 40% of the tumours are composed entirely of a single histologic pattern such as seminoma, spermatocytic seminoma, embryonal carcinoma, yolk sac tumour, choriocarcinoma, and teratoma (mature and immature). The remaining 60% contain a mixture of two or more histologic patterns, known as mixed germ cell tumour.[1]

CASE REPORT

A 18 year old male patient came with a complaint of pain abdomen since 3 months & swelling in the left side of scrotum since 1½ months. Swelling gradually increased in size, attained the present size and associated with heaviness in the scrotum. There is history of loss of weight, loss of appetite & back pain. On examination left testis is enlarged measuring 8 x 5 cm, hard in consistency, non-tender with nodular surface. Right testis is normal. On examining the abdomen a 6 x 4 cm firm non-tender lump is noted with nodular surface occupying left hypochondriac & lumbar regions. In the left supraclavicular fossa 3 x 2 cm hard non-tender matted lymph nodes are present (Figure 1). Other blood investigations are with in normal limits. Patient is non-reactive to HIV.

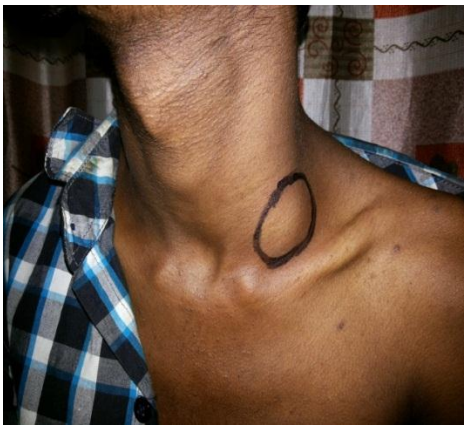

Figure 1: Clinical photo showing left supraclavicular lymphadenopathy.

Ultra sonography of scrotum revealed enlarged left testis with mixed echogenic lesion of 72 x 62 x 55 mm with macro calcific foci & cystic components (Figure 2). USG abdomen showed extensive retroperitoneal lymphadenopathy (Figure 3), large left Para aortic lymph nodal mass with left hydroureteronephrosis. Computed Tomography (CT) scan of the abdomen showed extensive retroperitoneal lymphadenopathy, left hydroureteronephrosis, multiple liver metastasis (Figure 4 & 5) and heterogeneous lesion in the left testis. CT scan of the chest showed bilateral multiple nodular lesions suggestive of metastasis & enlarged left supraclavicular lymph nodes.

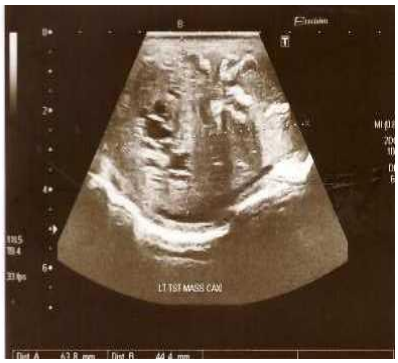

Figure 2: USG scrotum showing mixed echogenic left testicular mass.

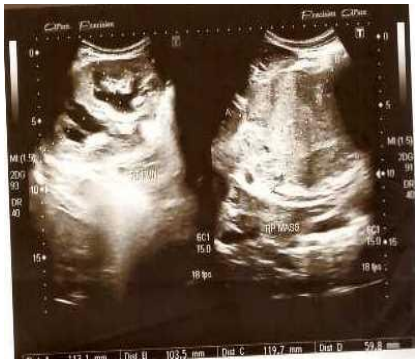

Figure 3: USG abdomen showing retroperitoneallymph nodal mass.

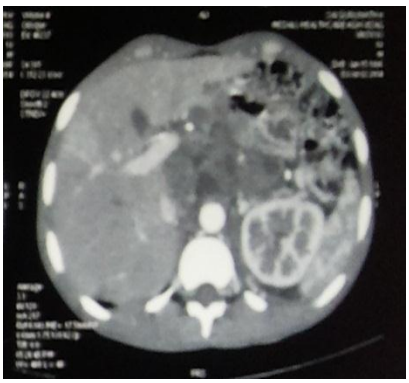

Figure 4: CT scan showing retroperitoneal lymph nodes, liver metastases, left hydronephrosis.

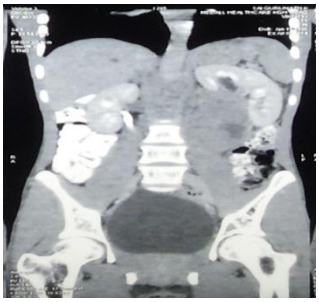

Figure 5: CT scan coronal view showing left retroperitoneal nodes displacing left kidney laterally and left hydroureteronephrosis.

Serum human chorionic gonadotropin (HCG) (2902 mIU/ml) and lactate dehydrogenase (LDH) (3200 U/lit) are elevated. Fine Needle Aspiration (FNA) of the left supraclavicular lymph node was done and smears showed pleomorphic tumour cells arranged in acinar and papillary patterns was suggestive of metastatic deposits of testicular tumour. A provisional diagnosis of testicular tumour with retroperitoneal and left supraclavicular lymphadenopathy with metastasis to liver & lung was considered.

