ACEM Primary Examination Vivas > Pathology > Neoplasia Organised by edvivas.com	
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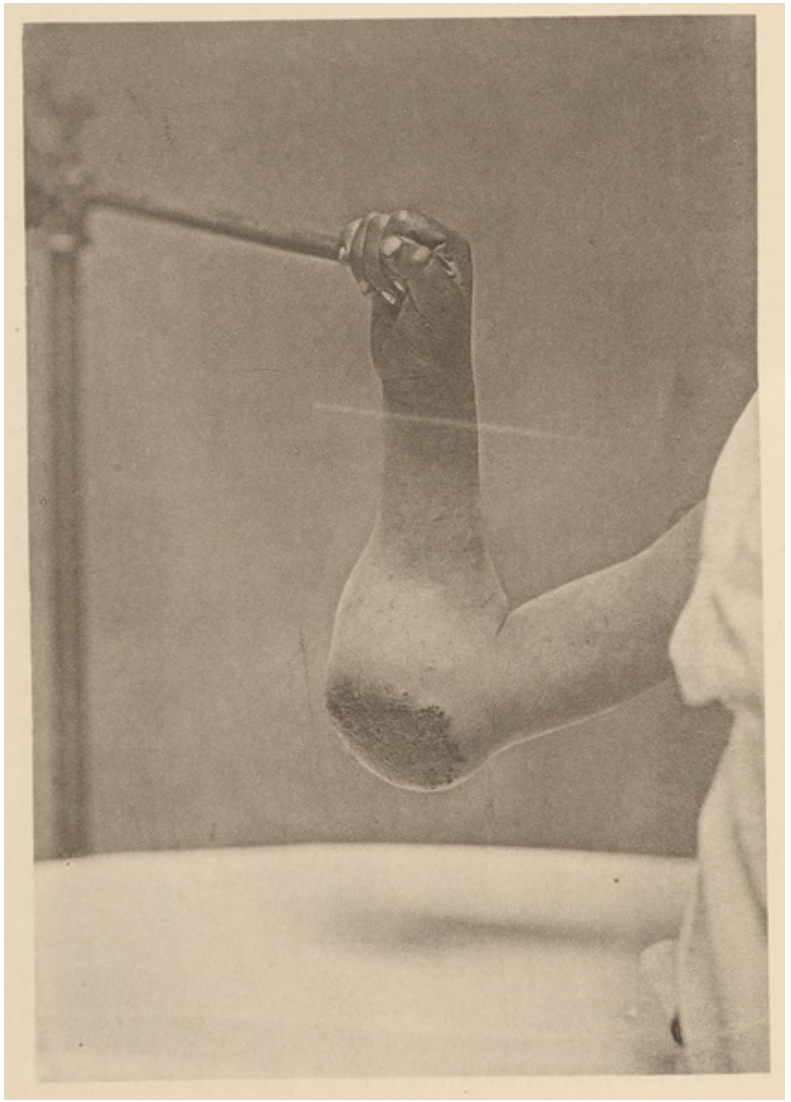

Invasion of ECF 2005-1

Invasion of extracellular matrix	How do cancers invade the extracellular matrix?. 3 out of 4	1. Loosening of intercellular junctions a. Downregulation of E-cadherin expression b. Mutations in gene for catenins 2. Attachment to BM / interstitial connective tissue components a. ↑density of laminin and intregrins 3. Degradation of ECM a. Proteases – by tumour cells or induced stroma cells b. 3 classes – serine, cysteine, matrix metalloproteases (MMPs) c. type IV collagenases (eg MMP9), plasminogen activators 4. Migration of tumour cells a. Within circulation, aggregate in clumps – homotypic / tumour-plt aggreg's b. arrest and extravasation of tumour emboli at distant sites c. adhesion to endothelium, egress through BM (same mechanisms as above)	
	What is the importance of matrix metalloproteinases in this process?	1. collagenases produced by tumour cells or surrounding stromal tissue 2. cleave type IV collagen of epithelial and vascular BMs 3. generate, from ECM, factors that promote angiogenesis, tumour growth and tumour cell motility	

Metastasis 2005-1

Metastasis	How do tumour cells metasise? Prompt "at a cellular level"	1. Invasion of extracellular matrix ~ clonal expansion, growth, diversification, angiogenesis ~ invasion of ECM ~ metastatic subclone ~ adhesion to and invasion of BM ~ passage through ECM ~ intravasation (blood/lymphatic) 2. vascular dissemination and homing ~ interaction with host lymphoid cells ~ tumour cell embolus...platelet tumour aggregates ~ adhesion to BM ~ extravasation ~ metastatic deposit ~ angiogenesis ~ growth	
	Why do some tumours metatasise to sites other than their natural blood and lymphatic drainage areas	a) adhesion molecules whose ligands expressed preferentially on target organs b) chemokine receptors for target chemokines highly expressed in some organs c) target tissue may be unpermissive environment (eg skeletal mm)	

