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Persisting Elevation of Alpha-fetoprotein After Chemotherapy for Germ Cell Tumour—Not Always Due to Viable Malignancy?

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SURGICAL RESECTION of a residual radiological abnormality in patients who become marker negative after chemotherapy has become standard therapy in patients with advanced germ cell tumours (GCT). It is commonly held that persisting elevated tumour markers after chemotherapy indicate unresectable, viable GCT [1, 2]. We describe a case in whom elevation of alpha-fetoprotein (AFP) after chemotherapy occurred in the absence of malignant germ cell elements on two occasions. Surgical resections of disease resulted in normalisation of serum AFP.

A 30-year-old man presented with bulky cystic retroperitoneal lymphadenopathy due to a non-seminomatous mixed germ cell tumour. Serum tumour markers were elevated: AFP 162 ng/ml (normal 0–15), β-human chorionic gonadotrophin (B-HCG) 35 MU/ml (normal 0–10).

After three cycles of POMB-ACE chemotherapy, serum AFP remained elevated (AFP 117 ng/ml, B-HCG 9 MU/ml), and the patient developed a mass in the neck. Aspiration of this mass revealed 23 ml of clear fluid in which both AFP (750 ng/ml) and B-HCG (184 MU/ml) were elevated well in excess of serum levels. Although cytology on the cyst fluid revealed no malignant cells, the elevation of tumour markers within the fluid was taken to indicate the presence of persisting malignant tumour. Despite three cycles of second-line therapy with VIP (etoposide, ifosfamide and platinum), the cervical mass persisted and serum AFP remained elevated, but relatively constant at 75–100 ng/ml for some 10 weeks without showing the rapid rise expected in the presence of growing viable tumour.

In view of the stabilisation of AFP, resection of the retroperitoneal tumour was performed. Extensive microscopic examination of the specimen revealed only immature teratoma with no viable germ cell elements. Serum AFP returned to normal within 2 weeks of this surgery. Three weeks later the neck mass was excised, histology revealing only mature cystic teratoma.

The patient remained well and marker negative until 14 months after surgery when AFP began to slowly rise again. Serial computed tomography and gallium scans remained normal until 5 months after the initial AFP rise, when a superior mediastinal mass was demonstrated, at which point serum AFP measured 54 ng/ml, B-HCG normal. Surgical excision was performed, pathology again revealing immature teratoma with chondrosarcomatous elements, but no malignant teratoma was detected.

The persistence of elevated tumour markers after aggressive platinum-based chemotherapy is of grave prognostic significance, and surgery is seldom considered. The case we describe, however, is distinguished by the very slow rise in AFP, essentially in plateau phase, arguing against the presence of rapidly growing malignant GCT. Moreover, the normalisation of B-HCG and a negative gallium scan suggested the effectiveness of the chemotherapy administered. We postulate that the high levels of AFP present within the benign cystic masses (derived from mature endodermal components) slowly leached into the circulation producing a misleading elevation of AFP.

We conclude that in patients with elevated but only slowly rising AFP following chemotherapy, surgical resection of residual mass should still be considered.

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