The patient was posted for surgery and left high inguinal orchidectomy was performed (Figure 6). Post-operative period was uneventful. Gross specimen measuring 10 x 6 x 5 cm. Cut section is greyish white. Histopathological examination shows structure of the testis with foci of intratubular neoplasia (Figure 7) and tumour composed of highly pleomorphic tumour cells arranged in diffuse sheets, papillary pattern and in glandular pattern with foci of 3-4 mitotic figures/HPF (Figure 8, 9, 10). Individual tumour cells have moderate eosinophilic cytoplasm, markedly pleomorphic vesicular nuclei with prominent nucleoli. There are foci of yolk sac component with reticular patter of arrangement of tumour cells, hyaline globules and Schiller-Duval bodies (Figure 11). There are occasional areas of semenomatous component, areas of necrosis with calcifications (Figure 12) and tumour emboli. Histological features are consistant with mixed germ cell tumour with predominanent embrional carcinoma, yolk sac tumour with foci of seminoma. The patient was followed up to 2 months and is on chemoradiotherapy.

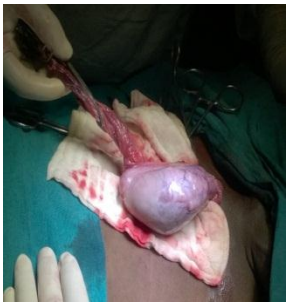

Figure 6: Showing intra operative picture of high inguinal orchiectomy (testis with spermatic cord).

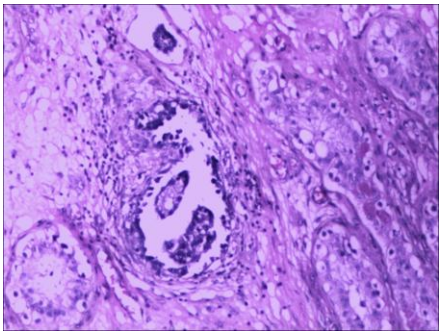

Figure 7: Microphotograph showing intratubular germ cell neoplasia with foci of tumour infiltration.

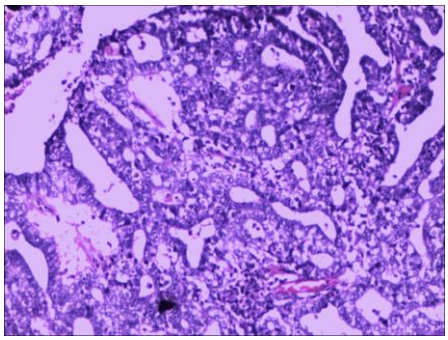

Figure 8: Microphotograph showing sheets of embryonal carcinoma consisting of tumour cells with ill-defined cell borders, large irregular nuclei and prominent nucleoli.

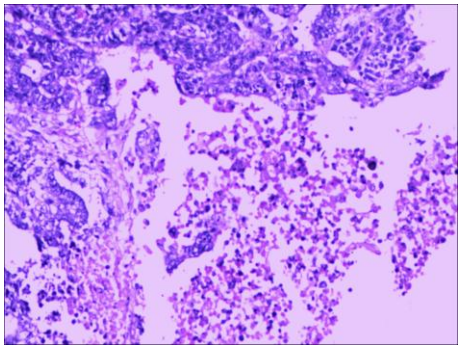

Figure 9: Microphotograph showing pleomorphic tumour cells with areas of necrosis.

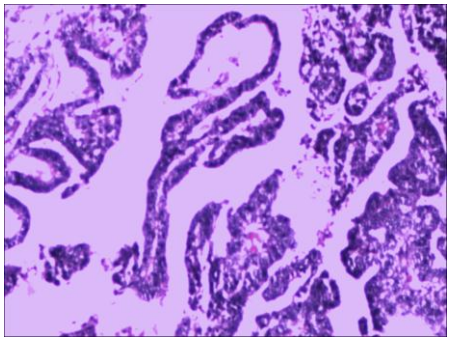

Figure 10: Microphotograph showing glandular arrangement of tumour cells.

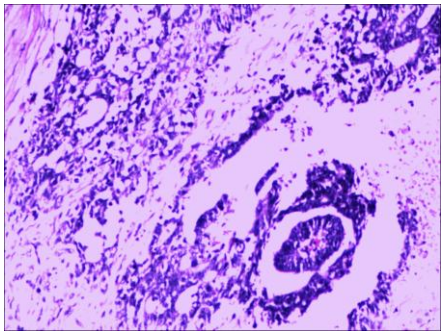

Figure 11: Microphotograph showing Schiller-Duval body.

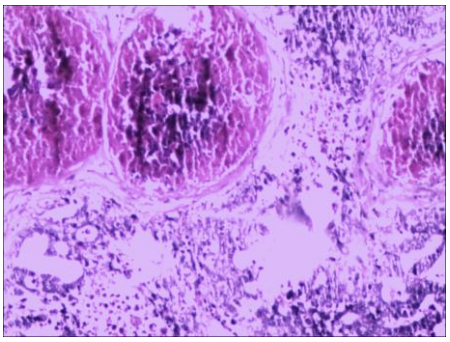

Figure 12: Microphotograph showing areas of necrosis with calcifications.