Paraneoplastic Syndrome 2005-1

Paraneoplastic syndrome	What is a paraneoplastic syndrome?	Symptom complex in cancer-bearing patient not readily explained by tumour spread local or distant, or by hormones produced by the tumour tissue itself	
	What are the mechanisms by which they can occur?	~ Ectopic hormone production* and give example ~ Immunologic/autoimmune Eaton-Lambert, dermatomyositis ~ Tumour antigens	
	examples	Cushings syndrome (ACTH) with small cell lung /pancreas/neural cas; hypercalcemia (PTH peptide) with sq lung /breast/renal/ leuk/ovarian; SIADH small cell lung /brain; hypoglycemia, carcinoid, Myasthenia-like, Acanthosis nigrans, Hypertrophic osteoarthropathy, thrombosis	

Paraneoplastic Syndrome 2009-2

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1:	What is a paraneoplastic syndrome?	A complex of symptoms that cannot be readily explained by the local or distant spread of a tumour or by elaboration of hormones from the tissue in which the tumour arose.	Generally accurate description required to pass
Question 2:	What are the main types of paraneolastic syndromes?	**1. Endocrinopathies** - Cushing - Small Cell Ca lung (ACTH) - SIADH - Small Cell Ca lung, intracranial (ADH) - Hypercalcemia - Squamous Cell Ca lung, breast (parathyroid like hormones, TNF, TGF, IL-1) - Carcinoid – bronchial adenoma, ca pancreas and stomach – serotonin/bradykinin) - Polycythemia – Renal (EPO) **2. Nerve and Muscle Syndromes** - Myasthenia (bronchogenic Ca - ? immune mechanism) - CNS/neuro (breast) **3. Dermatological** - Acanthosis Nigricans (gastric, lung, uterine) - Dermatomyositis (bronchogenic. Breast) **4. HPOA** - bronchogenic	Endocrinopathies with at least 2 examples and at least one other to pass. Prompt: What syndromes or abnormal laboratory findings may be related to these syndromes? What are the mechanisms of these syndromes?
Question 3:	What is the cause of cachexia in cancer?	Not generally understood Anorexia Elevated BMR ? humoral factors – TNF, cytokines, Other tumour produced factors.	

Invasion of ECF 2009-1

Question 3: **Tumour invasion and metastasis**	**Describe the steps involved in tumour cell invasion of the extracellular matrix**	1. **Detachment** ('loosening up') of the tumour cells from each other, with breaking of intercellular bonds 2. **Attachment** to extracellular matrix (ECM) components, via laminin and fibronectin receptors 3. **Degradation** of ECM, via type IV collagenase and plasminogen activator, creating passageways 4. **Migration** of tumour cells, which may then lead to vascular dissemination	Accept ≥ 3 of 4 **bolded words** (or a similar explanation) for a pass? Prompt: "*Detachment is the first step.*"
	Describe possible mechanisms that influence the distribution of metastases	1. **Tumour cell adhesion molecules** ligands *preferentially* expressed on target organ cells 2. **Chemokines** *for* target tissues 3. **Chemoattractants** *from* target organs	Prompt: "*Chemokines have an important role*"

Tumours in General 2013-2-A

Her symptoms are due to complications of lung cancer			
Pathology: Clinical effects of tumours	What is the definition of a neoplasm?	**Abnormal growth of a tissue** **Growth exceeds and is uncoordinated with that of the original tissue** **Growth continues in the absence of the stimuli which evoked the change** **(preys on host and serves no purpose)**	Must get the gist of all 3
	How may a malignant tumour affect the 'host'?	**Local and metastatic direct effects**. Pressure, Bleeding, ulceration, rupture and infarction. **Cachexia** **Hormonal**	3 of 4 bold
	(Prompt: what is meant by paraneoplastic syndrome?) Give examples of paraneoplastic endocrinopathies	**Paraneoplastic**: - *Endocrinopathy with 3 examples (Cushings, SIADH, Ca++ up, hypoglycaemia, Carcinoid synd, polycythaemia)* - *Nerve and muscle – myasthenia,* - *Skin - acanthosis nigricans, dermatomyositis* - *Bone: HPOA and clubbing* - *Blood/Vascular: anaemia, venous thrombosis*	3 examples of paraneoplastic syndrome