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Repeated episodes of difficulty with arousal following general anesthesia in a patient with ulnar neuropathy

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Abstract
Delayed emergence following general anesthesia may be due to serious life-threatening disorders and deserves prompt evaluation. Rarely, delayed emergence has been attributed to a psychiatric or psychological cause. This report describes an otherwise healthy and mentally sound 52-year-old woman who experienced repeated dissociative episodes following general anesthesia for minor surgical procedures. These episodes lasted for 5 h and resulted in admission to the intensive care unit. The current literature is reviewed to identify commonalities among previously reported cases and to discuss different psychiatric and psychological mechanisms that can play a role in the development of this disorder.

Key words Dissociative disorder · General anesthesia · Psychogenic coma

Introduction

Delayed emergence after general anesthesia may represent severe conditions, such as neurologic insults, metabolic derangements, hypothermia, or the residual effects of intravenous or local anesthetics [1,2]. However, there are rare situations in which delayed emergence from anesthesia may be the result of psychogenic factors ("psychogenic coma"); namely, factitious, conversion, and dissociative disorders [3–9]. Because psychogenic coma is a diagnosis of exclusion, patients undergo an extensive work-up to identify potentially life-threatening conditions. These patients usually have an underlying psychiatric or psychological condition, personality disorder, or external incentive for symptom fabrication [3,4,6–8,10,11]. We report a unique case of a woman with no history of psychiatric disorders or psychological conditions who displayed repeated dissociative episodes following general anesthesia.

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Case description

A 52-year-old, 49-kg woman was admitted for replacement of a peripheral nerve stimulator under general anesthesia. Her medical history included distal ulnar neuropathy from compression at Guyon's canal, for which she had undergone previous carpal tunnel release surgery. This surgery had been complicated by complex regional pain syndrome (CRPS). Failing conservative therapy, the CRPS was subsequently treated with an implantable peripheral nerve stimulator, in August 2007, 10 months prior to the present operation. Her preoperative medications included pregabalin, duloxetine, tramadol, and acetaminophen/hydrocodone. She denied prior anesthetic complications, except for postoperative nausea and vomiting.

The general anesthesia used in this patient included 2 mg of midazolam, 100 μg of fentanyl, 100 mg of propofol, 40 mg of succinylcholine, and 1% isoflurane. Following an uneventful 2-h operation the patient followed commands, responded to questions, and her trachea was extubated. Upon arrival to the recovery room, she was sleepy, but arousable and responded to questions. Vital signs during the first hour were within normal limits and she was not hypothermic. Her breathing was unremarkable with unlabored respirations with adequate tidal volumes and a respiratory rate ranging between 14 and 20 breaths per min. During the second hour in the recovery room, the nurse was unable to arouse the patient. She was unresponsive to intense tactile and painful stimuli induced by rubbing the sternum and pinching the toes. Her eyes were tightly closed, and opening the eyelids met resistance. It appeared that the patient's eyes were moving beneath the eyelids, but she would not open her eyes. Her pupils

were dilated, responsive to light, and 7 mm in diameter. Lifting the arm above her face and letting it drop did not result in any protective maneuver by the patient (though the anesthesiologist did not let the arm hit her face). The use of tetanic stimulation (50 Hz, 50 mA for 5 s) to stimulate the left ulnar nerve resulted in appropriate muscular adductor pollicis response, but this stimulus did not elicit any signs that would indicate that the patient perceived pain (no facial grimacing or limb withdrawal). In order to exclude residual opioid or benzodiazepine effects, 200 μg of naloxone, followed by 0.4 mg of flumazenil, was administered, without any improvement of consciousness. The electrolytes, hemoglobin, arterial blood gases, and thyroid function were normal. After the patient had not awakened for 2 h, a neurologist was consulted. There were no focal deficits, or abnormal posturing, and head magnetic resonance imaging scan was normal. Approximately 5 h after the operation, she started to mumble answers to questions without opening her mouth or eyes and started wiggling her toes to command. Over the next 12 h, her mental status returned to normal. She was discharged home on the second postoperative day. Before discharge she neither denied recollection of any problems nor was aware of any events or excessive sleepiness. The consulting neurologist attributed the delayed awakening to a "functional component".

