

## Case Report

# Emergence delirium – liberation with water!

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**Abstract**

Emergence agitation (EA) is a common, lesser spoken post anaesthetic complication which is considered as a normal course of anaesthesia by many. It can traumatise the parent and health care staff. An agitated aggressive child can cause harm to self and his surroundings. Its' cause, prevention and management has been a mystery for more than forty years now. There are no guidelines put forth for treatment of such a common complication (1). Incidence of EA increases with pre-operative anxiety, use of sevoflurane for maintenance, hypoglycaemia, dehydration, Otorhinolaryngology and ophthalmic procedures. The occurrence of maladaptive behaviour and its implications in the long run after an episode of EA are not known though some speculations for behavioural changes have been made. Research has shown premedication and Total Intravenous Anaesthesia can reduce its occurrence (1-2). We write this article in order to share our experienced of the same in a 11-year old child who was operated for adenoidectomy, myringotomy and grommet insertion.

**ABBREVIATIONS**

**EA:** emergence agitation, **TIVA:** total intravenous anaesthesia, **PAED:** Pediatric anaesthesia emergence delirium scale

**INTRODUCTION**

Emergence agitation was described as early as 1960 by Eckenhoff and colleagues. Defined as a temporary dissociated state of consciousness after discontinuation of anaesthesia, disturbance in awareness or attention to ones environment with perceptual alterations including hypersensitivity to stimuli and hyperactive motor behaviour, often presents with irritability, inconsolable crying, distress and inability to cooperate (1-2). Incidence of EA reported to range from 2% to 80%, yet a challenge to accurately identify patients who are at risk. Although the episode lasts for a short duration, there have been incidences where the effects may linger beyond the recovery period and children exhibited postoperative behavioural problems that had not existed beforehand (1-3). The long-term effects of which is still unknown. Hence from our experience we try to identify the risk, cause and put forth steps to prevent EA.

**CASE PRESENTATION**

11-year-old boy was brought to our hospital with complaints of bilateral reduced hearing since childhood and impaired speech. The child also had history of recurrent upper respiratory tract infection and ear pain. Presently child had no complaints of fever, cough, ear discharge. The child was diagnosed to have chronic

adenoiditis with bilateral acute otitis media and was posted for adenoidectomy and bilateral grommet insertion.

A thorough pre-anesthetic history taken and examination was done and the case was accepted under American Society of Anaesthesiologist physical status (ASAPS) II. Conceived from a 2<sup>nd</sup> degree consanguineous marriage, delivered through LSCS in view of hydramnios. Physical growth and development was appropriate for age matched controls but un able to hear. The child was moderately built and nourished weighing 43 kg with a height of 143 cm. General physical examination and systemic examination was normal. The child could comprehend, was cooperative, but had speech impairment. He had no history of previous surgery. Airway examination showed buck teeth, with adequate mouth opening and a Mallampatti Class 1. Routine pre-op blood investigations done were normal. The case was posted for the planned surgery under general anesthesia and controlled ventilation.

Child was kept Nil by mouth as per standard fasting guidelines. He was wheeled in to operation theatre comfortably, without need for any sedation. A 20 G cannula was present in the left hand. IV fluid ½ DNS was connected. Monitors were connected. Premedication was given through the IV line with inj. Fentanyl 90 mcg, Inj Midazolam 1mg and child was preoxygenated. Induced with inj. Propofol 90 mg for which he had severe pain in the injection site. After checking for adequate ventilation inj. Atracurium 30 mg was given. Airway

was secured with 6.5 mm CETT in first attempt under direct laryngoscopy and fixed at 20 cm after confirmation of bilateral equal air entry by 5-point auscultation and square waveform capnogram. Inj dexamethasone 4 mg was given to prevent post-operative vomiting. Anaesthesia was maintained with 50:50 O<sub>2</sub> and air with 1 MAC sevoflurane. Diclofenac suppository of 25 mg was inserted. Adenoidectomy and bilateral myringotomy and grommet insertion was performed over 1 hour and 10 mins. Inj paracetamol 1gm administered as analgesic. Post procedure child was reversed and extubated and shifted to recovery room.

In the recovery room child started complaining of throat pain and irritation. He had one episode of vomiting, inj.Ondansetron 4 mg was given. He refused to keep the oxygen face mask. He started having bouts of cough and demanded for drinking water. Inj Fentanyl 30 mcg iv was administered. He became very agitated, presented with aggressive behavior and was inconsolable, even in the presence of the father. Another 20mcg of inj.Fentanyl was given. He continued to remained agitated, throwing tantrums shouting for water and complaining of throat pain. The child did not allow us to connect monitors. We checked SPO<sub>2</sub> which remained >95% and sugar (GRBS) checked was 112mg/dl. Peripheral pulses were well felt and he was warm. Sips of water was given, saline nebulization and lignocaine gargles were given for which he did not cooperate. The father was asked to comfort the child which also was in vain. Ruling out all the possible causes for agitation post-operatively, we diagnosed emergence agitation with Pediatric Anaesthesia Emergence Delirium Score (PAED) of 11. Considering it as EA, a decision to sedate him was taken. Repeat boluses of Inj.Fentanyl 20 mcg iv and Inj.Midazolam 1 mg iv was given, which failed to calm him. After 40 minutes Inj.Propofol 20 mg iv boluses given thrice to sedate him (total of 60mg). His aggression only grew which worsened on seeing his father. He then wanted to walk and wanted more water. Inj. Haloperidol 0.5mg iv boluses was given twice in the next thirty minutes with no effect. His agitation started within 10 mins after emergence from Anaesthesia and lasted for about 2 hours. We allowed him to drink water as we were running out of options to control his agitation, he drank around 250-300 ml of it to his heart's content and then calmed down to sleep.

