GLOBAL SCIENCE LIBRARY

CASE REPORT

Archives of Clinical Research & Trials

Anesthesia Induced Rhabdomyolysis After Lymph Node Biopsy: A Case Report

Christa Milici[1], Joanna Sesti[1], Jaimie Bell[1], Amber Turner[2] and Subroto Paul[1*]

[1]Thoracic Surgery, RWJBarnabas Health, USA

[2]Department of Surgery, RWJBarnabas Health, USA

Abstract

We present a case of a 48 year old Caucasian male with mediastinal lymphadenopathy of unclear etiology who underwent a fiberoptic bronchoscopy with biopsy under general anaesthesia using succinylcholine. He was discharged home the same day in stable condition. The next morning he presented to his local emergency room with muscle aches and pain, as well as tea colored urine. His creatine kinase level was found to be severely elevated suggestive of rhabdomyolysis. Genetic testing confirmed a mutation in the RYR1 gene; however, he did not meet criteria for malignant hyperthermia, giving him a diagnosis of anaesthesia induced rhabdomyolysis (AIR).

Keywords: *rhabdomyolysis; RYR1 gene; ryanodine receptor 1-related myopathy*

Abbreviations: *AIR: Anaesthesia Induced Rhabdomyolysis; CK: Creatinine Kinase; CT: Computer Aided Tomography; MH: Malignant Hyperthermia; U/L: Units Per Liter*

Introduction

Anaesthesia induced rhabdomyolysis is a rare complication of general anesthesia. The signs and symptoms of AIR may resemble malignant hyperthermia; however, it is not associated with hypermetabolism so it is important to be able to distinguish between the two. An initial diagnosis is made based on clinical signs and symptoms. Early treatment is essential to prevent end organ failure. With prompt recognition and treatment, patients are able to make an excellent recovery. Written consent was obtained from the patient for publication in accordance with HIPAA regulations.

Report

A 48 year old Caucasian male with a past medical history of antiphospholipid syndrome presented to the emergency department with abdominal pain. A computer aided tomography scan (CT scan) of the abdomen and pelvis was obtained, which showed acute diverticulitis of the sigmoid colon, and incidentally, noted mediastinal lymphadenopathy with small, bilateral lung nodules. A CT scan of the chest was then performed that confirmed the presence of bilateral sub-centimeter lung nodules, the largest 6 mm, and diffuse mediastinal adenopathy. A diagnostic, fiberoptic bronchoscopy with endobronchial ultrasound was scheduled.

The patient was placed under general anesthesia using succinylcholine and the inhalation anesthetic, sevoflurane. The procedure was uncomplicated, the patient was extubated, and transferred to the post anesthesia care unit to recover. His vital signs at that time were stable. A chest X-ray was performed which revealed no acute cardiopulmonary process, and the patient was discharged home. The final cytology report revealed lymphocytes and ill-defined granulomas; it was negative for malignant cells. Bacterial, fungal, and acid-fast bacilli cultures were negative.

The following morning the patient developed muscle aches. He had also noted that after the procedure his urine was “tea colored.” He presented to his local emergency room where a creatinine kinase (CK) level was drawn and found to be elevated to 34,232 U/L. Physical exam revealed globally depressed deep tendon reflexes. He was afebrile, pulse was 82 beats per minute and regular, blood pressure was 118/67 mmHg, and his respiratory rate was 15 breaths per minute. His past medical history was significant for antiphospholipid syndrome diagnosed 11 years prior to his procedure after experiencing symptoms of blurry vision and muscle cramping. Workup at that time revealed an elevated CK to 3,000 U/L with increased transaminases leading to extensive serologies and a muscle biopsy that showed mitochondrial deficiency. His biopsy slides demonstrated no conclusive answer to the etiology of his myopathy. His baseline

CK fluctuated between 1,000-3,000 U/L. Further workup showed he had positive antiphospholipid antibodies. At that point he began to take low dose aspirin every day. Eight years after his diagnosis, he had a left middle cerebral artery stroke. Hypercoagulable workup revealed a positive Lupus anticoagulant. He was felt to have antiphospholipid syndrome with thromboembolism and was started on Coumadin

After admission, he was treated with intravenous hydration until his CK levels returned to baseline, around 3,049 U/L. At this point he was discharged home. In order to determine if he had one of the ryanodine receptor 1-related myopathies, a genetic test was ordered and performed. Testing came back positive for the RYR1 gene variant.

Combined with the clinical diagnosis of myopathy and onset of rhabdomyolysis after being placed under general anesthesia with succinylcholine and sevoflurane, the patient was given a diagnosis of anesthesia induced rhabdomyolysis. Although the patient was found to have a mutation in the RYR1 gene, the patient did not meet criteria for malignant hyperthermia.

Discussion

Anesthesia induced rhabdomyolysis (AIR) is a syndrome that involves skeletal muscle breakdown which results in the release of myoglobin, elevated serum creatinine kinase, and potentially life threatening hyperkalemia after exposure to succinylcholine or other volatile anesthetic agents [1]. Clinically, patients may remain asymptomatic or can have a life threatening event. Acutely, AIR can lead to hyperkalemic cardiac arrest. Subacutely, AIR can lead to delayed myoglobinuria and elevated serum CK [1]. It can lead to systematic complications if it is not diagnosed and treated in a timely fashion. This is more likely to occur in patients with muscular dystrophy [1].

