تعليق عليه [WU1]:

UMOURS OF THE TESTES

Testicular cancer represents around 1–1.5 % of male neoplasms and there is clear evidence of an increased incidence.

The vast majority are ***germ cell tumours*** and the peak incidence of (seminomas is in the fourth decade of life, non-seminomatous germ cell tumours in the third decade). They are the most common form of tumour in young men.

Risk factors:

* a history of testicular maldescent.

*a history of a contralateral testicular tumour.

*Klinefelter's syndrome.

Classification and pathology

Tumours of the testis are classified according to their predominant cellular type:

- ***germ cell tumours*** (90–95%) :
seminoma, embryonal cell carcinoma, yolk sac tumor, teratoma, and choriocarcinoma.
- ***interstitial tumours*** (1–2%): *Leydig cell tumours.*
- ***lymphoma*** (3–7 %).
- ***other tumours*** (1–2%).

Seminoma

A seminoma typically has a cut surface which is homogeneous and pinkish cream in color. It appears to compress neighboring testicular tissue. There are

two histological variants, one with a more anaplastic appearance and another that is characterized by cells which closely resemble different phases of maturing spermatogonia (spermatocytic seminoma).

Seminomas metastasize mainly via the lymphatics , and less hematogenous. The lymphatic drainage of the testes is to the para-aortic lymph nodes near the origin of the gonadal vessels. The contralateral para-aortic lymph nodes are sometimes involved by tumour spread, **but the inguinal lymph nodes are affected only if the scrotal skin is involved.**

Non-seminomatous germ cell tumours (NSGCT):

may be tiny but can reach the size of a coconut.

There are a number of histological types of non-seminomatous germ cell tumours (NSGCT), which may coexist within a single tumour:

- **Embryonal carcinoma**. Highly malignant tumours that occasionally invade cord structures.
- **Yolk sac tumour**. Tumours with this component secrete alpha fetoprotein (AFP).
- **Choriocarcinoma**. Often produces human chorionic gonadotrophin (HCG). This is a highly malignant tumour that metastasizes early via both the lymphatics and the blood stream.
- **Teratoma**. yellowish in color with cystic spaces containing gelatinous fluid contain more than one cell type with components derived from ectoderm, endoderm, and mesoderm.

may 'mature' with well-differentiated tissue elements to 'immature' with undifferentiated primitive tissues. All can metastasize.

Interstitial cell tumours

Arise from Leydig or Sertoli cells. A Leydig cell tumour ***masculinises***; a Sertoli cell tumour ***feminises***.

small well circumscribed tumours with a yellow cut surface.

Approximately 10 per cent are malignant.

Most prepubertal tumours (25%):

produce androgens, which cause sexual precocity, Regression of the symptoms after orchidectomy may be incomplete.

Most post-pubertal interstitial cell tumours (75%):

produce feminizing hormones leading to gynaecomastia, erectile dysfunction, loss of libido and azoospermia.

Clinical features

*Usually a painless testicular lump.

*A sensation of heaviness can occur if the testis is two or three times its normal size, but only a minority of patients experience pain.

*In a few cases, an episode of trauma calls attention to the swelling.

*Some cases may simulate epididymo-orchitis.

* rarely some patients present with severe pain and acute enlargement of the testis because of haemorrhage into the tumour.

* Rarely, the predominant symptoms are those of metastatic disease:

Intra-abdominal disease may cause abdominal or lumbar pain and the mass may be discovered in the epigastrium. Lung metastases are usually silent but they can cause chest pain, dyspnea and hemoptysis in the later stages of the disease. The primary tumour may not have been noticed by the patient, and indeed may be so tiny that it can be detected only by ultrasonography.

On examination:

*there is an intratesticular solid mass.

*May with, a lax secondary hydrocele.

*Around 5 per cent of cases have gynaecomastia (mainly the NSGCT).
*Metastatic disease is rarely apparent clinically and is more usually identified by formal staging investigations.

*In 1–2 per cent of cases, the tumour is bilateral at the time of diagnosis.

Investigation :

The diagnosis is confirmed by

1. **ultrasound scanning** of the testis. It is a mandatory test in all suspected cases of testicular tumour.

2. Blood tests to measure the levels of **tumour markers** which are raised in around 50 percent of cases. A rise in **AFP** is seen in around 50–70 per cent of NSGCTs and a rise in **HCG** is seen in 40–60 per cent of NSGCTs and around 30 per cent of seminomas. Also used later to monitor the response to treatment. .
3. Orchidectomy and send for histopathology.

4.chest x-ray will occasionally demonstrate the 'classical' cannon ball metastases

5. **computed tomography** (CT) of chest and abdomen for metastasis. and for monitoring the response to therapy.

Staging of testicular tumours:

In confirmed cases, staging is an essential step in planning treatment.

While **TNM staging** is the most widely used system for the staging of testicular cancer.
the older staging system of stages 1–IV is still considered valuable in determining the treatment options:
The stages are:

- Stage I: the tumour is confined to the testis;
- Stage II: nodal disease is present but is confined to nodes below the diaphragm;

- Stage III: nodal disease is present above the diaphragm;
- Stage IV: non lymphatic metastatic disease (most typically within the lungs)

Treatment

1.Scrotal exploration and orchidectomy for suspected testicular tumour

The orchidectomy is undertaken via an inguinal incision.

2.Management by staging and histological diagnosis (after orchidectomy)

The treatment of patients with testicular tumours is usually successful, even in cases that are advanced at presentation. This largely reflects the excellent response of these tumours to chemotherapy and (for seminomatous tumours) to radiotherapy.

Stage I tumours

Seminomas :

radiosensitive, adjuvant radiotherapy to the para-aortic nodes was the mainstay of treatment for stage I disease.

* excellent response to platinum-based chemotherapy led to chemotherapy for most men with stage 1 seminoma.

*However, current practice uses CT and tumour marker-based surveillance protocols with chemotherapy being reserved for men who demonstrate relapse.

NSGCTs:

are not radiosensitive, but they are highly sensitive to combination chemotherapy (so called BEP chemotherapy).

Some good prognosis NSGCTs can be managed by surveillance protocols (using regular CT scanning and tumour marker means.)

Stage II–IV tumours

*Combination BEP chemotherapy is the mainstay of treatment for stages II–IV seminoma and NSGCT.
* Retroperitoneal lymph node dissection is sometimes needed in some cases of NSGCT when retroperitoneal masses remain after chemotherapy .

Prognosis

Depends on several factors including
the histological type and the stage at presentation.

For seminoma, if there are no metastases, 90–95 % of patients will be alive five years after diagnosis.
If there are poor prognostic features, the survival rate drops to around 70 %.

For NSGCTs, a five-year survival rate of more than 90 %, is achievable in patients with good prognosis tumours while for more advanced tumours the five-year survival rate is about 60%.

TESTICULAR TUMOURS IN CHILDREN

These are usually ***anaplastic teratomas***. They occur before the age of three years and are often rapidly fatal.

TUMOURS OF THE EPIDIDYMIS

These may be:

benign mesothelioma

malignant sarcoma

secondary carcinoma.

They are extremely rare but should not be forgotten when the patient presents with a non-cystic lump in the epididymis.

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