Review of previous anesthetic and medical history

Review of the patient's past medical records revealed that she had had four prior surgeries. In December 2005 she had had general anesthetic with a laryngeal mask airway (LMA) for right carpal tunnel release surgery aimed at relief of distal ulnar neuropathy. The anesthetic included 160 mg of propofol, 50 μg of fentanyl, midazolam 2 mg, and 1.1% isoflurane. Her postoperative course was unremarkable, and she was discharged home the same day. Prior to this surgery she was not taking any medications. In July 2006 she underwent reexploration of her right hand carpal tunnel. Her preoperative medications at that time included: gabapentin, tramadol, and acetaminophen-propoxyphene. This time the primary anesthetic consisted of a continuous axillary block with 0.5% bupivacaine (total 36 ml) in conjunction with a general anesthetic. The latter was delivered using an LMA, 100 mg of propofol, and 0.3% isoflurane and nitrous oxide. Her trachea was extubated in the operating room and she was noted to be fully oriented and conversant. Upon arrival to the recovery room she became unresponsive. This lasted for 5 h and resulted in an intensive care unit admission. A neurologist was consulted, and, aside from unresponsiveness, no neurologic abnormalities were found. A head magnetic resonance scan was normal. Her extensive medical evaluation did not reveal any condition that could account for the decreased consciousness. At the time, the neurologist attributed the slow awakening to a presumptive "opioid overdose," although the anesthesia record showed that she received only 150 μg fentanyl. The patient had two subsequent procedures requiring general anesthesia. One was in December 2006 for revision of the ulnar nerve entrapment, with anesthesia consisting of propofol 140 mg, succinylcholine, fentanyl (160 μg), midazolam (2 mg), 1.2% isoflurane, and nitrous oxide. At the time of this operation her preoperative medications were the same as those with her previous surgery (July 2006). In August 2007 she had undergone surgery for implantation of a peripheral nerve stimulator under general anesthesia. At the time of this operation she was taking pregabalin, duloxetine, and tramadol. Both these anesthetics were uneventful.

In contemplating implantation of the peripheral nerve stimulators she was evaluated by a psychiatrist who found neither evidence of mental health issues nor evidence of a primary psychiatric illness or personality disorder. Rather, he felt that the patient was a successful mother, wife, and teacher with rational career ambitions and that her adjustments and adaptations for her disability were appropriate. No Axis I or Axis II DSM-IV diagnoses were made.

Discussion

We described a patient who had five general anesthetics over a 2-year period and on two occasions developed delayed emergence (5 h) which resembled a comatose state. All organic causes were excluded. During all anesthetics similar drugs were used, and opioids and benzodiazepines were never administered in excessive amounts. Furthermore, pharmacologic reversal with naloxone and flumazenil did not result in emergence. Her preoperative medications did not vary substantially between the different anesthetics. The use of duloxetine, gabapentin, and pregabalin was for the treatment of neuropathic pain, and not for treatment of a mood disorder. Also, she used opioids minimally, as she disliked the side effects. Based on these considerations, we feel the most probable etiology of the delayed awakening was psychogenic.

Psychogenic coma following general anesthesia is rare [3,4,6–8,10–15]. Most previous cases involved female patients undergoing otolaryngologic or gynecologic procedures. Repeat episodes of psychogenic delayed awakening have been reported in four patients.[3,4,7,12] Our patient differed in that she experienced episodes of uneventful emergence from general anesthesia that alternated with episodes of delayed

awakening. Review of her medical record could not identify an obvious organic mechanism to explain the delayed awakening. Regardless, delayed emergence in a patient who previously experienced "functional" delayed awakening should alert the anesthesiologist to the possibility of a psychiatric cause. Indeed, in the majority of previously reported cases, the patient had a significant psychiatric history, including primary psychiatric disorders (depression, bipolar disorder, posttraumatic stress disorder, anxiety, or substance abuse history) [4,6–8,11,14] and/or personality disorder (borderline and histrionic personality traits) [3,4]. Our patient had undergone preoperative psychiatric evaluation which did not reveal either a psychiatric or a personality disorder.

There are several conditions that can be associated with psychogenic delayed awakening after general anesthesia. Intentional production of symptoms can represent either malingering or a factitious disorder. *Malingering* is a technique employed by feigning the sick role for secondary gain (such as avoidance of work or military service). No delayed awakening from anesthesia has been attributed to malingering. *Factitious disorders*, such as Munchausen syndrome, differ from malingering in that patients feign illness to fulfill a psychological need to assume a sick role rather than for obvious secondary gain [16]. Albrecht et al. [3] described two cases of delayed awakening, in two female patients with histrionic personalities, that were attributed to a factitious disorder. In both cases, symptoms were easily reversed: by a cold caloric test and a placebo injection [3]. Other cases of factitious disorder-delayed awakenings were reversed by airway obstruction [6], electrical tetanic stimulation [12], and with the inhalation of ammonia salts [11]. Unintentional causes of psychogenic delayed awakening include conversion and dissociative disorders. *Conversion disorders* can present as weakness or paralysis of the entire body, and are the result of an underlying psychological conflict. Conversion disorder has been implicated as the cause of delayed awakening in a patient with a history of severe physical and sexual abuse [7]. *Dissociative disorders* are conditions that involve disruptions or breakdowns of memory, awareness, and identity and/or perception, and indifference towards painful stimuli [17]. Dissociative disorders as a cause of delayed emergence from anesthesia have been rarely reported [8,13,14]. Our patient most likely experienced a dissociative disorder, as she lacked traits associated with malingering, factitious disorders, or obvious internal psychological conflicts putting her at risk for a conversion disorder.

