Case Report

Anesthesiology considerations during an orthopedic surgery in a child with Friedreich's Ataxia: A Case Report

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ABSTRACT

Anesthesiology considerations during an orthopedic surgery in a child with Friedreich's Ataxia: A Case Report.

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Friedreich's ataxia is an autosomal recessive inherited neurodegenerative ataxic condition that is often accompanied with multiple comorbidities like scoliosis, cardiac myopathies, diabetes mellitus, abnormal reflexes, dysarthria and it is a challenging task for the anesthesia providers in regards to the pharmacologic, physiologic, and pathologic effects that would occur with this disorder. We present a case report of a 10-year-old male child who was scheduled for a Galeazzi fracture surgery.

Key words: Friedreich's ataxia, pediatric patient, anesthesia.

INTRODUCTION

Friedreich's ataxia is the most common autosomal recessive ataxia where both the mother and father are carriers of the mutated gene. This disorder owns its name to Nikolaus Friedreich, a German pathologist, who was the first person to detail the characteristics of FRDA in 1863[1]. The primary pathology involves degeneration of the dorsal root ganglia, posterior columns, corticospinal, ventral, and lateral spinocerebellar tracts, and the dentate nuclei of the cerebellum.

The characteristics that are found to be consistent in all FRDA diagnoses are ataxia of the extremities, gait unbalance, absence of lower

extremity reflexes, and dysarthria. The most common co-morbidities for the FRDA patients are cardiomyopathies, such as concentric cardiac hypertrophy and supraventricular arrhythmias, scoliosis, diabetes mellitus, dysphagia, nystagmus, optic neuropathy, reduced proprioception, and cognitive deficits[2].

Surgery in patients with FRDA may be required for associated problems (e.g., pes cavus or kyphoscoliosis) or for problems unrelated to the neurological disease. As it mentioned earlier the multisystem effects of FRDA can make the delivery of anesthesia a challenging task to the anesthesiologists regarding to the pharmacologic, physiologic, and pathologic effects that occur with this disorder. Specific concerns to anesthesia include cardiac abnormalities, restrictive lung disease in patients with kyphoscoliosis, neurodegenerative effects, and endocrine dysfunction.

In this case report, we describe the preoperative evaluation and anesthetic management of a child with FRDA scheduled for a Galeazzi fracture (fracture of the distal third of the radius with dislocation of the distal radioulnar joint). surgery on the left hand with general anesthesia.

CASE REPORT

A 10-year-old male (weight 36 kg, height 135 cm) with known Friedreich' s ataxia, was referred for an anesthetic evaluation to be scheduled for a Galeazzi fracture surgery to the left hand with general anesthesia.

A thorough preoperative history and assessment was obtained. According to his parents he was born at the end of the 38th week after a normal pregnancy. At the age of seven years old the young patient begun to have symptoms like incoordination of hands, arms, and legs, slurring of speech, difficulty with writing and eating, slow eye movements and problems with balance. Finally, the FRDA was confirmed at this boy at the age of eight years old, with the method of long PCR and TP-PCR. It was found a defect in the trinucleotide GAA in the frataxin (FXN) on chromosome 9q13. The two allelic genes of frataxin had 850(+/-50) and 1050(+/-50) GAA repeats, respectively.

At the same time, the echocardiogram (2D and Color Echo) showed concentric left ventricular hypertrophy and preserved left ventricle ejection fraction (EF=65%).

After the diagnosis of FRDA the young patient was at a program with physiotherapy (twice a week), occupational and speech therapy (three times a week). He was not taking any drugs. There was not a history of previous surgery or allergic reactions but only G-6PD deficiency.

Presently, the patient was at a stable condition without a clinical decompensation and according to his parents his exercise tolerance was good. His last cardiac evaluation 2 months ago revealed the known concentric left ventricular hypertrophy with IVSd (Interventricular septum thickness at end-diastole) 11-11,5mm and LVIDd (Left ventricular internal dimension at

end diastole) 41mm and preserved left ventricle ejection fraction (EF=65%). On pediatric examination pulse rate was 94 bpm, blood pressure was recorded to be 110/64 mmHg and SpO2 96% in room air. On auscultation of the heart a systolic murmur 1/6 was heard at the aortic area. On auscultation of the lungs the respiratory sound was normal. The chest x-ray was normal and there were not any scoliotic changes of the spine. From his preoperative investigations the hemoglobin level, the serum electrolytes, creatinine, glucose, aspartate transaminase (serum glutamic oxaloacetic transaminase), alanine transaminase (serum glutamic pyruvate transaminase), and alkaline phosphatase were within the normal limits. The routine coagulation tests (PT, INR and aPTT), as the number of his platelets were normal. The patient was classified as an American Society of Anesthesiologist class II.

