malignant hyperthermia

features of malignant hyperthermia

Timing	Clinical signs	Changes in monitored variables	Biochemical changes
Acute	Sustained jaw rigidity after succinylcholine		
	Tachypnoea	Increased minute ventilation	
	Rapid exhaustion of soda lime	Rising end-tidal CO_2	Increased $PaCO_2$
	Hot soda lime canister		Decreased pH
	High pulse rate	Tachycardia	
	Irregular pulse	Ventricular ectopics	Increased $[K^+]$
		Peaked T waves on ECG	
Intermediate	Patient hot to touch	Rising core body temperature	
	Cyanosis	Falling SpO_2	Decreased PaO_2
	Dark blood in wound		
	Irregular pulse	Ventricular ectopics	Increased $[K^+]$
		Peaked T waves on ECG	
Late	Generalized muscle rigidity		
	Prolonged bleeding		
	Dark urine		Increased creatine kinase
	Oliguria		Myoglobinuria
	Irregular pulse	Ventricular ectopics	
		Peaked T waves on ECG	Increased $[K^+]$
	Death		

- muscle rigidity (esp trismus) is due to continuous actin-myosin interaction & either follows suxamethonium or develops during the course of anaesthesia
- body temperature rises by 1 degree every ten minutes
- DIC may occur as a result of the release of tissue clotting activators from muscle & through the resulting hyperthermia

general

- malignant hyperthermia is an inherited disorder of the skeletal muscle that can be pharmacologically triggered to produce a combination of hypermetabolism, muscle rigidity and muscle breakdown
- ther reporteed incidence of varies from 1:40000 to 1:50000 anaesthetics
- estimates of population prevalence of genetic susceptability are between 1:5000 & 1:10000
- the diagnosis of malignant hyperthermia may not be obvious at first
- the primary features of malignant hyperthermia are a direct consequence of loss of skeletal muscle calcium homoestasis with a resulting increase in intracellular calcium ion concentration

drug use in malignant hyperthermia

Contraindicated drugs	Safe drugs
Halothane	Nitrous oxide
Enflurane	Barbiturates
Isoflurane	Propofol
Desflurane	Etomidate
Sevoflurane	Ketamine
Succinylcholine	Opiates
Verapamil	Amide/ester local anaesthetics
Nifedipine	Noradrenaline
Diltiazem	Adrenaline
	Dopamine
	Dobutamine

treatment

general
1. abandon the procedure or terminate surgery as soon as possible
2. stop inhalational agents & maintain anaesthesia with iv drugs whilst surgery is concluded
3. give 100% oxygen and hyperventilate with 2-3Xs predicted minute ventilation
4. active cooling measures should be commenced including infusion of cold iv solutions, application of ice to the axillae and groins and a cooling mattress
5. give dantrolene by rapid infusion. Dantrolene is the only drug which is effective in limiting the accumulation of calcium within muscle cells. 20mg of dantrolene is in vials with 3gm of mannitol & requires 60ml of water to reconstitute. Repeated doses of dantrolene should be administered until pyrexia, tachycardia & rise in ETCO2 subside
6. give a large dose of glucocorticoid (eg 2gm of methylprednisilone)
7. acidosis is treated with HCO3- and hyperkalaemia is treated with treatment guided by regular blood gases and electrolyte measurements
8. a diuresis of 2ml/kg/hr is maintained to limit renal tubular damage by myoglobin
9. given inotropes to maintain CO
10. be aware that body temperature may be unstable for 24-48 hours
11. after acute episode monitor for electrolyte abnormalities, myoglobinuria and DIC

if procedure cannot be abandoned then:
(i) use a regional block
(ii) use safe agents

ensure follow-up with patient and family