BILATERAL SYNCHRONOUS TESTICULAR GERM CELL TUMORS

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Case Report

A 23-year-old male presented with history of feeling of heaviness in the lower abdomen, indigestion and constipation since 3 months. Pain and discomfort appeared two weeks ago. He denied any history of injury or previous surgery to the testicles. On examination, he was found to have unilateral, firm and nontender masses in right testis. Left scrotal sac was underdeveloped and empty. Ultrasound done showed right testis was enlarged in size and measured 9.2 cms , The entire parenchyma was replaced by multiple hypoechoic mass lesions, Color Doppler showed increased flow. Left testis was undescended and seen in left side of pelvis , It was also enlarged in size and measured 9.0 cms. The entire testicular parenchyma was replaced by multiple hypoechoic mass lesions. Color Doppler showed increased flow. The diagnosis was germ cell tumor of both testes. Beta HCG and Alpha fetoprotein were recommended. There was no evidence of para aortic lymphadenopathy. He had a number of investigations which were reported as follows:

Full blood count – Normal

ESR – 59 mm/ 1st Hour Westergren (Normal range is 0-9 mm)

Serum urea and electrolytes – Normal

Random Blood Glucose - Normal

Liver Function test – Normal except for Alkaline phosphatase which was 648 u/l

(Normal is 100- 290 u/l)

Coagulation screen – Normal

Hepatitis Profile – Normal

Serum ß-HCG - 61.35 mIU/L (normal range upto 2.6 mIU/L),

Serum LDH- 1007 U/L (normal range - 125 -243 U/L)

Alpha-feto Protein (AFP) - Normal.

He underwent bilateral radical orchidectomy and had an unremarkable post-operative recovery. Histological examination of the specimens from both testis showed that the neoplastic cells have abundant amount of eosinophilic cytoplasm. The nuclei were pleomorphic, vesicular and contained prominent nucleoli. Special stains PAS$^{+/-}$D for glycogen was positive in tumour cells. Immunohistochemical stain PLAP was positive in the neoplastic cells. Features were consistent with Bilateral Seminoma. No non-seminomatous germ cell components were seen. The capsules were intact on both sides. The epididymal tissues were tumour free and spermatic resection margin was tumour free. Post orchiectomy CT scan of chest and abdomen was normal. There was no evidence of metastasis.

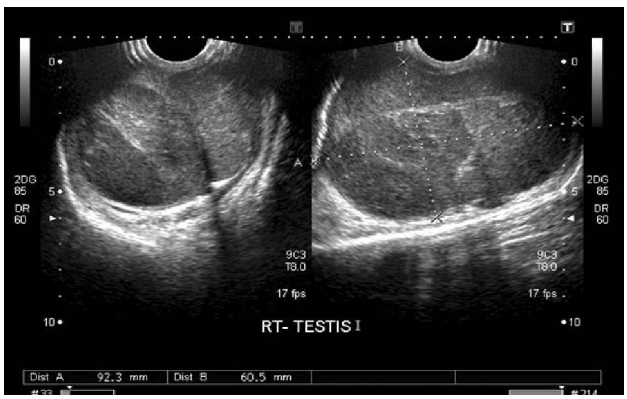

Figure 1: Ultrasound showing enlarged right testis, the entire parenchyma replaced by multiple hypoechoic mass lesions.

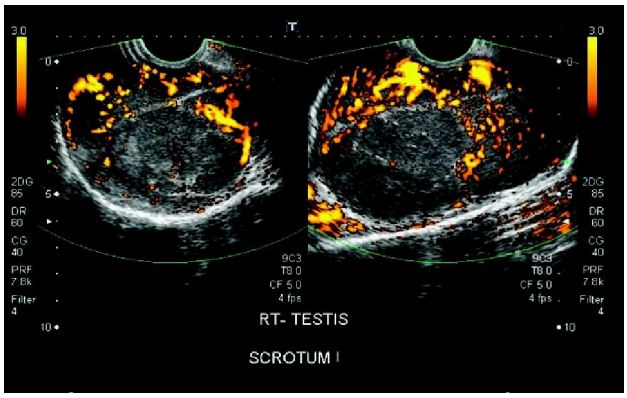

Figure 2: Color Doppler showing increased flow in right testis.

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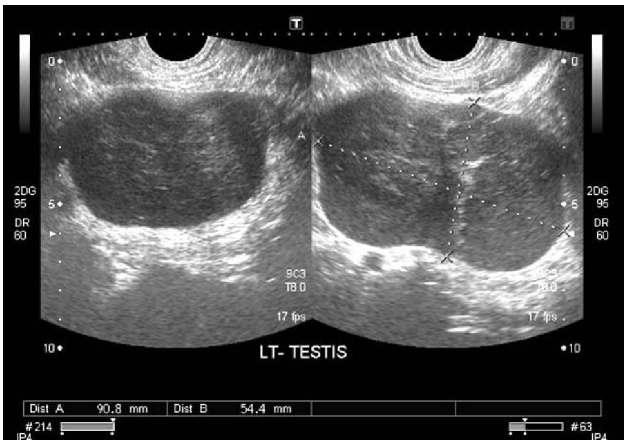

