O&G and Paeds VIVAs (Pathology)



Aug 2015

2014.1.C.3

Question 4 Pre-eclampsia Subject: Path LOA: 2	4.1 Describe the pathogenesis of pre - eclampsia.	4.1 Endothelial dysfunction, vasoconstriction leads to hypertension, increase vascular permeability causing proteinuria & oedema.	Bold + 1 to pass
	4.2 What is the clinical course of pre eclampsia?	4.2 > 34 weeks typically has HT, oedema, proteinuria Headache and visual disturbance Eclampsia is progression to seizures and coma	2/3 bold to pass (prompt: what happens in untreated pre-eclampsia?)
	4.3 What morphological changes occur in the placenta?	4.3 Infarcts, haematomas, villous ischaemia, syncytial knots, fibrinoid necrosis	1 to pass

2012.1.1

Question 5	What is Sudden Infant Death	The sudden death of an infant under 1 year of age which remains	Accurate definition (age
	Syndrome?	unexplained after thorough investigation and autopsy.	& unexplained nature)
ALTE/SIDS			
	What risk factors have been identified?	Parental risks- young mum <20, maternal smoking or drug use, low SES,	At least 3 risk factors
LOA: 2		deficient pre-natal care	1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1
		Infant risks- premature, low BW, male, SIDS in sibling, brainstem	
		anomalies.	
		Environment- prone sleeping, soft bedding and co-sleeping,	
		hyperthermia	

2008.1

Q5. Pre-eclampsia	What are the proposed pathogenesis and consequences of pre-eclampia?	Placental ischaemia is the key feature leading to 1.Reduction in PGI2, PGE2, 2.Inc renin/angiotensin II, 3.Inc thromboxane and endothelial dysfunction, 4.Resulting in systemic hypertension & DIC.	Pass criteria: placenta ischaemia & 1 other point.
	Describe the clinical course of pre- eclampsia.	Usually starts after 32 weeks gestation, charcaterised by 1.Hypertension, oedema and proteinuria. 2.Headache and visual disturbances are common. 3.Ecclampsia is characterised by convulsion and coma.	Need 2/3 to pass.
	Describe the morphological changes in the placenta.	1. Placental infarcts, 2. retroplacental haematoms, 3. villous ischaemia, 4. prominent syncytial knots, 5. thickened trophoblastic basement membrane, 6. villous hypovascularity, 7. fibrinoid necrosis, 8. intramural lipid deposition.	Needs to give 3 to pass.