On patient arrival in the operating room non-invasive monitoring was established (ECG, SPO2, NIBP) and patient were monitored continuously during the procedure. It is mentioned that the child was very cooperative although we have not advised any premedication before the surgical procedure and a peripheral venous access with a 20G cannula was secured from the pediatric ward. His blood pressure was 100/55 mmHg, his heart rate was 84 beat/min, and oxygen saturation was 98% in the room air. Before induction to anesthesia oxygen at 5L/min was given via a simple face mask. The patient was given 1mg midazolam intravenously. Induction of anesthesia was performed in the supine position by using propofol (total dose of 100mg), lidocaine 50mg and fentanyl 50μg. To facilitate tracheal intubation 20mg rocuronium was administered.

The patient was intubated with a 6.0 mm internal diameter cuffed oral endotracheal tube following a Cormack Lehane grade I view on laryngoscopy. The position of the tracheal tube was confirmed by auscultation of the lungs and capnography as usually. The patient was ventilated with an air/oxygen mixture (FiO2 40%), tidal volume 320 (9ml/Kg), respiratory rate 20 breaths/min and a positive end-expiratory pressure (PEEP) of 3 cm H_2O. During and after induction, the patient remained haemodynamically stable throughout the procedure. A second peripheral venous 20G cannula was placed at the right dorsal foot because we wanted to maintain anesthesia with continuous infusion of propofol 2% (6mg/Kg/h) and remifentanil (4μg/Kg/h). A low dose of sevoflurane (0,3 MAC) it was also used. In addition, an upper air warming blanket was applied to preserve normal body temperature.

End-tidal carbon dioxide tension was maintained at 35-37 mmHg. During the perioperative period, his heart rate ranged from 78-85 beats/min and his systolic and diastolic blood pressure varied from 100 to 109 mmHg and from 55 to 60 mmHg, respectively. The oxygen saturation was maintained at 99-100%. The

surgery lasted 80 minutes approximately and there was not any blood loss because a tourniquet was established at the left arm for 85 minutes. We administered 300ml of a solution with a 1 to 4 sodium to glucose ratio (0.18% NaCl + 4.3 % Dextrose) intravenously during the procedure. The patient's blood glucose during surgery was 174mg/dL.

In the operating room prior to surgical incision were given also antibiotics in dosage prescribed by the pediatrics (cefuroxime 1,5gr and amikacin 750mg IV), and 3mg dexamethasone and 3,5mg ondansetron as antiemetics. During closing of the surgical site, the remifentanil and propofol infusions were discontinued. For postoperative pain management, we administered paracetamol 500mg IV as a loading dose and 1,5mg morphine 5min before the end of delivering remifentanyl. The residual neuromuscular blockade was not monitored but because it was almost two hours ago from the induction of anesthesia, we reversed it with sugammadex 80mg I.V (2mg/KG) at the end of the surgery and the patient recovered consciousness 5 minutes later.

After extubation, the patient was removed to PACU (post anesthetic care unit) for continuous hemodynamic and respiratory monitoring for 60 minutes. During this period, the heart rate, the blood pressure, and respiratory frequency were maintained within normal limits. The rest of the postoperative period in the ward was uneventful. Patient was discharged from hospital 24 hours after the surgery.

DISCUSSION

The majority of FRDA cases are caused by a defect in the trinucleotide GAA (guanine, adenine, adenine trinucleotide) in the frataxin (FXN) gene found on chromosome 9[3]. In fact, there are two types of FRDA, a homozygous GAA trinucleotide repeat expansion in the first intron of the FXN gene on the long arm of chromosome 9q21.11 which is the most predominant form (96-98%) and the second type is heterozygous, characterized by a point mutation or exonic deletionbeing the least predominant form (2-4%)[4,5]. Frataxin is a mitochondrial protein and has a role in iron homeostasis and antioxidation. The mutation leads to reduced levels of frataxin, with subsequent accumulation of iron and impaired electron transport in the respiratory chain in the mitochondria, oxidative stress, and decline in mitochondrial adenosine triphosphate (ATP) production.[1,2,5,6] The resulting impairment in mitochondrial function causes pathology in the peripheral and central nervous system, the heart myocardial fibers and the pancreatic islets of Langerhans.