Figure 3: Ultrasound showing left undescended testis, seen in left side of pelvis, it is enlarged in size, the entire parenchymareplaced by multiple hypoechoic mass lesions

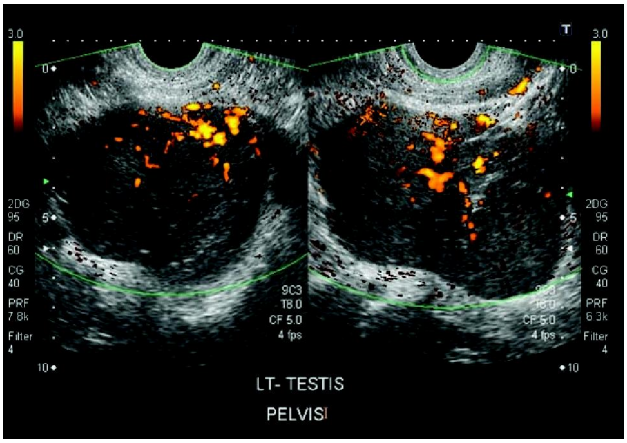

Figure 4: Color Doppler showing increased flow in left testis.

Discussion

Testicular tumors constitute 1% of all malignancies. Bilateral testicular tumors are rare and constitute only 2-3%.[1] Among bilateral testicular tumors, only 5 to 24% occur synchronously and the remaining 7-83% are metachronous.[2] Synchronous bilateral primary germ cell tumors of the testis are exceedingly rare.[5] Most synchronous testicular tumours are seminoma. Other tumours reported include embryonal carcinoma, teratocarcinoma and choriocarcinoma.[3]

Seminoma is the most common pure germ cell tumor of the testis and accounts for 35-50% of all germ cell tumors. Patients typically present with a self detected testicular mass that is sometimes associated with an ill-defined aching sensation in the lower abdomen, inguinal region, or scrotum.[7] Epide- miological risk factors for the development of testicular tumors are: a history of chryptorchidsm, Klinefelter's syndrome, familial history of testicular tumor among first grade relative (father / brothers), the presence of a contralateral tumor and infertility.[4]

Classification

The recommended pathological classification (modified from the World Health Organization) is shown below.

1. Germ cell tumours

- Intratubular germ cell neoplasia
- Seminoma
- Spermatocytic seminoma (mention if there is sarcomatous component)
- Embryonal carcinoma
- Yolk sac tumour:
- Reticular, solid and polyvesicular patterns
- Parietal, intestinal, hepatoid and mesenchymal differentiation
- Choriocarcinoma
- Teratoma (mature, immature, with malignant component)
- Tumours with more than one histological type.

2. Sex cord stromal tumours

- Leydig cell tumour
- Sertoli cell tumour (typical, sclerozing, large cell calcifying)
- Granulosa (adult and juvenile)
- Mixed
- Unclassified

3. Mixed germ cell/sex cord stromal tumours[4]

Germ-cell tumor can occur in testis, retroperitoneum, mediastinum and pineal gland.[5]

Metastatic Sites

Common metastasis seen in testicular malignancy is to retroperitoneal and mediastinal lymph nodes. Choriocarcinoma metastasizes hematogenously.[1]

Diagnosis

LDH, ∝-fetoprotein and ß-HCG are the useful tumor markers.[1] An estimated 5–25% of men with testicular seminomas have elevated levels of ß-HCG, which is produced by syncytiotrophoblastic giant cells.[9] Serum AFP does not show an increase in seminomas,

and increased HCG is found in only 6–10% of pure seminomas. Increased LDH values are noted in 8% of patients with stage I seminoma, compared with approximately 80% of advanced seminomas.[6]
Currently, diagnostic ultrasound serves to confirm the presence of a testicular mass and to explore the contralateral testis. Its sensitivity in detecting a testicular tumour is almost 100%, and it has an important role in determining whether a mass is intra- or extra-testicular.[4] In seminoma US typically demons-trates a rounded, well-circumscribed, hypoechoic, and homogeneous mass that does not contain significant cystic or calcific foci. The tumor is less aggressive than other testicular neoplasms and is therefore usually confined by the tunica albuginea.[8] Magnetic Reso-nance Imaging (MRI) offers higher sensitivity and specificity than ultrasound for diagnosing tumours and may be able to differentiate seminomatous from non-seminomatous tumours.[4] MR imaging provides higher resolution and sensitivity for differentiation of testicular tissue, capabilities that facilitate improved depiction of small tumors that may not be detectable with US. Seminoma typically has a nodular appearance and homogeneous low signal intensity on T2-weighted images.[7]

Treatment and Management

Because there are no lymphatic or vascular connections between the testes, it is thought that synchronous tumors develop independently as two separate primary tumors.[10]
Radical orchidectomy is the method of choice for testicular cancer. It removes the primary tumors and provides the histological diagnosis. AFP, ß-HCG and LDH are measured serially post orchidectomy. If they were raised pre operatively and the levels fall quickly post operatively then it is likely that all the cancer has been removed by the orchidectomy.[6]

Conclusion

Few cases of bilateral synchronous germ cell tumours have been reported. Seminoma is the most common type. Treatment anad management depend on the staging of tumour.

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