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Role of Anesthesiologist in An Unanticipated Anesthetic Challenge in A Day Care Procedure

KEYWORDS

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ABSTRACT ***Introduction:*** *Mitochondrial respiratory chain disorder account for significant and varied presentations in paediatric practice. There are many metabolic stressors that can lead to de-compensation in patients with mitochondrial disease. Mitochondrial cytopathy is caused by defect in electron transport chain, causing inability to synthesize ATP.*

MATERIAL AND METHODS: *This case report is of 10 year old male posted for PEG who underwent upper GI scopy under local anaesthesia who have been suspected to have mitochondrial cytopathy with status dystonicus based on MRI brain findings. He was given 2 puffs of 2% lignocaine for upper GI scopy before procedure after which he became drowsy and started to desaturate. According to emergency protocol he was mask ventilated with 100% oxygen with JRC circuit and procedure was completed with 2% sevoflurane and DNS as maintenance fluid in 30 min. The procedure went uneventful and recovered completely from anaesthesia.*

RESULTS AND DISCUSSION: *Intra-operative and postoperative events were uneventful. Analgesic such as lignocaine can affect electron transport chain and affect the production of ATP in these kind of patient. So it is better to avoid the usage of lignocaine as analgesic in patients with mitochondrial cytopathy.*

INTRODUCTION:

A mitochondrial disease was described in 1962, when Luft and colleagues reported a case of a 35-year-old euthyroid woman with myopathy, excessive perspiration, heat intolerance, polydipsia with polyuria, and a basal metabolic rate 180% of normal.[1] Approximately 1.5 billion years ago the aerobic mitochondria took up residence inside the anaerobic ancestor of the modern eukaryotic cell, and although most of the mitochondrial genes migrated to the nucleus eons ago, 37 of them—some of which encode absolutely vital functions—still reside within the mitochondria themselves.[2] Mitochondrial respiratory chain disorder account for significant andvaried presentations in paediatric practice. There are many metabolic stressors that can lead to de-compensation in patients with mitochondrial disease. Mitochondrial cytopathy is caused by defect in electron transport chain, causing inability to synthesize ATP.

CASE REPORT :10 year old male patient who was having complains of abnormal posturing of limbs, asymmetrical in onset with more right in upper limb, patient also had slurring of speech and delayed milestones for 6years of duration. On CNS examination there was no eye to eye contact. He was looking around with posturing of dysmorphic face and he also had poor auditory tracking with contracture of all four limbs. On the basis of MRI finding such as symmetrical signal changes in lentiform nucleus, left caudate nucleus and inferior olivary nucleus, he was diagnosed with probable mitochondrial cytopathy. The Patient was posted for percutaneous entero-gastrostomy (PEG) and he was shifted to endoscopy room as surgeon wanted to do an upper gastro scopy . The surgeon used 2% lignocaine 2 puff as a local anaesthetic for the procedure, and patient suddenly appeared drowsy ,with fall in saturation. On call anaesthetist was called for help. The patient was given 100% o2 with JRC circuit after which saturation improved and he was sedated with 2% sevoflurane with DNS as maintenance fluid. Procedure took almost 30 minutes. Post procedure patient was stable.

DISCUSSION:MELAS was first described by Pavlakis et al[3] in 1984. It is characterized by normal early development, followed by onset of exercise intolerance, stroke like episodes, seizures and dementia. In a large series reviewed by Hirano et al,[4] all patients became symptomatic before the age of 40 years and had evidence of lactic acidosis. The skeletal muscle biopsy revealed RRF. KSS is characterized by progressive external ophthalmoplegia, cardiac conduction block, pigmentary retinal degeneration, dementia, CSF protein > 100 mg/dl and variable number of red ragged fibres on muscle biopsy. Other features include deafness, ataxia, episodic coma and endocrinal abnormalities.[5] Movement disorders have been described due to mitochondrial abnormalities.[6,7] Dystonia has been the most frequent movement disorder[7] besides myoclonus, chorea, athetosis, and tremors. Treatment options for patients of mitochondrial cytopathy are limited. Dietary manipulations including high carbohydrate diet have been recommended to compensate for impaired gluconeogenesis and to decrease lipolysis. The patients should avoid extremes of temperature and drugs like phenytoin and barbiturates.[8] Anecdotal success has been reported with a number of vitamins and co-factors (thiamin, riboflavin, vitamin C and K, carnitine) and glucocorticoids.[9,10] Coenzyme Q10 has been found to be consistently beneficial in some studies.[11,12] Inhalational agents such as halothane, isoflurane, sevoflurane Inhibit complex 1 activity, this effect is less marked in vitro with sevoflurane than with halothane or isoflurane Sevoflurane and isoflurane have been reported in many cases with successful outcomes .[13] Propofol also has been used successfully in some patients with mitochondrial disease for induction.[13]

CONCLUSION:

Even though we couldn't find any article on lignocaine causing respiratory depression ,we would recommend not to use any kind of local anaesthetics in a patient with mitochondrial cytopathy.

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