Testicular tumours are uncommon but are increasing in incidence\(^1\). It is curious that despite the fact that the testis is usually easily palpable, a testicular tumor often escapes detection until after it has metastasized\(^2\). Effective chemotherapy means that early stage and low bulk disease are almost always curable, so early diagnosis and accurate staging are important.\(^1\)  

**Epidemiology**  
For an unknown reason the incidence of testicular tumours has been increasing by 15-20% in successive five-year periods\(^3\). Incidence of Testicular tumor is highest in Scandinavia (9 cases/hundred thousand/year) and lowest among the black in the U.S.A. (1 case/hundred thousand/year).\(^1\) Their peak incidence occurs among men aged 25-35 years.\(^3\)  

**Aetiology**  
The following risk factors are thought to be associated with testicular tumours:  
1. Young age.  
2. Maldescent testes  
3. Congenital urogenital abnormalities-hypospadias, inguinal hernia and the 'bell clapper' deformities.  
4. Fetal exposure to maternal estrogens.  
5. Race- Caucasians > blacks.  
6. Trauma and viral (mumps) orchitis.  

The hypothesis is that the above risk factors exert their influences via a common effect. Failure of germ cell differentiation is the initiating step of malignant change, which is promoted by hormononal stimulation in puberty, resulting in a peak incidence in the next 15-20 years\(^5\).  

**Table: 1.0 WHO pathologic Classification of Testicular tumours**\(^4\)  

<table>
<thead>
<tr>
<th>A. Germ cell tumours</th>
<th>B. Sex cord stromal tumours:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Tumours of one histologic pattern-&lt;br&gt;-Seminoma&lt;br&gt;-Spermatocytic seminoma&lt;br&gt;-Embryonal carcinoma&lt;br&gt;-Yolk sac tumour&lt;br&gt;-Polyembryoma&lt;br&gt;-Choriocarcinoma&lt;br&gt;-Teratoma&lt;br&gt;</td>
<td>a. Well differentiated forms-&lt;br&gt;-Leydig cell tumour&lt;br&gt;-Sertoli cell tumour&lt;br&gt;-Granulosa cell tumour&lt;br&gt;</td>
</tr>
<tr>
<td>2. Tumours showing more than one histologic pattern-&lt;br&gt;-Teratocarcinoma&lt;br&gt;-Choriocarcinoma&lt;br&gt;-Others.</td>
<td>b. Mixed forms&lt;br&gt;c. Incompletely differentiated forms.</td>
</tr>
</tbody>
</table>

According to predominant cellular types the most commonly encountered Testicular tumours are classified as:\(^2\)  
- Seminoma (40%)  
- Teratoma (32%)  
- Combined Seminoma & Teratoma (14%)  
- Interstitial tumours (1.5%)  
- Lymphoma (7%)  
- Others (5.5%)  

**Pathology**  
Tumours arise from all the tissue types present in the testis and among them 99% neoplasms are malignant.\(^1\,2\,5\)
Seminoma
Derived from seminiferous tubular epithelium, the cells resemble spermatocytes. Macroscopically, the enlarged testis is found smooth and firm, cut surface is homogenous and pinkish cream in colour.2

Teratoma
Arises from totipotent cells in the rete testis and often contains a variety of cell types. Macroscopically, the usual variety is yellowish in colour with cystic spaces containing gelatinous fluid. Nodules of cartilage are often present1.

Interstitial cell tumours
Arise from Leydig and Sertoli cells, which are usually benign5,6. A Leydig cell tumour masculinizes because it secretes androgens. On the other hand the Sertoli cell tumours feminizes which leads to gynacomastia, loss of libido.2

Carcinoma in situ1
Usually asymptomatic but in 50% cases there is a chance of developing invasive germ cell cancer in 5 years.

Presentation
Every testicular swelling must be assessed with the possibility of tumour in mind.

Presenting complaints
Classical presentation: painless enlargement of the testicle (75% cases). Pain : initial symptom (15% cases). A sensation of heaviness, there may be a history of trauma to the affected side (10%).7

On examination
The testis is enlarged, usually smooth, firm & heavy. Loss of testicular sensation is a very early sign.

Secondary hydrocele occurs in 1 in 7 testicular neoplasm.8 The epididymis becomes flattened or incorporated in the growth and so difficult to feel.2,4

Sign of vas7 : helps in the differential diagnosis between a testicular neoplasm and an inflammatory lesion, which cause the vas to become considerably thickened; in neoplasm it remains normal in all respect.

Rectal examination shows the prostate and seminal vesicle to be normal. Secondary retroperitoneal deposits may be palpable on the side of the tumours. Occasionally the patient may present with the signs of metastasis like enlarged supraclavicular nodes, abdominal or lumber pain, chest pain, dysponea and haemoptysis.

Atypical presentation : Epididymo-orchitis, urinary infection, severe pain and acute enlargement of testis due to haemorrhage into a neoplasm, gynecomastia (mainly in Teratomas in 1-5% patients).2,9

The Hurricane tumour : is a ferocious malignancy that kills in a matter of weeks.2,10

Essentials of diagnosis3
@ Painless, firm mass within the testicle in a man aged 18-40.
@ Elevated serum levels of beta hCG, AFP, LDH or all three.
@ Enlarged retroperitoneal nodes on abdominal CT scan.
@ Palpable abdominal mass in advanced cases.

Differential diagnosis1,11
- Epididymo-orchitis
- Testicular torsion
- A syphilitic gumma
- Granulomatous orchitis
- Tuberculosis.