FRDA occurs primarily in the Caucasian population with an incidence between 1:29,000 to 1:50,000 in different countries (in northern Spain has been estimated 1 in 20,000, in Ireland 1 in 23,000, in France 1 in 43,000, and in Germany 1 in 47,000)[7]. FRDA presents equally in

males and females between the ages of 10 to 15, however, later onset can be seen after the age of 25[5,8]. The typical presentation of FRDA includes varying degrees of ataxia in all four limbs usually first present in the first or second decade of life that progresses to truncal ataxia and paraplegia, eventually causing in most patients to be wheelchair dependent by the third decade of life, absent lower extremity reflexes, pyramidal signs, and dysarthria. Most patients have an abnormal electrocardiogram due to hypertrophic cardiomyopathy with the degree of left ventricular wall thickness to be associated with the length of the repeat triplet expansion in the FXN gene. In most cases, left ventricular concentric remodeling occurs in the beginning of the disease process, with subsequent concentric hypertrophy developing in advanced stages of the disease, and a small percent of patients develop eccentric hypertrophy. Systolic function is not impaired in most FRDA patients, and a reduction in ejection fraction is typically seen when end stage cardiomyopathy develops[9]. Common ECG findings are T-wave abnormalities such as flattening or inversion of the waves often seen in the inferior and lateral leads, bundle branch blocks, atrioventricular conduction blocks, atrial, and ventricular arrhythmias. More infrequently, atrial flutter or fibrillation can be present, and to an even lesser degree ventricular arrhythmia.[10] Typical echocardiogram characteristics include concentric left ventricular hypertrophy, preserved left ventricle ejection fraction, decreased peak longitudinal strain, and sparkling texture[9,11].

From the musculoskeletal manifestations the most common are scoliosis with a prevalence rate of 63 to 100% and cavo varus foot deformities which progress with age. Dysphagia is another common problem in these patients related to delayed pharyngeal swallowing reflex and lingual dysfunction. Aspiration, as well as the risk of silent aspiration is prevalent, and 10% of deaths in individuals with FRDA is caused by pneumonia[12].Other signs are optic atrophy, deafness, diabetes mellitus or glucose intolerance.

Diagnosis of FRDA usually begins with a thorough history and examination of the patient and based on clinical presentation (“triad of ataxia, areflexia, and positive Babinski reflex”). Genetic molecular analysis tests confirm the diagnosis.

Unfortunately, there is no specific treatment for FRDA at this time and this disorder is progressive and associated with a decreased life expectancy of 35 to 40 years[5]. The young patients need holistic care which should include physiotherapy, occupational, and speech therapy, as well as palliative care, many medical specialists like neurology, cardiology, orthopedics, geneticist, psychiatry, dietetics, ophthalmology, audiology, and endocrinology and clinical monitoring of neurological, musculoskeletal, cardiac, pulmonary, endocrine, visual, auditory, and behavioral. The main causes of mortality in

FRDA are related to cardiac dysfunction with congestive heart failure and arrhythmias being the leading causes, followed by stroke, ischemic heart disease, and pneumonia[5].

Frequently these patients must undergo to various surgical interventions related to their disease as spinal, orthopedic, and cardiac procedures. Spinal surgeries include corrective surgery for scoliosis when there is scoliotic curvature greater than 60 degrees and based mainly on severity of associated symptoms[15]. Orthopedic procedures usually include correction of cavo varus foot deformities with tenotomies or primary triple arthrodesis[3]. The most common cardiac operations include the insertion of prophylactic automated implantable cardioverter-defibrillators for treatment of ventricular tachyarrhythmias and rarely heart transplantation for individuals with life threatening cardiomyopathies.[16] And of course, all the patients with FRDA could have to undergo to various other surgeries which are not related to their disease like the general population.