DISCUSSION

Approximately 95% of all testicular neoplasms are of germ cell origin. For unclear reasons, there has been a worldwide increase in the incidence of these tumours.[1] More importantly, Testicular tumours are the most common malignant diseases, developing in men between 20 and 40 years of age and are the third leading cause of death among men of this age group.[2] The most well documented risk factor for the development of germ cell tumours is cryptorchidism. Testicular dysgenesis, a history of contralateral tumour, and genetic factors also play a role. Recently HIV infection is thought to be a risk factor.[3] The most consistent chromosomal abnormality is an isochromosome of the short arm of chromosome 12, I (12p), which is present in 56% of seminomas and in83% of nonseminomatous tumours.[4] Approximately 1.35% of patients also have a first degree relative with germ cell tumour of the testis. The relative risk for first degree relatives is 3-10 fold.[5] Patients with germ cell tumour in one testicle have a 500-1000 fold greater chance of developing a contralateral testicular carcinoma.

The average age of presentation for patients with mixed germ cell tumours is 30 years. The most frequent combination is that of embryonal carcinoma, seminoma, yolk sac tumour, teratoma, and choriocarcinoma. Despite the presence of seminoma these tumours are managed as Non Seminomatous Germ Cell Tumors (NSGCTs). Unfortunately many of these patients present late usually

with some complications which are difficult to treat and carry bad prognosis. Still if they complete the chemotherapy they have a reasonable survival period depending on the associated complications.

Germ cell tumours except spermatocytic seminoma[6] spreads lymphogenously to retroperitoneal and mediastinal nodes and to the left supraclavicular node (Virchow node). Haematogenous metastases usually go to lungs, liver, brain, or bones. The likelihood of a patient presenting with metastatic disease is approximately 20% for seminomas and higher 30 to 60% for nonseminomatous tumours.[3] The metastasis usually reflects the histology of the basic primary tumour. However, different histologic cell types are found more often in metastases than in primary tumours. This may be due to maturation of one germ-cell type into another cell type. In general, 90 to 100% of patients with localized tumours can be cured and up to 70% with metastatic disease can be cured. The therapy and prognosis of testicular tumours can depend largely on clinical stage and on histological type. Another important factor significantly contributing to the definite cure is completion of the treatment and regular follow-up of the patient. β-human chorionic gonadotropin and/or alpha-fetoprotein serum levels are elevated in about 75% of patients with NSGCT, the levels drop with treatment, and usually, rise with recurrence but not always.[7] A 2 year disease-free interval indicates cure in about 90% of patients.[8]

Seminomas are monomorphic and necrosis is uncommon. They tend to remain localized to the testis for a longer period of time and thus present at an earlier clinical stage. They are extremely radiosensitive and have cure rates greater than 95%. Nonseminomatous germ cell tumours, including mature and immature teratomas, have a variegated appearance and often display haemorrhage and necrosis. These tumours are biologically more aggressive and radio resistant. These tumours still carry a relatively high rate of remission with aggressive chemotherapy, including 60-75% remission with clinical stage III disease.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Jonathan I. Epstein. The lower urinary tract and male genital system. In: Vinay Kumar, eds. Robbins and Cotran Pathologic Basis of Disease. 8th ed. Philadelphia: Saunders Elsevier; 2010: 987.
2. Coleman MP, Esteve J, Damiecki P, Arslan A, Renard H. Trends in cancer incidence and mortality. IARC Sci Publ. 1993;(121):1-806.
3. Courtney M. Townsend Jr, R. Daniel Beauchamp, B. Mark Evers, Kenneth L. Mattox. Urologic surgery. In: Michael Coburn, eds. Sabiston Text Book of Surgery. 19th ed. Philadelphia: Saunders; 2012: 2076.
4. Ulbright, Thomas M. Germ cell neoplasms of the testis. Am J Surg Pathol. 1993;17:1075-91.
5. Brodsky, Gilbert L. Pathology of testicular germ cell tumors. Hemtol/Oncol Clin N Am. 1991;5:1095-125.
6. Sesterhenn IA, Davis CJ Jr. Pathology of germ cell tumours of thetestis. Cancer Control. 2004;11(6):374-87.
7. Ulbright T, Roth L. A pathologic analysis of lesions following modern chemotherapy for metastatic germ cell tumors. Pathol Annu. 1990;25:313-40.
8. Rosai J. Male reproductive system. In: Rosai J, eds. Rosai and Ackerman's Surgical Pathology. 8th ed. Chicago: Mosby Co.; 1996: 1265-1295.

DOI: 10.5455/2320-6012.ijrms20150163
Cite this article as: Kumar SV, Bhagavan Naik M, Harshvardan MSR, Bhagya Lakshmi A. Mixed germ cell tumour of testis: a case report with review of literature. Int J Res Med Sci 2015;3:327-30.