Journal of Radiology Nursing 36 (2017) 152–157

Contents lists available at ScienceDirect

Journal of Radiology Nursing

journal homepage: www.radiologynursing.org

Malignant Hyperthermia CE

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Keywords:
Malignant hyperthermia
Dantrolene
Out of the operating room
Anesthesia
Radiology & imaging
Radiology nursing

A B S T R A C T

With the increase in need for anesthesia services outside the operating room, nonoperating room personnel are exposed to situations unique to the surgical environment. Malignant hyperthermia (MH) is an inherited genetic disorder that is triggered specifically by drugs and gases used to induce and maintain anesthesia. If left untreated, this disorder can progress into a cascade of events that can ultimately lead to death. The purpose of this review is to provide radiology and imaging personnel with the knowledge to assist anesthesia providers in the diagnosis, evaluation, and treatment of MH.

Introduction

Malignant hyperthermia (MH) is a pharmacogenetic-inherited disorder specific to anesthetics that has the potential for serious morbidity and mortality if not promptly treated. With the increase in anesthesia services outside the operating room (OOR) and the integration of interventional cardiology, radiology, and imaging into hybrid platforms, nonperioperative health care providers need to be prepared for anesthesia emergencies. Approximately, a third of anesthesia cases reported to the National Anesthesia Clinical Outcomes Registry between 2010 and 2014 were performed OOR (Nagrebetsky, Gabriel, Dutton, & Urman, 2017). It is therefore crucial that radiology and imaging personnel be appropriately trained and prepared in the issues specifically affecting patients undergoing anesthesia.

The purpose of this focused review is to offer radiology and imaging personnel the knowledge to assist in the preparation, evaluation, and treatment of a potentially life-threatening disease contributable to anesthetics.

Funding and conflicts of interests: Both authors report no conflicts of interests, and this project was not funded and did not receive any financial support.

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Background

MH affects skeletal muscle, specifically resulting in sustained skeletal muscle contraction when exposed to a triggering agent. The sustained muscle contraction creates a hypermetabolic phenomenon and that can progress into a life-threatening cascade of events (Figure 1). Without the appropriate treatment or antidote, this progression can affect vital organs leading to end-organ failure and even death. The goal of treatment is to administer the antidote and counteract the physiological processes caused by the hypermetabolism and sustained muscle contraction. The timeliness of symptom recognition and subsequent treatment are factors that may decrease morbidity and mortality associated with MH. Larach, Gronert, Allen, Brandom, & Lehman (2010) report that the time between the first presenting sign and antidote administration is important, and any delay increases the likelihood of complications. Therefore, recognizing signs specific to MH and having the knowledge and availability of the antidote are details fundamental to the successful treatment of MH.

MH was first described in a letter to *The Lancet* in 1962 by Michael Denborough, an anesthetist at the Royal Melbourne Hospital, Victoria, Australia (Denborough, Forster, Lovell, Maplestone, & Villiers, 1962). He and his colleagues were presented a patient with a family history of 10 deaths while undergoing an anesthetic. Ether, identified as a