The current recommendations for administration of anesthesia in patients with FRDA is to manage them in a similar way with patients diagnosed with amyotrophic lateral sclerosis (ALS)[13]. Other diseases which are related with mitochondrial dysfunction include Parkinson's disease, Huntington's disease, and Charcot-Marie-Tooth[14]. Recent research is focused on genetics, supportive treatment, medication trials, cardiac dysfunction, cardiac transplant, cerebral studies, prosthetics, and surgical correction of kyphosis[2,5,10].

The main anesthesia considerations for patients with FRDA include:

Before any surgical procedure, a thorough preoperative history and assessment should be completed. FRDA is progressive and its severity varies from patient to patient. So, it is important to evaluate the extent of the disease progress and gather appropriate testing[17]. Baseline vital signs should be collected and recorded. The essential laboratory tests for individuals with neuromuscular disorders should include a basic metabolic panel, creatinine kinase, myoglobin, complete blood count, and type and cross if blood loss is expected to be significant[18]. A neurologic evaluation should be useful to assess both sensory and motor baseline function.

Beyond the chest x-ray for FRDA patients with scoliosis, pulmonary function studies should be assessed additionally, including vital capacity and expiratory volume. When vital capacity is less than 30% of predicted normal value there is increased risk for pulmonary complications and may require respiratory support or prolonged continuous mechanical ventilation postoperatively[3]. The mode of ventilation in the operating room must include settings appropriate for restrictive lung disease processes and avoidance of left ventricular outflow tract obstruction (LVOT), such as decreased tidal volumes and increased respiratory rate[13].

Because hypertrophic cardiomyopathies are prevalent among FRDA patients a 12-lead ECG should be obtained in all patients preoperatively to assess any rhythm abnormalities and a 5 lead to monitor intraoperative cardiac function. Also, a cardiac echocardiogram is indicated to assess the degree of cardiac dysfunction and diagnose other problems such as left ventricular hypertrophy and mitral regurgitation which can be common. Left ventricular ejection fraction is typically preserved, until end stage cardiac disease. For patients with implantable cardioverter-defibrillators we must determine the type of device, the degree of dependence, if a recent interrogation has taken place, and if a magnet will need to be utilized[13].

It is crucial to avoid left ventricular outflow tract (LVOT) obstruction. Preoperatively, replacement of fluid deficit should be considered to replenish intravascular volume and decrease the risk of LVOT obstruction. During induction, sympathetic stimulation should ideally be avoided with direct laryngoscopy. We can blunt this response using as premedication a beta blocker or volatile anesthetic. The choice of inhalation agent is also significant, because desflurane can result in sympathetic nervous system stimulation, causing tachycardia. We can use alpha-adrenergic agonists to treat any deterioration in preload or afterload whereas with beta-adrenergic agonists there is the risk to increase the inotropy and chronotropy action of the heart so they should be avoided. Normal sinus rhythm should be maintained and therefore beta blockers such as esmolol and metoprolol can be considered to reduce tachycardia. Additionally, a cardioverter-defibrillator should be present within the operating room, in the case the patient develops supraventricular tachydysrhythmia[13].

Neuromuscular blocking agents for induction and maintenance should consider the FRDA patient's neurologic deficits. Succinylcholine should be avoided, because the patients with FRDA may be at risk for hyperkalemia as FRDA is a neurodegenerative disease and anesthetic management should be treated in a similar fashion to ALS patients.[13] Furthermore, according to Katz and Murphy[18] , the administration of succinylcholine to patients with muscular dystrophies, motor neuron diseases, and intrinsic muscle disease should be avoided because it is related with an increased risk for malignant hyperthermia, rhabdomyolysis, and cardiac arrest.

Nondepolarizing muscle relaxants can be administered, but a reduced dose should be considered, as patient's response can be variable[17]. We must also monitor the co-administered medications of the patient as corticosteroids, aminoglycosides, vancomycin, quinidine, ester-type local anesthetics, furosemide, calcium channel blockers, and beta-blockers as they can cause a prolonged block[17]. For the reversal of residual neuromuscular blockade from rocuronium sugammadex is safe and Katz and Mur-

phy[18] have reported that sugammadex has safely been used in patients with other types of neuromuscular disorders, such as Duchenne muscular dystrophy, Becker's muscular dystrophy, and myasthenia gravis. Neostigmine can also be used for the reversal of residual blockade from neuromuscular blockers as stated Romero and Joshi [17] in ALS patients.

