# MALIGNANT HYPERTHERMIA /PICKLED PIG

# MALIGNANT HYPERTHERMIA

- MH is a hypermetabolic syndrome involving skeletal muscle characterized by hyperthermia, tachycardia, tachypnea, increased oxygen consumption, cyanosis, cardiac dysrhythmias, metabolic acidosis, respiratory acidosis, muscle rigidity, unstable arterial blood pressure, and death.

# ETIOLOGY

- It is an inherited pharmacogenetic disorder of humans, swine, dogs, and horses
- MH is inherited as an autosomal recessive gene in swine *but as an* autosomal dominant gene in humans, horses, and dogs.
- Genetic mapping of the MH locus in pigs and humans placed it in the vicinity of the RYR1 gene , which encodes the sarcoplasmic reticulum ryanodine receptor (calcium release channel).

# OCCURENCE

- When exposed to halogenated anesthetics or succinylcholine, genetically MH susceptible (MHS) individuals exhibit tachycardia , hyperthermia, elevated carbon dioxide production, and death if the anesthetic is not discontinued.
- In swine and human metabolic acidosis and muscle rigidity are severe.
- In dogs metabolic acidosis is usually moderate & muscle rigidity is minimal.

- In swine, stresses such as fighting, transport, and exercise also trigger its onset & it is found in **porcine stress syndrome**.
- Halothane causes MH in pig & horses (persistant muscle contraction due to release of $Ca^{++}$ from sarcoplasmic reticulum) & hyperthermia in others.
- Serum CK and AST enzyme activities are markedly elevated because of extensive myonecrosis.

# CLINICAL SIGNS

- ↑sed muscle metabolism & muscle contracture are due to the effects of the RYR1 mutation on the gating properties of the $Ca^{++}$channel.
- REASON:- As $Ca^{++}$ release channels opens,there is efflux of $Ca^{++}$ from the SR terminal cisternae into the myoplasm, which is exacerbated by the MH triggering agents. The SR calcium ATPase is unable to resequester the $Ca^{++}$back into the SR lumen fast enough, and the myoplasmic $Ca^{++}$concentration rises.

- The resulting MH episode is due to $Ca^{++}$ stimulation of phosphorylase, myofilament contractile activity, & the resultant activation of aerobic & anaerobic metabolism to fuel the contraction.

# Clinical sign….

- Muscle stiffness or fasciculations.
- Ventricular tachycardia develops early and continues until serum $K^+$ reaches cardiotoxic levels.
- Blanching and erythema followed by blotchy cyanosis in the skin of light-colored animals.
- Body temp. rapidly increases & can reach 113° F (45°C).
- Disease is usually fatal.

- Rigor mortis develops within minutes, and muscle temperature is significantly increased.
- Affected muscles of the dead animal are pale, soft and appear exudative or wet.
- Pale, soft exudate pork syndrome is often linked to MH.

# DIAGNOSIS

- Exposure of animal to a volatile anesthetic or stressful event.
- CAFFEINE CONTRACTURE TEST - involves in vitro exposure of extracted muscle tissue to caffeine and halothane. Muscle from MH-susceptible subjects will contract when exposed to lower concentrations of caffeine and halothane, compared with normal muscle.

- MOLECULAR GENETIC TEST - This DNA-based assay is performed on a small sample of anticoagulated blood to detect mutation in the ryanodine receptor gene and can identify homozygous MH-resistant and MH-susceptible animals as well as heterozygous carriers.

# TREATMENT

- Early detection, during giving anesthesia.
- Exposure to the volatile anesthetic must stop.
- Breathing tubes and $CO_2$ canisters must be changed.
- Dantrolene sodium must be given at 4-5 mg/kg, IV & that to early in the course of the disease because muscle blood flow is significantly reduced as the disease progresses.