Malignant hyperthermia (MH) is an autosomal dominant inherited disease which usually results in the response of volatile inhalational anesthetics and/or succinylcholine (a muscle relaxant) [2]. If given these drugs, patients at risk for malignant hyperthermia may develop symptoms such as muscle rigidity, rhabdomyolysis, high fever, acidosis, and tachycardia [3]. Patients who are at increased risk for this disorder are usually unaware that they are susceptible to malignant hyperthermia until they undergo a procedure under general anesthesia. AIR may resemble MH, however AIR is not associated with hypermetabolism [1].

There are specific proteins within the dystrophin-glycoprotein complex which are responsible for muscle membrane stability. These are abnormal in patients with certain myopathies [4]. The mechanism of calcium release from storage in the sarcoplasmic reticulum in the skeletal muscle is abnormally accelerated. Dowling et al found that in a response to certain signals, the RYR1 channel releases calcium ions from the sarcoplasmic reticulum into the surrounding cell fluid. The increase in the calcium ion concentration stimulates the muscle fibers in the body to contract which allows it to move. Any kind of inhalational anesthetic or depolarizing muscle relaxant can act as a trigger for this mechanism to occur [2]. This is because mutations in the RYR1 or CACNA1S gene cause the RYR1 channel to either open more easily or slowly depending on the response to certain drugs. This causes more calcium ions than normal to be released into muscle cells, causing the muscles to contract abnormally leading to muscle rigidity in these patients [1]. This also generates heat and acid so these patients are generally found to have an increased body temperature and acidosis on presentation [4].

AIR is a clinical diagnosis once other causes such as malignant hyperthermia, muscle trauma, mitochondrial disorders, and numerous metabolic myopathies are ruled out [1]. Malignant hyperthermia is initially given as a clinical diagnosis and is later confirmed with a muscle biopsy or genetic testing. Gupta and Hopkins found that recent advances in DNA sequencing technology can confirm MH susceptibility with a sensitivity of 40% which avoids the need for a muscle biopsy. MH is rare and develops at a frequency of 1 in 5,000 to 50,000 general anesthesia cases [5]. There have been six forms of malignant hyperthermia susceptibility that were found, with mutations in the RYR1 gene being the most common [4].

A grading scale is used to make an initial clinical diagnosis of malignant hyperthermia and if a patient grades higher than 50 points on the scale, a clinical diagnosis of malignant hyperthermia can be made. This clinical grading scale includes muscle rigidity, myonecrosis, respiratory acidosis, temperature increase, and cardiac involvement. The patient in this case study graded 40 points on this scale due to his generalized rigidity, CK>20,000 U/L after succinylcholine administration, and tea colored urine [2]. While he did have an RYR1 mutation, the clinical features of this patient gave him a diagnosis of AIR. The patient did not grade high enough to clinically make a diagnosis of MH nor did he experience hypermetabolism.

AIR can occur at any time and the treatment priority includes stopping the halogenated agent, correcting hyperkalemia, volume resuscitation, and diuresis [6]. Without prompt treatment, the complications of AIR can be life threatening due to end organ failure [1]. Patel and Pisklakov found the most commonly observed complications to be acute tubular necrosis, disseminated intravascular coagulation, and compression palsies. For this reason, early diagnosis and treatment is critical.

Author Contributions

Christa Milici: This author wrote the manuscript
Joanna Sesti: This author helped write and edit the manuscript
Jaimie Bell: This author helped write and edit the manuscript
Amber Turner: This author helped write and edit the manuscript
Subroto Paul: This author was involved in the original case and helped write and edit the manuscript.

References

1. Patel A, Pisklakov S. Anesthesia Induced Rhabdomyolysis During Corrective Spine Surgery: A Case Report. *British Journal of Medicine and Medical Research*. 2013; 4: 1302-1307.
2. Badoo S, Igbal A, Nageeb R. A Case Report of Suspected Malignant Hyperthermia Where Patient Survived the Episode. *Saudi Journal of Anesthesia*. 2017; 2: 232-235.
3. Brislin R, Theroux M. Core Myopathies and Malignant Hyperthermia Susceptibility: A Review. *Pediatric Anesthesia*. 2013; 1: 834-841.
4. Litman RS, Griggs SM, Dowling JJ, Riazi S. Malignant Hyperthermia Susceptibility and Related Diseases. *Anesthesiology*. 2018; 1: 159-167.
5. Rosenberg H, Pollock N, Schiemann A, Bulger T, Stowell K. Malignant Hyperthermia: A Review. *Orphanet Journal of Rare Diseases*. 2015; 10: 93.

6. Gupta P, Hopkins P. Diagnosis and Management of Malignant Hyperthermia. *BJA Education*. 2017; 7: 249-254.

7. Lawal T, Meilleur K, Todd, J. Ryanodine Receptor 1-Related Myopathies: Diagnostic and Therapeutic Approaches. *Neurotherapeutics*. 2018; 15: 885-899.

Correspondence: Subroto Paul, MD, MPH, Thoracic Surgery, RWJBarnabas Health, 101 Old Short Hills Road Ste. 302, West Orange, NJ 07052, USA, Tel: 973-322-6757, Fax: 973-322-6752, E-mail: Subroto.Paul@rwjbh.org

Rec: Jun 20, 2020; Acc: Jul 10, 2020; Pub: Jul 13, 2020

Arch Clin Res Trials. 2020;1(1):101
DOI: gsl.acrt.2020.000101