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# Malignant Hyperthermia: Clinical Management

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**ABSTRACT**

Malignant hyperthermia is a subclinical myopathy in which general anesthesia triggers an uncontrollable contraction of skeletal muscle that leads to a life-threatening hypercatabolic state and an increase in body temperature. The disease is primarily autosomal dominant; mutations in receptors (especially ryanodine receptor type 1) predispose to volatile anesthetic agents or succinylcholine causing an accumulation of intracellular calcium in skeletal muscle that leads to its overactivation and hypermetabolism. In the acute setting, diagnosis is based mainly on clinical presentation and end-tidal capnography, which reveals an increase in end-tidal CO2. Initial treatment measures include discontinuation of the agent causing the reaction and administration of dantrolene. In non-acute settings, there are specific diagnostic tools such as the caffeine-halothane contracture test to confirm suspected cases. MH is a lethal disease and has a high mortality rate if not treated early.

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## INTRODUCTION

Malignant hyperthermia is a disorder of pharmacological origin that affects skeletal muscle, which occurs as a consequence of a hypermetabolic response to potent anesthetic gases such as halothane, sevoflurane, desflurane, and the depolarizing muscle relaxant succinylcholine, and rarely with vigorous exercise and is exposed to excessive heat. The incidence of reactions is 1:50,000 per 100,000 anesthetics. HD affects humans, some breeds of pigs, dogs, horses, and possibly other animals. The classic signs of malignant hyperthermia include hyperthermia, marked increase in heart rate, tachypnea, increased carbon dioxide production, increased oxygen consumption, acidosis, muscle rigidity, and rhabdomyolysis, all related to an exaggerated metabolic response to substances. mentioned above. 1.2

The syndrome is likely to be fatal if not treated quickly and properly. Early recognition of the signs of MH, in particular the elevation of carbon dioxide at the end of expiration, provides clues for early clinical diagnosis. The pathophysiological changes of MH are due to the uncontrolled increase in myoplasmic calcium, causing rigidity that in turn activates biochemical processes related to muscle activation, causing ATP depletion. Due to ATP depletion, the integrity of the muscle membrane is compromised, leading to hyperkalemia and rhabdomyolysis in almost all cases. In most cases, the syndrome is caused by a defect in the ryanodine receptor. 3.4

## ETIOLOGY

It has been identified that one of the most common etiologies involved in this pathology is the mutation in the RYR-1 gene located on chromosome 19q13.1. The diagnostic methods that have been found are summarized in the interaction of the muscle sample and its reaction to substances such as halonate and caffeine. Genetic tests to measure susceptibility to this pathology are increasing and can be used more frequently and thus be used routinely. The antidote of choice in these cases is dantrolene, which is a specific antagonist in the pathophysiological changes involved in this pathology and thus can reverse them. Thanks to impressive advances in understanding the clinical manifestations and pathophysiology of the syndrome, mortality from MH has

been significantly reduced, now occurring in less than 6% of patients.

## DIAGNOSIS

Diagnosis is generally clinical, based on intraoperative signs and symptoms (eg, muscle and jaw rigidity, hyperthermia) related to increased end-tidal CO2 and signs of muscle breakdown. Patients with a positive family history, suspicious medical history, or masseter muscle stiffness should receive confirmatory preoperative testing to rule out the risk of HM and, if so, take all necessary precautions to avoid a fatal conclusion. 7

## PREOPERATIVE DIAGNOSES

Gold Standard: Caffeine-Halothane Contracture Test (CHCT)

Some muscle is obtained under regional anesthesia, the muscle sample is processed to determine the viability of the tissue. After tissue viability testing by electrical stimulation, the muscle strips are placed in a bath. Three strips are exposed to halothane at different concentrations and the remaining three strips are exposed to caffeine. If any of the fibers contract, the test is considered positive and a definitive diagnosis of HM would be made. 8

Disadvantages: Only available at select testing centers, which will likely require the patient to travel. 8

Molecular genetic tests: low sensitivity, but highly specific and less expensive and invasive than CHCT. 8

## INTRAOPERATIVE DIAGNOSTICS

**Clinical features:**

Early signs: increased heart rate, tachypnea, cyanosis, general rigidity. 9

Late signs: elevated body temperature that can reach 45°C/113°F, signs of secondary organ damage such as complex arrhythmias or bundle branch blocks, oliguria secondary to acute kidney injury, hemorrhage and/or thrombosis due to disseminated intravascular coagulation, myoglobinuria, pain, swelling and weakness of the affected muscles, important respiratory parameters: continuous increase in CO2.9

Blood tests: arterial blood gases: ↑ pCO2, ↓ pO2, and ↑ lactate (mixed respiratory and metabolic acidosis), electrolyte abnormalities: hyperkalemia, hypercalcemia, myoglobinemia, ↑ creatine kinase. 9

Treatment

Suspension of possible triggering agents and immediate administration of the antidote of choice: dantrolene (antagonist of the ryanodine receptor). 9

**Mechanism of action**

Ryanodine receptor antagonist. 9

Prevents release of calcium from the sarcoplasmic reticulum of striated muscle → reduced muscle rigidity and hyperthermia. 9

**Indications**

Neuroleptic malignant syndrome. 9

Malignant hyperthermia. 9

Adverse effects

CNS: headache, dizziness, mental/mood changes, seizures, hallucinations, insomnia, malaise. 9

Gastrointestinal: nausea, diarrhea or constipation, vomiting. 9

Other: muscle weakness, allergic reaction, blood count changes. 9

**Interactions**

Increase in the activity of nondepolarizing muscle relaxants. 9

Dantrolene interacts with calcium antagonists, potentially leading to hyperkalemia, which can be unpredictable → cardiac arrhythmias. 9

Cooling measures (e.g., ice packs, cool water blankets, ice-water immersion, IV iced saline, forced air cooling). 9

## CONCLUSION

Knowledge of the pathophysiology of MH helps to understand its clinical manifestations, which in turn should lead to proper diagnosis and successful treatment. Activated carbon filters are a recent application that will help remove the anesthetic that triggered the reaction, but the key to preventing mortality and morbidity is rapid administration of dantrolene. Counseling and referral of the patient for diagnosis and family follow-up is the responsibility of the anesthesiologist.

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