He was monitored in recovery after another two hours. His vitals remained stable. He did not show any signs of aggression. He was shifted to ward on room air, conscious and calm again.

## DISCUSSION

EA is a very common occurrence experienced by pediatric anaesthetist in the post-operative care unit where symptoms occur within 30 min of termination of anaesthesia and lasts for 15-30 min or can be persistent and has been reported to continue for up to 2 days, which is very distressing to the health care worker (2). Agitated behaviour in a child can be a manifestation of pain, hypoxia, hypoglycemia, separation anxiety, ect and the perioperative physician must be able to differentiate these causes from ED which is often difficult (2-3).

Its presentation can vary from being delirious to being extremely agitated with kicking, biting, bending their head backwards and with lack of eye contact with the care givers and inconsolable. Pediatric anaesthesia emergence delirium

scale (PAED) is a more validated and commonly used diagnostic tool introduced by Sikich and Lerman in 2004 (2,4) (table 1). Diagnostic and Statistical Manual of Mental Diseases (DMS-IV and V) have defined specific symptoms of EA and have found eyes stared or averted and non-purposeful movement, kicking and inconsolability as independent signs of emergence agitation in children (3,5).

Based on PAED our child score was 11 as his actions were purposeful, he was truly aware of his surroundings, restless and extremely inconsolable. Though it is more common in preschool children our child was 11 years old, but the presence of impaired hearing and speech could have been a contributing factor in our case. EA has been reported more commonly in otorhinolaryngological and ophthalmic surgeries the reason being rapid induction and emergence from anaesthesia. Other reason for its common occurrence in head and neck surgery is the feeling of suffocation (2). Our case was posted for adenoidectomy with bilateral myringotomy and grommet insertion and the procedure lasted for about 1 hour and 20 minutes. Voepel-Lewis et al found both ENT procedures and a rapid emergence from anaesthesia are independent predictors for this complication, however no satisfactory explanation is available the association between ENT surgery and EA (6, 7). Pre-existing behavioural changes like anxiety, temper tantrums, adaptability can increase the risk for EA (1, 4). Though our case seemed relaxed while shifting him to the operation theatre we must have premedicated him as the child had thrown similar tantrums in the ward while securing an IV line which we found out retrospectively. He also had history of exhibiting similar behaviour during one of his previous hospital admissions. Poor quality of previous medical experience also contributes to EA (4, 6).

Anaesthetic factors contributing to EA has been linked with the use of the new volatile agents (sevoflurane and desflurane) with which greater incidence has been reported in comparison to halothane, reason being rapid emergence from anaesthesia (2, 4). As it has been observed even in non-painful procedures like MRI a theory suggesting that the agents themselves that have some neuro-pharmacological stimulus in the immature nervous system has been put forth (2). EA must not be considered as a clinical manifestation of pain, but one cannot exclude the involvement of peri-operative pain in the genesis of this complication. In our case sevoflurane was used for the procedure which lasted for 1 hour 20 mins. Pain was adequately managed intraoperatively. Other factor contributing in our case could have been at pain he felt during induction with IV propofol. Parental presence during induction can be tried when both the child and the parent are

**Table 1:** Pediatric Anaesthesia Emergence Delirium Scale (PAED)

1 - The child makes eye contact with caregiver
2 - The child action is purposeful
3 - The child is aware of his surroundings
4 - The child is restless
5 - The child is inconsolable
1, 2, and 3 are scored: 4 = not at all, 3 = just a little, 2 = quite a bit, 1 = very much, 0 = extremely. 4 and 5 are scored: 0 = not at all, 1 = just a little, 2 = quite a bit, 3 = very much, 4 = extremely.

anxious. Parental anxiety itself being a contributing factor for emergence in a child can be reduced with this. We feel that other methods for induction can be used to prevent the incidence of emergence postoperatively.

Our case had most of the described risk factors (2, 3): male child, ENT surgery, not premedicated, though anxiety was not noted history of such behaviours were there, painful induction, use of sevoflurane and also the hearing deficit. We made a diagnosis of EA after ruling out hypoxemia, pain, hypoglycaemia, hypotension and airway obstruction; using PAED scale score being 11.

Pharmacological intervention is used when the child is inconsolable with high risk for self-injury. Though no clear recommendations have been put forth in such cases pharmacologic intervention seems justifiable when reassurance doesn't help and agitation continues to persist (4, 6). Use of alpha 2 agonist in the peri-operative period has shown to significantly reduce the incidence (2). We tried reassuring the child, parental presence after which we sought the help of pharmacological agents like midazolam, fentanyl, propofol, haloperidol. We found that at times giving in to the child's demands, like in our case allowing him to drink water can help reduce the severity of agitation but always keeping in mind the risk and benefit in doing so.

Postoperative emergence delirium or agitation is a diagnosis of exclusion: any child who emerges from anaesthesia who remains agitated after other pathological causes are ruled out (1). EA still remains unpredictable in incidence and severity, and can take the anaesthetist entirely by surprise. Given the known relationship of peri-operative factors in the incidence of ED, the incidence can be reduced by modifying anaesthesia technique in children with the highest risk either by reducing/avoiding exposure to volatile agents, using TIVA, use of anxiolytics pre-emptive treatments and other advanced techniques to comfort the child (2). Out of the available data the best evidence for treatment is a-2 agonists (2, 7) and propofol as a single dose (8) or as TIVA (9). We found that giving in to the child's demands when no associated risks or weighing the risk and benefit might help calm the child sooner.

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