

Emergence Delirium in a Schizophrenic Patient who Underwent Craniotomy for Elevation of Depressed Skull Fracture under General Anaesthesia: A Case Report

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Abstract

A 21-year-old male patient with schizophrenia was scheduled for craniotomy for elevation of a right temporal region depressed skull fracture. General anaesthesia was induced with i.v sodium thiopentone and endotracheal intubation was facilitated with i.v rocuronium and maintained with sevoflurane and mechanically ventilated with volume controlled ventilation. There were no complications intraoperatively. However, postoperatively in the post anaesthesia care unit, the patient developed emergence delirium which responded to intravenous diazepam.

Schizophrenia is a mental disease characterized by thought disorders, delusions and delusions. It is related to excess dopaminergic activity in the brain. Lifetime risk of getting schizophrenia is 1% and has equal incidence between males and females and a peak incidence in the late teens [7]. Schizophrenia is treated with antipsychotics which are predominantly dopamine receptor antagonists. Use of antipsychotics to manage this condition is associated with impaired biological response to stress such as surgery and anaesthesia therefore increasing the risk of hypotension, arrhythmias, lack of pain sensitivity, abnormal immune system, water intoxication and pituitary-adrenal and autonomic nervous system dysfunction.

Case Description

A 21-year-old male patient presented to the Accident and Emergency (A/E) unit with a one day history of assault to his right temporal region with an unknown object. He had no history of neurological sequelae. He had a computed tomography (CT) scan done which showed a depressed skull fracture at the right temporal region with no cerebral oedema or haemorrhage. He was subsequently booked for an elevation of the depressed skull fracture in four days. He was a known schizophrenic on sulpiride 600 mg twice a day for the past four years. He had no previous anaesthetic history and no known allergies. He had no history of smoking but drank alcohol once a month. He is not a known hypertensive or diabetic patient.

On examination he weighed 72 kg and airway assessment was Mallampati 1. Cardiovascular and respiratory systems were normal. On neurological examination he was calm and well oriented in time, place and person (Glasgow coma scale: 15/15) and no signs of raised intracranial pressure. Mental health assessment revealed no hallucinations, normal insight, his speech and thoughts were coherent and congruent. His full blood count, urea and electrolytes were normal. Informed consent was obtained from the patient. The patient was fasted preoperatively for 6 h with no solid intake, only clear fluids till 2 h preoperatively. He was given oral lorazepam 2 mg as well as the morning dose of sulpiride. The anaesthetic management of

Keywords: Emergence delirium; Craniotomy; General anaesthesia; Post anaesthesia care unit

Introduction

Emergence delirium is an abnormal mental state that develops as a result of anaesthesia administration as the patient moves from been unconscious to complete wakefulness. It has an incidence of 4-31% with more research done in the paediatric population [1]. The pathophysiology is not well understood. Lepouse et al. described increased incidence of emergence delirium in patients given preoperative benzodiazepines, preoperative anxiety, pain, undergoing breast and abdominal surgery, long procedures, use of inhaled anaesthetics as well as use of neuromuscular blockers as risk factors [2]. While patients on chronic antipsychotics drugs were often not agitated postoperatively [2,3]. While Kudoh et al. in 2004 highlighted from their study the increased incidence of postoperative confusion in chronic schizophrenia patients who had their treatment discontinued [4]. Emergence delirium can be managed through a preemptive strategy of non-pharmacological interventions, selecting appropriate anaesthesia, multimodal analgesia regimes, use of propofol, dexmedetomidine, midazolam and dexamethasone [5,6].

the case was discussed with the attending specialist anaesthetist.

In theatre intravenous access was secured with a 20G cannula on the left arm and 0.9% normal saline set up. The pre-use anaesthesia machine check was done and monitoring comprised pulse oximetry, electrocardiography, capnography, gas analysis and non-invasive blood pressure (NIBP). The baseline blood pressure was 130/59, pulse rate 76 and pulse oximetry 99% in room air. General anaesthesia was induced with fentanyl 100 mcg, sodium thiopentone 350 mg, rocuronium 70 mg. The airway was secured with a cuffed endotracheal tube (ETT) 7.5 ID after administration of i.v rocuronium and fixed at 22 cm on the lips. Cormack – Lehane classification was grade 1 and correct placement of the ETT was confirmed by visualizing the ETT pass the vocal cords, continuous capnography tracing and auscultation of the chest. Only a drop in the blood pressure by 25% of the baseline blood pressure was noted after induction which responded to an increase in the intravenous fluid rate. Anaesthesia was maintained by sevoflurane with end tidal sevoflurane 1.8-1.9% and ventilatory mode was volume control with a target end tidal carbon dioxide of 36–38 mmHg. Analgesia comprised of morphine 7 mg and paracetamol 1 g. Intraoperative targets were to not raise intracranial pressure by avoiding hypercarbia by appropriate patient set volume control ventilation, avoiding hypoxia by maintaining an oxygen–air mixture with an FiO_2 of not less than 40%, avoiding hypotension and hypertension by allowing adequate depth of anaesthesia and ensuring adequate analgesia. He underwent surgery in the supine position with his head tilted to the left, and the procedure lasted 70 min. His vital signs remained within 10% of the baseline vitals intraoperatively. There were no complications intraoperatively minimal with blood loss less than 100 ml. Intravenous neostigmine 2.5 mg and glycopyrrolate 0.4 mg were given at the end of the procedure and after extubation criteria was met, an awake extubation was done in the theatre suite and the patient was transferred to the post anaesthesia care unit (PACU).