The selection of volatile anesthetic agents in individuals with FRDA with mitochondrial deficits can pose a risk. The selection of which inhalational agent will be based on the clinical picture and effects of each agent. Patients with mitochondrial deficits may be more sensitive to sevoflurane than other agents, however, if muscle relaxation is needed, sevoflurane or desflurane would be the ideal choice. Isoflurane or desflurane may be advantageous if cardiac output needs to be preserved, though caution should be exercised with use of desflurane, related to the risk of tachycardia. The use of BIS monitoring should be considered to administer the minimum amount of anesthetic needed for the proposed procedure. Finally, inhalation agents may be safer than intravenous sedation, as volatile anesthetics are able to be exhaled and do not require extensive metabolism[19].

The intravenous anesthetic agents can also inhibit mitochondrial function of complex I and specifically etomidate, ketamine, and barbiturates. Propofol and thiopental have been given in bolus doses with minimal negative consequences, however, long-term infusions may have an increased risk of propofol infusion syndrome related to inhibition of mitochondrial function of complex's I, III, and indirectly complex II[19]. Furthermore, there are several case reports in which propofol for induction and maintenance of anesthesia has been used without adverse effects. The opioids do not interfere with mitochondrial function; however, the use of morphine theoretically may cause some degree of mitochondrial dysfunction and has to be used with caution[19].

The selection of intravenous fluids should be patient and procedure specific. According to Romero & Joshi[17], the administration of lactate fluids should be omitted in patients with mitochondrial deficits. Hypovolemia, from fluid deficit and blood loss, should be avoided to decrease the risk of LVOT obstruction[13]. When is anticipated significant intraoperative blood loss, that should be managed with various approaches. Antifibrinolytics (tranexamic acid) can be considered to reduce bleeding. Also, blood salvaging systems, such as a cell saver, can help allowing the autologous blood to be returned to the patient. It is crucial to prevent hypothermia with administering warm fluids and a forced air warming device, to reduce bleeding and coagulation dysfunction[20].

Local anesthesia is an option in FRDA patients; however, it may present a challenge related to the presence of scoliotic changes. Respiratory status should be monitored in neuraxial blocks higher than the level of T10, as respiratory

muscle involvement may become compromised [13]. There are several successful case reports with spinal and combined spinal/epidural anesthesia in FRDA patients.

Retrospectively, our case was planned and managed according to the available literature on FRDA patients. Appropriate cardiac studies, thoracic x-ray, and labs were obtained preoperatively. We avoided the use of a depolarizing muscle relaxant and a reduced amount of a non-depolarizing agent, rocuronium, was administered. The patient remained hemodynamically stable during the procedure; we had no blood loss because a tourniquet was established at the left arm for 80 minutes. We administered 300ml of a solution with a 1 to 4 sodium to glucose ratio intravenously during the procedure. We had chosen this intravenous solution because it was important to avoid the hypoglycemia, which is a risk in young patients with FRDA. The patient's blood glucose during surgery was 174mg/dL. We reversed the possibility of residual neuromuscular blockade with sugammadex 80mg I.V (2mg/KG) at the end of the surgery but it was a failure of us the absence of a nerve stimulator. For postoperative pain control, we administered paracetamol 500mg IV as a loading dose and 1,5mg morphine 5min before the end of delivering remifentanyl. He was extubated 5 min after the sugammadex and his postoperative status was uneventful. The addition of BIS monitoring should have been considered to titrate and guide anesthetics agents in this population.

CONCLUSION

Although FRDA is a rare disorder necessitates understanding of the disease process and the multisystem effects that can be compromised under anesthesia. It is particularly important in patients with FRDA to have a detailed preoperative assessment and planning of anesthesia prior to any surgical procedure, as the perioperative period can pose considerable risks. Various approaches to anesthesia techniques have been demonstrated successfully through case reports. It is preferable to choose regional and neuraxial anesthesia techniques in many surgeries to reduce pulmonary and cardiac compromise. When general anesthesia is necessary in this population the selection of medications must be vigilant as monitoring for the proposed procedure.

Postoperative derangements and complications may develop from respiratory, cardiac, endocrine, neurologic, and musculoskeletal system so it is crucial to take place a collaborative approach between the care team and providers in the preoperative, perioperative, and postoperative period to maximize optimal surgical and patient outcomes. Comprehension of potential complications and management of this population improves patient safety and outcomes.

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Consent for publication

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Competing interests

The authors declare that they have no competing interests.

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