

Germ Cell Tumors:

- Seminomas.
- Non-seminomatous germ cell tumors.

	Epidemiology	Clinical appearance	Histology	Metastasis	Prognosis
Seminoma	<ul style="list-style-type: none"> - Rare in prepubertal children - Age: 40-50 	<p>Tumor appearance:</p> <ul style="list-style-type: none"> - Pale - Fleshy - Homogenous - Soft - Well-demarcated <p>NO HEMORRHAGE OR NECROSIS</p>	<ul style="list-style-type: none"> - Large uniform cells with distinct cell borders. - Clear, glycogen-rich cytoplasm - Round large nuclei - Cells arrayed in small lobules - Intervening delicate fibrous septa - Lymphocytic infiltrate 	<ul style="list-style-type: none"> - Remains localized to the testis for a long period of time. - Metastases to paraaortic and Iliac LN's. - Hematogenous metastasis: Could occur later. 	Good Prognosis

Non Seminomatous Germ Cell Tumors (NSGCT)					
Embryonal Carcinoma	Age: 20-30	<ul style="list-style-type: none"> - Ill-defined mass (unlike seminoma) - Foci of hemorrhage and necrosis 	<ul style="list-style-type: none"> - Sheets of undifferentiated cells + Primitive gland-like structures. - Nuclei: Large & hyperchromatic - Indistinct cell borders (Unlike seminoma) 		Bad Prognosis More aggressive than seminoma.
Yolk Sac Tumors	Most common primary testicular neoplasm in children < 3 yo	<ul style="list-style-type: none"> - Serum Tumor marker: AFP- Alpha fetoprotein. - Mass is well demarcated (Unlike embryonal carcinoma) 	<ul style="list-style-type: none"> - Schiller-Duvall Bodies: Structures resembling primitive glomeruli 		Children: Good prognosis Adults (rare): Worse prognosis
Choriocarcinoma	Age: 20-30	<ul style="list-style-type: none"> - Serum: High level of HCG - Necrosis and Hemorrhage: extremely common. 	<ol style="list-style-type: none"> 1. Syncytiotrophoblasts: Large multinucleated cells containing HCG. 2. Cytotrophoblasts: Single, fairly uniform nucleus. 	- Early metastasis to the lungs and CNS	Bad Prognosis

Teratoma	<ul style="list-style-type: none"> - All ages. Common in infants and children. - 2nd most common after Yolk Sac tumors. 	<ul style="list-style-type: none"> - Reminiscent of the normal derivatives of more than one germ layer (ectoderm, mesoderm, endoderm) 	<ul style="list-style-type: none"> - Different types of mature tissues. 	<p>In post pubertal males: Malignant → capable of metastasis.</p>	<p>Prepubertal males: Benign course – Good prognosis</p> <p>Post pubertal males: Malignant – Bad prognosis.</p>
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Prostate Pathology:

	Epidemiology and Location	Pathogenesis	Clinical Symptoms & Appearance	Microscopic appearance	Notes
BPH Benign Prostatic Hyperplasia	<ul style="list-style-type: none"> - Age: Common in men > 40 - Location: Transitional zone (inner) of the prostate + part of the central zone. 	<ul style="list-style-type: none"> - Androgen-dependent proliferation of epithelial and stromal elements. - DHT is synthesized in the prostate. It supports growth of prostatic epithelial & stromal cells by binding to androgen receptors. 	<p>Morphology:</p> <ul style="list-style-type: none"> - Enlarged prostate by well circumscribed nodules bulging from the cut surface. - Compressed urethra (since BPH involves the inner portions of the prostate) 	<p>Proliferating glands & fibromuscular stroma.</p> <p>Hyperplastic glands are lined by 2 cell layers:</p> <ol style="list-style-type: none"> 1. Tall columnar epithelial cells. 2. Peripheral flattened basal cells. 	<p>Does not occur in males with genetic diseases that block androgen activity (androgen-dependent).</p>

			<p>Clinical Symptoms:</p> <ul style="list-style-type: none"> - Hesitancy - Lower urinary tract obstruction (Risk of UTI) - Nocturia, frequency and urinary urgency. - Intermittent interruption of urinary stream. 		
Prostate Carcinoma	<ul style="list-style-type: none"> - Most common form of cancer in men >40. - Location: Peripheral zone of the prostate. - 	<ol style="list-style-type: none"> 1. Androgens: Doesn't develop in males castrated before puberty. 2. Environment: Westernized dietary habits. 3. Heredity: Increased risk among affected first degree relatives with prostatic cancer. 4. Acquired somatic mutations: TMPRSS2-ETS fusion genes 	<ul style="list-style-type: none"> - Digital rectal examination: Palpable hard nodules. - Elevated serum Prostate Specific Antigen (PSA) level. 		<p>Bone metastases → osteoblastic lesions on bone scans.</p>

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