On arrival at the PACU after connecting to a multiparameter monitor (pulse oximetry and non-invasive blood pressure) the patient became very agitated, removing his gown and wound dressing, threatening PACU staff members and trying to climb off the stretcher. Attempts were made to verbally re-orientate the patient and inquired if he was in pain which he said he did not have but wanted to leave. Using the Richmond agitation-sedation score his was +4. We called in other theatre staff members to help to physically restrain him to prevent him injuring himself and others. The patient was given diazepam 10 mg intravenously. He became calm and sedated. He was kept him in the PACU for 45 min to monitor him then transferred him to the normal surgical ward after the PACU discharge criteria was met. Postoperatively clear instructions were given for the patient to be recommenced on his usual evening dose of sulpiride, pethidine and paracetamol, monitoring of vital signs—respiratory rate, pulse, blood pressure and oxygen saturation. The patient was reviewed the next morning (18 h later) on the ward, he was fully awake, calm and orientated in time, place and person. He only complained of mild pain. He did not have any

other reported incidence of delirium in the ward since leaving the PACU till his discharge.

Discussion

This case report highlights issues relating to chronic schizophrenic patients on treatment with antipsychotics and their interaction with anaesthetic agents.

An anxiolytic (benzodiazepine) was given to the patient to calm him down as anxiety is associated with increased incidence of emergence delirium although preoperative benzodiazepines pose a risk of emergence delirium which was noted in this patient after getting preoperative lorazepam which was unnecessary since the patient was calm preoperatively. Sulpiride was continued preoperatively for the patient but contrary to Kudoh et al. reported that there was a high incidence of postoperative confusion if antipsychotics were discontinued [4]. Our index case still had emergence delirium despite receiving antipsychotic therapy. Lepouse et al. also reported that long term antipsychotic treatment reduced the risk of postoperative agitation; the patient in this report was on sulpiride for four years [2].

The choice of sodium thiopentone as induction agent was in view of the benefit of maintaining autoregulation of the cerebral circulation, reducing cerebral metabolic rate thereby reducing intracranial pressure (ICP) [8]. Propofol used as total intravenous anaesthesia (TIVA) or target controlled infusion is advantageous in neuroanaesthesia as autoregulation is preserved to the fragile brain with rapid recovery and reduced incidence of emergence delirium [5]. In 2013 Baradari et al. did a study that showed reduced incidence of emergence delirium in paediatric patients when a laryngeal mask airway was used while in this case an ETT was used which could have increased the risk of emergence delirium [9]. An ETT was used for this case as it guaranteed a secure airway in an abnormal position and lack of access of operative field. Munk et al. in 2016 showed that use of an ETT was significantly related to emergence delirium [10]. In contrast, volatile agents increase cerebral blood flow and ICP but ICP is unaffected by concentrations of <1 MAC of sevoflurane and isoflurane but still increasing the risk of emergence delirium [2,9,10]. A balance was maintained due to unavailability of TIVA pumps by using sevoflurane at concentrations of <1 MAC to maintain adequate brain perfusion vs. the risk of emergence delirium.

Pain management was done through a multimodal approach opioids and paracetamol intravenous to prevent hypertension as well as having adequate analgesia prevents postoperative confusion. The procedure was of short duration reducing the risk of emergence delirium [2].

In the PACU, emergence delirium can be dangerous and have serious consequences for the patient such as injury to the patient and PACU staff, increased pain and bleeding. Munk et al. documented a notable consequence of emergence agitation was the need for additional staff in order to restrain the agitated patient [10]. As evidenced in this case report, more theatre staff were required to physically restrain the patient who had to be called in from other theatre suites, longer stay in the PACU as

well as administration of diazepam for sedation. A shorter acting agent such as midazolam would have been a better option as it would be easier to reassess the patient after 1-2 h in contrast to diazepam after 12 h. As there was no complaint of pain in the PACU as a potential cause of the emergence delirium no analgesia was given. Postoperatively, the patient was recommenced on sulphuride to avoid unexpected episodes of negative and positive symptoms of schizophrenia.

Conclusion

In summary, we have described a patient with chronic schizophrenia on sulphuride who was given a general anaesthetic for a craniotomy for a depressed skull fracture who developed emergence delirium. Agents or drugs used were to allow continued adequate cerebral perfusion but with risk of emergence delirium due to limited resources such as the TIVA pumps. Preventive measures were employed to reduce the risk of emergence delirium *via* preoperative continuation of sulphuride, reducing anxiety by use of anxiolytics and adequate intraoperative pain control but the patient still had emergence delirium alluding to the speculation that emergence delirium occurs due to other factors.

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