

Non-Seminomatous Germ Cell Tumour *in situ* of Testis with Extensive Metastases

Sir,

Germ cell neoplasia *in situ* (GCNIS) is the precursor lesion of most germ cell tumours (GCTs). The precise pathogenesis of GCNIS is vaguely understood. About 50% of the patients known to have GCNIS will develop testicular cancer at some point during the next 5 years.¹ Occult / microscopic / regressed germ cell tumours / fibrous scars as the only evidence of a GCT, producing widespread metastases, are rare. Herein, we present a case of an adolescent boy with non-seminomatous GCNIS of the left testis with extensive metastases.

A 16-year male presented with right hypochondrial pain for 3 months. There was a significant loss of weight and appetite. On examination, anaemia and massive hepatomegaly were detected. The physical examination of the testes was normal. Chest examination revealed normal vesicular breathing. Investigations revealed low haemoglobin and normal blood biochemistry. CT chest and abdomen revealed multiple enhancing soft tissue nodules scattered in both lung fields predominantly in subpleural regions, the largest noted along the posterior basal segment of the right lower lobe which measured approximately 4.5 x 3.3 cm. Another enhancing lesion was identified along the medial aspect of the right middle lobe closely abutting the pericardium. This lesion measured approximately 6.8 x 4.4 cm (TS x AP). There was another large pleural-based peripherally enhancing lesion identified involving the apical segment of the right upper lobe. This lesion measured approximately 5.0 x 5.4 cm in TS x AP dimensions.

A large peripherally enhancing lesion was noted in segment VIII of the right lobe of the liver measuring approximately 15.3 x 9.7 cm in TS x AP dimensions with heterogenous central low attenuation areas likely representing central areas of necrosis. Another lesion with heterogenous enhancement measuring approximately 10 x 12 mm was identified in segment VII of the liver. There were multiple retroperitoneal nodal deposits, the larger deposit was seen in the left para-aortic region at the level of the bifurcation of the aorta. It measured approximately 61 x 54 mm. It showed central low attenuation, suggestive of the necrotic component. Another large nodal deposit in the left para-aortic region measured approximately 32 x 28 mm was noted, along with multiple large necrotic pulmonary nodules (Figure 1A-C). MRI brain was consistent with right temporoparietal mass with vasogenic oedema (Figure 1D). Ultrasound of the testes revealed a well-defined echogenic mass with a cluster of calcification (1.02 x 0.7 cm) at upper pole of the left testis consistent with intra-testicular neoplastic lesion. Tumour markers showed beta Human Chorionic Gonadotropin (β -HCG) of 225,000 IU/ml, Alpha Fetoprotein (AFP) of 1,421 ng/ml, and Lactate Dehydrogenase (LDH) of 2,799 U/l.