Malignant Hyperthermia ASNA ID # 2-0.1934 has been approved for .95 contact hours, and is valid through August 30, 2019. This continuing education activity was approved by the Alabama State Nurses Association Approval Committee, an accredited approver by the American Nurses Credentialing Center's Commission on Accreditation.
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http://dx.doi.org/10.1016/j.jradnu.2017.07.005
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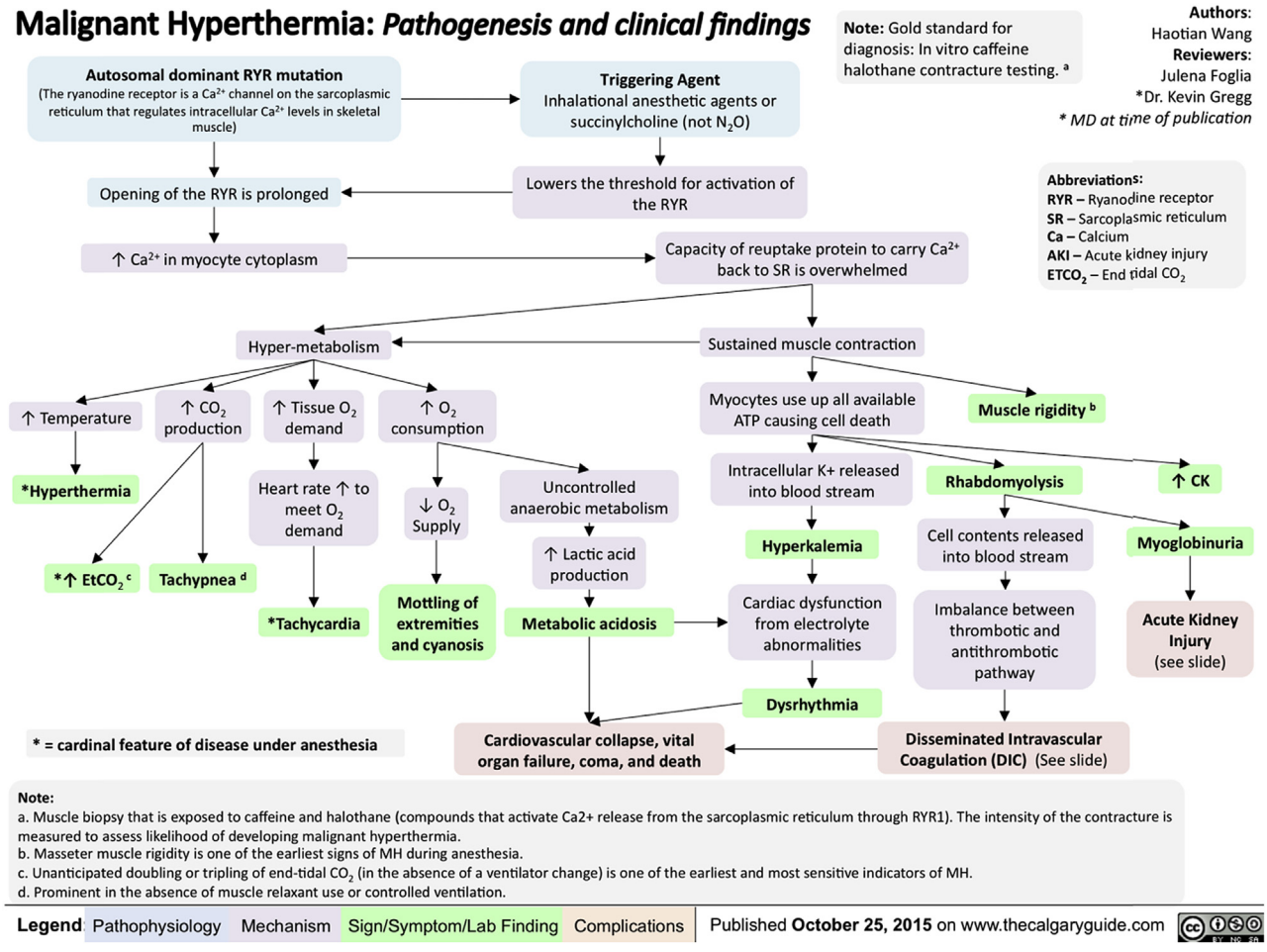

Figure 1. Pathogenesis. Reprinted with permission from the Calgary Guide.

probable causative agent, was therefore avoided, and halothane, a newer halogenated inhalational agent, was administered instead. Despite this alteration, the patient decompensated with the onset of the anesthetic. The presenting signs described by the authors were hypotension, tachycardia, and hyperthermia. The patient recovered with medical support and survived what we now recognize as MH, which before 1970 carried a mortality rate of approximately 70% to 80% (Krause, Fiege, Weisshorn, & Wappler, 2004). It is now understood that all halogenated agents, derivatives of ether, and the muscle relaxant succinylcholine are triggering agents for MH.

MH is an autosomal dominant disorder in humans, which explains the familial pattern seen with Dr. Denborough's case reported in *The Lancet*. The incidence of MH ranges from 1:5,000 to 1:100,000 anesthetics and affects every ethnic group worldwide (Rosenberg, 2010). Although considered a rare occurrence, the prevalence of MH susceptibility may be as high as 1:2,000 persons (Watson & Brandom, 2015). This gap between the genetic prevalence and MH incidence may be attributed to those susceptible not expressing the disease until after more than two anesthetic exposures or by having no anesthetic exposure at all. Data taken from the North American MH Registry from 1987 to 2006 identified 286 very likely or almost certainly MH cases (Larach et al., 2010). A subanalysis of these data found that 77 of 152 (50.7%) reporting past anesthetics had two or more uneventful past anesthetic exposures. This highlights the unpredictability of MH when exposed to triggering agents and the importance of avoiding those triggers regardless of past anesthetic history.

The susceptibility to MH is caused by mutations found on several genes with *RYR1* recognized as the most common. Although genetic testing is available, it is not as reliable as the caffeine-halogenated contracture test, which entails procuring a thigh muscle sample from the person in question. The muscle is exposed to triggering agents and provides the diagnosis of MH if positive. This method provides higher sensitivity (100%) and specificity rates (80%) and therefore remains the gold standard for MH susceptibility diagnosis (Rosenberg, 2010). However, it should be limited to those

Table 1
Triggering/nontriggering agents

Triggers	Nontriggers (safe to use)
Halogenated ethers	Nitrous oxide
Enflurane (Ethrane)	All benzodiazepines
Halothane (Fluothane)	Nondepolarizing muscle relaxants
Isoflurane (Forane)	Atracurium (Tracrium)
Sevoflurane (Ultiva)	Cis-Atracurium (Nimbex)
Desflurane (Suprane)	Rocuronium (Zemuron)
Depolarizing muscle relaxant	Vecuronium (Norcuron)
Succinylcholine (Anectine)	Pancuronium (Pavulon)
Heat-inducing exercise	Doxacurium (Nuromax)
	Intravenous anesthetics
	Propofol (Diprivan)
	Etomidate (Amidate)
	Dexmedetomidine (Precedex)
	Sodium pentothal (Thiopental)
	Ketamine (Ketalar)