If there is any doubt about the diagnosis surgical exploration should be undertaken.
Investigations
1. Ultrasound of the testis.
2. Chest radiograph
3. Two separate measurements of AFP and beta-hCG.
4. CT scan and MRI to detect intra-abdominal and intra-thoracic secondaries.\(^1,12\)

Tumour markers
In the context of testicular tumours the value of serum markers is fourfold-
- Evaluation of testicular mass.
- Staging of germ cell tumours.
- Assessing the tumours burden.
- Monitoring the response to therapy.

The biologic markers used in testicular tumours are alpha feto protein (AFP), beta sub-unit of human chorionic gonadotropin (beta hCG), lactate dehydrogenase (LDH), placental lactogen and placental alkaline phosphatase among which the first three are most widely encountered. HCG levels are occasionally raised in seminomas (15%), but AFP elevation indicates the presence of a teratomatous element.

Either (AFP and hCG) or both of these markers are elevated in more than 80% of the patients with non seminiferous germ cell tumours at the time of diagnosis. It can be noted that elevated serum levels of AFP are also encountered with liver cell carcinoma\(^4,15\).

Table: 1.3 Serum tumour marker categories and stage grouping for testis cancer\(^1\)

<table>
<thead>
<tr>
<th>SX</th>
<th>Serum markers not available</th>
</tr>
</thead>
<tbody>
<tr>
<td>SO</td>
<td>Serum marker study levels within normal limits</td>
</tr>
<tr>
<td>LDH</td>
<td>HCG (U/ml)</td>
</tr>
<tr>
<td>AFP (ng/ml)</td>
<td></td>
</tr>
<tr>
<td>S1</td>
<td>&lt;1.5 N and &lt; 5000 and &lt; 1000</td>
</tr>
<tr>
<td>S2</td>
<td>1.5-10 N or 5000-50000 or1000-10000</td>
</tr>
<tr>
<td>S3</td>
<td>&gt;10 N or &gt; 50000 or &gt; 10000</td>
</tr>
<tr>
<td>N: Indicates the upper limit or normal for the LDH assay.</td>
<td></td>
</tr>
</tbody>
</table>

Metastases
Lymphatic spread common to all form of testicular tumours and in general the retroperitoneal para aortic nodes are the first to be involved. Subsequent spread may occur to the mediastinal and supraclavicular nodes.\(^16\) Seminoma tends to remain in the testis for a long time (70% present in clinical stage1) and usually spread via lymphatics. Hematogenous spread primarily to the lungs but liver, brain and bones are also involved. Teratomas not
only metastasize earlier but also use a haematogenous route more frequently. Management
Management requires expertise in chemotherapy, radiotherapy and surgery, in addition to active surveillance with CT scanning. Inguinal orchiectomy with high ligation of the cord at the internal ring is the proper initial treatment for all kind of testicular tumours.

Seminoma
Seminomas are very sensitive to radiotherapy.

Stage 1 and Low volume stage 2 diseases
Radiotherapy to para-aortic and ipsilateral pelvic Nodes.

Large stage 2 tumour volume
Cisplatin based chemotherapy is used as relapse rate after radiotherapy is high.

Stage 3 and 4 disease
Cisplatin based chemotherapy, with radiation for residual disease.

Teratoma
Stage 1 with negative serum markers
Orchiectomy followed by intensive surveillance. If the disease is progressive chemotherapy or retroperitoneal lymph node dissection is done.

Stage 1 with positive serum markers/definite metastatic disease chemotherapy until the Markers are negative.

Stage 2 and 3 disease - Four to six cycles of chemotherapy followed by surgical excision of residual disease.

Chemotherapy
Cisplatin in combination with vinblastine & bleomycin forms the basis of modern chemotherapy for Teratomas.

Treating persistent or recurrent disease
Intensive chemotherapy, surgical resection, and, occasionally, radiotherapy are required in persistent or recurrent cases. High dose chemotherapy with stem cell support is usually considered (Success rate- 30%)..

Treatment toxicity
Chemotherapy with Cisplatin causes nausea and vomiting, alopecia, fatigue, neutropenia, and sepsis. Bleomycin may cause lung toxicity and fatal pneumonitis as well (0.5-1% case).

Prognosis
Prognosis of testicular tumours depends on histological type and the stage of the growth. Large tumours volume, high levels of tumour markers, presence of undifferentiated tumours in primary and involvement of liver, lungs, bone or CNS indicate unfavorable prognosis. For patients with favorable prognostic factors the long-term survival rates are over 90%.

Follow up
The aim of follow up care is to detect a relapse, to monitor and treat toxicity related to therapy, to detect contra lateral cancers, and to offer support and counseling. A regular follow up should comprise clinical examination, estimation of serum markers and chest radiograph.

Further development
The optimum treatment for metastatic non-seminomatous germ cell tumours inpatients with a good prognosis has been defined but the treatment of patients with poor prognosis and recurrent disease still needs improvement. Surveillance may become more appropriate, especially if staging accuracy improves. Public education and awareness must also be priorities to help patients avoid unnecessary delays in
presentation that may have an impact on survival. But in Bangladesh yet there is no actual study regarding the incidence, mode of presentation, treatment and final outcome of the patients presenting with testicular tumours. Therefor, a multiconstitutional, prospective, randomized clinical study is necessary to improve the survival and quality of life of the patient with testicular tumour in others countries.

References
3. Surgical Diagnosis & Treatment, 10th Ed; 959.
4. Robbin's Pathologic basis of Disease, 6th Ed; 1018,1024.
Review Article
