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REVIEW OF THE TREATMENT OF TESTICULAR CANCER*

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Epidemiology and Etiology

Tumors of the testis form 1% of neoplasms in males, 90% of germ cell origin and the incidence has been increasing since the 1940's. In England and Wales 1 boy in 500 will develop testicular cancer before the age of 50. A second primary develops in 1.5% of patients. There is no obvious familial incidence. There is considerable variation between the different countries and races, the incidence in Africans being 0.1 per 100.000; in black people in the USA being 0.5 - 1.0 per 100,000; and USA whites 2,8 - 4,5 per 100,000. High incidences are also found in Norway (4,3 - 4.6 per 100,000) and in West Germany (3.0 - 4.7 per 100,000).

In 10 - 12 per cent of patients there is a history of cryptorchism but orchidopexy does not prevent tumor formation. Many patients have a maternal history of infection, especially tuberculosis, epilepsy and previous stillbirth. Tumors are more common among those in the professions than among the working classes[1].

Pathology

The majority of lesions are non-seminomatous. In small numbers Sertoli cell and Leydig cell tumors are also seen.Sertoli cell tumors are found largely in infants and children, and if malignant, metastasize within one year. In adults 40% are associated with gynecomastia. Leydig cell tumors are found in all age groups. In children they are often associated with macrogenitosomia and in adults gynecomastia is common.

Ten percent are malignant, but metastases usually appear after an interval of 3 - 4 years[2].

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* This abstracts is a summary of a meeting held in Erice, Sicily in 1983. The proceedings of this meeting will shortly be available under the title of "Testicular Tumours and Other Tumours of the Genito-Urinary Tract", edited by P.H. Smith and M. Pavone-Macaluso and published by Plenum Press, New York.

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Diagnosis

Professor Blandy emphasized the importance of self examination by the patient; suspicion by the doctor and of exploration by the surgeon[3]. This is of particular importance since the small tumors are often cured by orchiectomy alone, whilst chemotherapy is also curative in many of those with early dissemination.

Classification

Although it may seem logical to use the TNM system many surgeons and physicians prefer to classify the patient as:-

1. Localized at the testicle,

2. With small volume metastases in which the metastases are limited to the retroperitoneal nodes and are less than 5 cm in diameter, and

3. Large volume metastases in which the retroperitoneal nodes are over 5 cm in diameter, or in which lesions are present elsewhere.

Investigation

Changes in the last few years which have been particularly remarkable have been the increasing use of markers - alpha fetoprotein (AFP) and beta human chorionic gonadotrophin (HCG) together with the increasing availability of computerized axial tomography (CT scan).

Both AFP and HCG are usually negative in seminoma. If either is positive a careful search should be made for non-seminomatous elements in the tumor or its metastases. Approximately 40% of patients with non-seminomatous lesions have positive markers[4]. Of the many markers being evaluated placental alkaline phosphatase may prove to be of value in the diagnosis and the management of patients with seminoma[5].

The staging of testicular tumors has been greatly improved by the introduction of CT scanning as is shown in Table I[4].

TABLE I

To Demonstrate the Improvement of Staging in Non-Seminomatous Tumors of the Testicle Following the Use of Markers

	Clinical Stage I	Pathologic Stage I	Staging Error
Without markers	72	52	28 - 32%*
With markers	57	52	9 - 14%*

* Based on three patients considered stage II on follow-up (Javadpour, 1984).

TABLE II

Modification of Clinical Stage by CT Scan

(Showing the value of the CT Scan in diagnosing lesions not otherwise evident)

		Unsuspected metastases shown on CT		
Stage	No. of patients	Abdominal nodes	Mediastinal nodes	Pulmonary metastases
I	21	1	—	4
II	22	—	1	5
III	12	—	—	7
Total	55	1 (1.8%)	1 (1.8%)	16 (29%)

(Peckham, 1984).

The CT scan has also been especially useful in diagnosing small pulmonary metastases which have not shown during investigation by other techniques (Table II).

Professor Peckham summarized the clinical staging of testicular tumors by outlining the necessity for:-

1. Pre-and postoperative markers (every month for the first year, every other month in the second year and yearly thereafter),
2. Histology of the tumor and cord,
3. Lymphography and IVP,
4. CT scan of abdomen and thorax,
5. Ultrasound of liver and retroperitoneum.

His view is that the diagnostic techniques are now sufficiently accurate to allow a policy of surveillance in patients with non-seminomatous tumors apparently localized to the testicle[6].

Treatment

1. Seminoma

For patients with stage I disease and for those with stage II disease in which the nodes are less than 5 cm in diameter, it is still the custom to carry out orchiectomy and radiotherapy to the para-aortic region. In these patients' survival should exceed 90%. For patients with more advanced disease orchiectomy is followed by chemo-

therapy using a platinum containing regime — often cisplatin, vinblastine and bleomycin — which has increased the prognosis from 10 - 30% to over 70% for 5-year survival.

2. Non-Seminoma

It is increasingly practical to carry out a policy of surveillance on patients with stage I disease as long as there is easy access to estimation of markers and CT scanning. In the absence of this supervision retroperitoneal node dissection may still be appropriate for stage I and for early stage II disease.

Stage I. Using this surveillance technique for stage I disease it appears that fewer than 20% of patients are likely to relapse. This form of therapy is unlikely to be of value for embryonal cell carcinomas in which 40% of the patients followed be Peckham[6] relapsed within one year.

Stage II A. 85% of patients survived following radiotherapy and this form of treatment or node dissection may be acceptable. Node dissection is probably perferable since radiotherapy impairs the capacity of the patient to tolerate subsequent chemotherapy if this is needed.

Stages II B, C, III and IV. All these patients should receive chemotherapy with a cisplatinum containing regime. It is common to give 4 cycles of induction therapy and to consider maintenance therapy for up to one year. In an EORTC trial in which low (0.15 mg/kg) and high (0.2 mg/kg) dose of vinblastine was used the high dose showed no evidence of benefit but did have greater toxicity. It is therefore not to be recommended. Following such regimens one may expect an incidence of complete remission in 88% of patients with low volume metastases and in 59% of those with more extensive disease[7].

The introduction of platinum combination chemotherapy has increased the survival of these patients from 10 to 70% within the last decade. If residual disease persists following chemotherapy debulking surgery may be carried out if the patient is marker negative. If still marker positive it is usual to give further induction therapy with an alternative regime before considering surgery.

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