– SUPPORTIVE TREATMENT

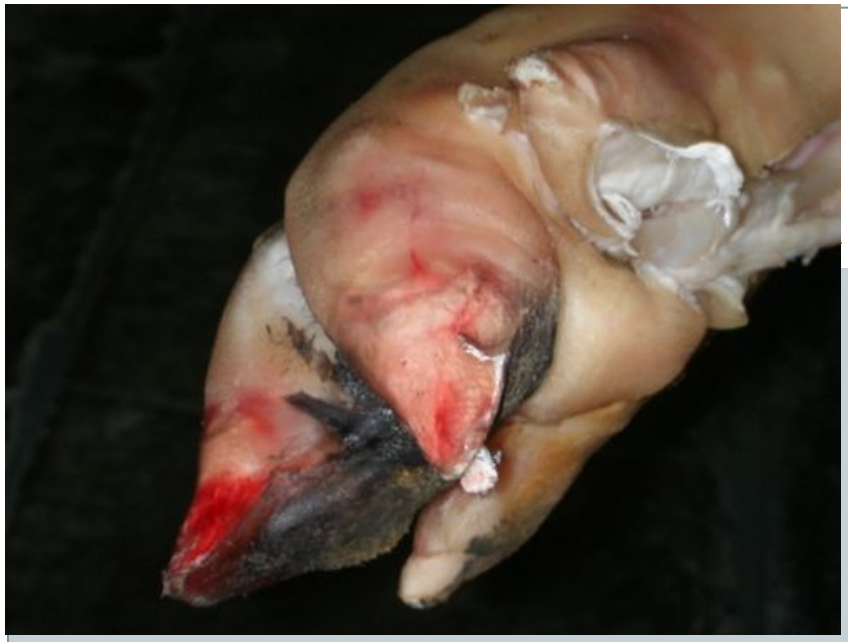
- Fluid therapy
- Management of acidosis through ventilatory support and administration of sodium bicarbonate.
- Increases in core body temperature can be managed by surface cooling and/or chilled saline lavages.
- Other supportive measures include oxygen enrichment of inspired gases and treatment of cardiac dysrhythmias.

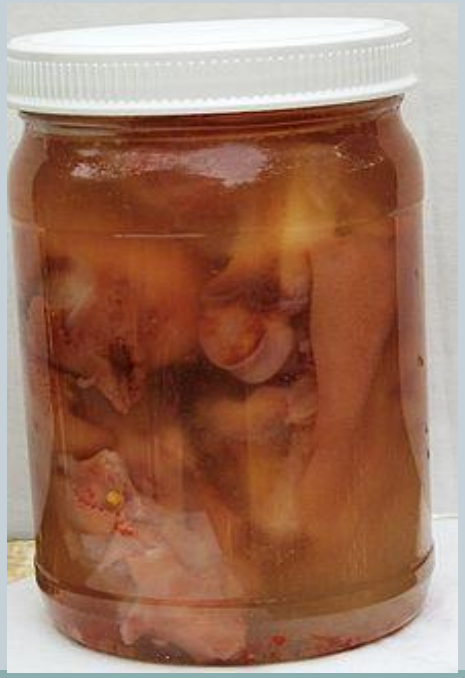
# CONTROL

- Genetic selection.
- With the advent of DNA-based assays, it is possible to cull MH-susceptible animals and carriers.
- Better managemental practices to minimize stress should be followed.
- If a documented MH survivor or a suspected susceptible animal requires anesthesia and surgery, dantrolene should be given at 3-5 mg/kg, PO, 1-2 days before anesthesia.

- Acepromazine and droperidol inhibit development of MH, and propofol has not been reported to trigger MH.
- Amide local anesthetics are safe to use in MH-susceptible animals

- Pickled pigs feet is a type of pork associated with Cuisine of the Southern United States, African American soul food, and Korean cuisine.






MATT & DANA
PICKLED
PIGS FEET

- The feet of hogs are typically salted and smoked in the same manner as other pork cuts, such as hams and bacon.
- It is common to preserve them in a manner very similar to home canning and processes for pickled vegetables; typically a saturation of hot vinegar brine is used. Such methods allow them to be preserved without the need for refrigeration until the jar is opened.

- Pigs feet that are pickled are usually consumed as something of a snack or a delicacy rather than as the primary focus of a meal as its meat course.
- However, pigs feet are not always pickled and in the aforementioned cultures, may be cooked as a part of a meal, often with vinegar and water to preserve their natural flavor. They have a high fat content, with almost an equal portion of saturated fat to protein.