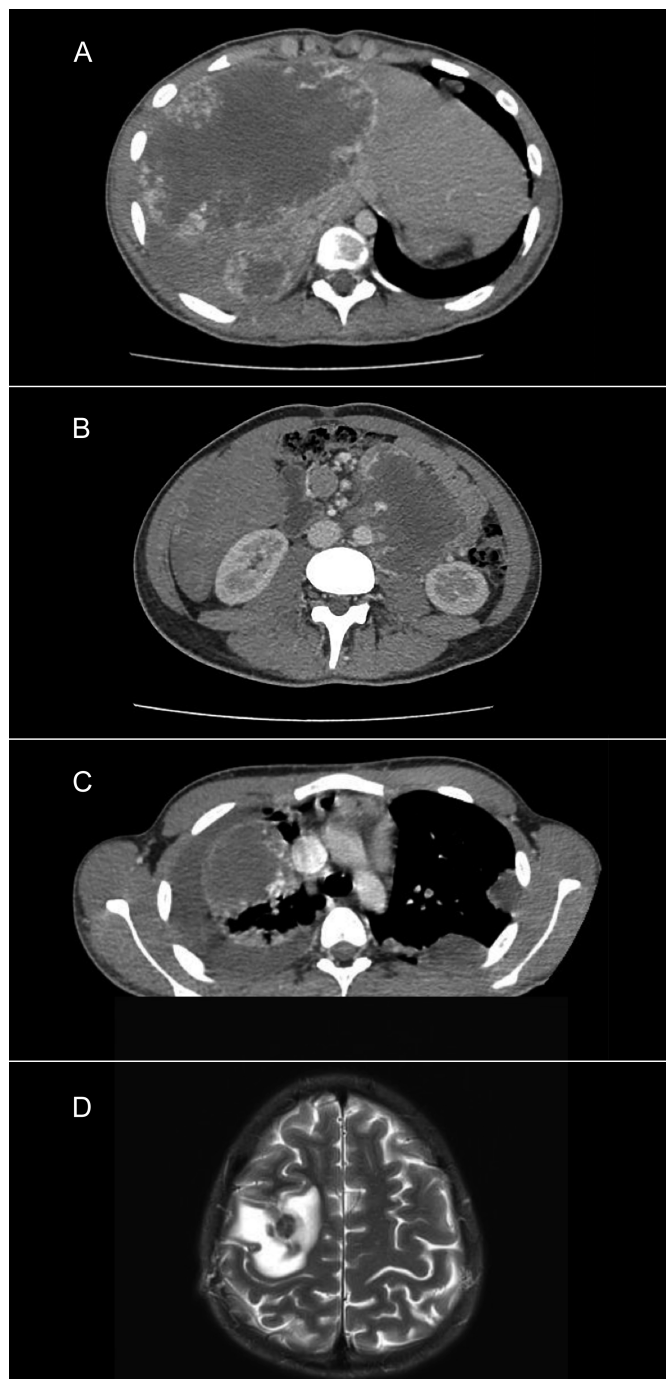


Figure 1: (A) A large peripherally enhancing lesion is noted in segment VIII of the right lobe of the liver. It measures approximately 15.3 x 9.7 cm in TSxAP dimensions and shows peripheral enhancement; (B) A large confluent necrotic nodal mass is noted in the left para-aortic region measuring approximately 8.0 x 7.2 cms in APxTS dimensions showing peripheral enhancement; (C) Multiple enhancing soft tissue nodules scattered in both lung fields and a large pleural-based peripherally enhancing lesion is identified involving the apical segment of the right upper lobe. This lesion measures approximately 5.0 x 5.4 cm in TSxAP dimensions; (D) A multiloculated abnormal signal intensity lesion identified within the cortical and subcortical location of the right frontoparietal region. The lesion measures approximately 2.1 x 2.2 x 1.7 cm in CC x AP x TS dimensions with significant perilesional vasogenic oedema.

He underwent a left orchiectomy. The histopathology was consistent with testicular parenchyma showing areas of fibrosis with extensive GCNIS, atrophic tubules, dystrophic calcification, and Leydig cell hyperplasia. Immunohistochemistry showed OCT 3-4, PLAP, and CKAE1/AE3 positivity highlighting extensive GCNIS. The epididymis and spermatic cord were histologically unremarkable. He was treated with standard BEP (Bleomycin, Etoposide, and Cisplatin) chemotherapy protocol.

So far, he received three cycles with a decline in the serum tumour markers. He is planned to complete a total of 4 cycles followed by whole brain radiation therapy.

Less than 5% of testicular GCTs may undergo spontaneous complete or partial regression. Spontaneously regressed GCTs often first present as metastases, usually with elevated GCT serum markers. Histologic findings pathognomonic of a regressed GCT include fibrotic scar with atrophic tubules, GCNIS, coarse, shard-like calcifications within expanded tubular profiles, Leydig cell clusters exceeding a normal tubular diameter and intratubular microliths/psammomatous-type calcifications.² In this biopsy, the entire testis was submitted, and no viable GCT was present on multiple serial and step sections were examined. However, histologic features including fibrous scar with extensive GCNIS and coarse dystrophic calcification favoured completely regressed GCT. Cases of widespread metastases with occult testicular tumours or only fibrous scars in the testis as the only evidence of a GCT are rarely documented.³ Testicular GCTs, especially choriocarcinoma and seminomas can regress leaving homogeneous scars. Such scars can be associated with testicular atrophy, shrunken seminiferous tubules with decreased or absent spermatogenesis, GCNIS or residual viable tumour such as teratoma.⁴

In conclusion, GCNIS with extensive metastases is a rare aggressive presentation of GCTs and needs to be treated timely with conventional GCT chemotherapy protocols.

COMPETING INTEREST:

The author declared no competing interest.

AUTHOR'S CONTRIBUTION:

AHO: Conceived the idea, performed literature search and drafted the manuscript.

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