Theoretically, in cases stemming from intentional production of symptoms, such as factitious disorders, coma symptoms are likely to be observed as the patient is feigning coma to assume the sick role or for secondary gain. In contrast to intentional causes, coma symptoms from unintentional psychological causes should persist even if the patient is not aware that he/she is being observed. However, the utility of observing whether symptoms persist when the patient believes he/she is not being observed as a means of distinguishing intentional and unintentional coma has not been evaluated.

Because cases of delayed awakening may represent a serious condition, they require prompt investigation. Some signs could alert the anesthesiologist to the possibility of a psychogenic cause. Eye movements, persistent gaze, and tightly shut eyelids are suggestive of a psychogenic coma [4]. Our patient resisted opening of the eyelids and was witnessed to move her eyes when her eyelids were closed. Testing of the oculocephalic reflex, or doll's eye maneuver, or the oculovestibular reflex, or cold caloric test, can assist in making the diagnoses of a psychogenic delayed emergence [3,4,7]. Protective reflexes, such as dropping the arm towards the patient's face, are often intact [4,6,7,11,12], but in our patient this did not appear to be the case. In confirmed cases of psychogenic coma, supportive care and reassurance are recommended [9]. Repeating noxious stimuli is not humane and is not advocated [9]. Cases have been reversed with the administration of benzodiazepines or antipsychotics and this could be considered in recalcitrant cases [7,9,13,15].

In conclusion, we described a patient who had psychogenic delayed awakening in two out of five general anesthetics. The most likely etiology of this was a dissociative disorder, as the patient did not have motives for malingering, evidence of a factitious disorder, or a conversion disorder. This event resulted in a prolonged hospitalization and in substantial increase in diagnostic cost.

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References

1. Douglas JH 3rd, Ross JD, Bruce DL. Delayed awakening due to lidocaine overdose. J Clin Anesth. 1990;2:126–8.
2. Munis JR, Marcukaitis AW, Sprung J. Delayed emergence from anesthesia associated with absent brainstem reflexes following suboccipital craniotomy. Neurocrit Care. 2006;5:206–9.
3. Albrecht RF 2nd, Wagner SRt, Leicht CH, Lanier WL. Factitious disorder as a cause of failure to awaken after general anesthesia. Anesthesiology. 1995;83:201–4.
4. Downs JW, Young PE, Durning SJ. Psychogenic coma following upper endoscopy: a case report and review of the literature. Mil Med. 2008;173:509–12.
5. Eisendrath SJ. Factitious physical disorders. West J Med. 1994;160: 177–9.
6. Maddock H, Carley S, McCluskey A. An unusual case of hysterical postoperative coma. Anaesthesia. 1999;54:717–8.

7. Meyers TJ, Jafek BW, Meyers AD. Recurrent psychogenic coma following tracheal stenosis repair. Arch Otolaryngol Head Neck Surg. 1999;125:1267–9.
8. Weber JG, Cunnien AJ, Hinni ML, Caviness JN. Psychogenic coma after use of general anesthesia for ethmoidectomy. Mayo Clin Proc. 1996;71:797–800.
9. Baxter CL, White WD. Psychogenic coma: case report. Int J Psychiatry Med. 2003;33:317–22.
10. Hwang J-L, Kuo M-C, Hsieh B-C, Chang C-H, Jou L-C, Chen W-H, Wu G-J. An acute psychiatric episode following transvaginal oocyte retrieval: case report. Hum Reprod. 2002;17:1124–6.
11. Orr DL, Glassman AS. Conversion phenomenon following general anesthesia. J Oral Maxillofac Surg. 1985;43:817–9.
12. Adams AP, Goroszeniuk T. Hysteria. A cause of failure to recover after anaesthesia. Anaesthesia. 1991;46:932–4.
13. Haller M, Kiefer K, Vogt H. Dissociative stupor as a postoperative consequence of general anesthesia. Anaesthesist. 2003;52:1031–4.
14. Pertek JP, Omar-Amrani M, Artis M, Vignal JP, Chelias A. Failure to recover after anesthesia attributed to a transient dissociative state. Ann Fr Anesth Reanim. 2000;19:257–60.
15. Chang Y, Huang CH, Wen YR, Chen JY, Wu GJ. Dissociative amnesia after general anesthesia—a case report. Acta Anaesthesiol Sin. 2002;40:101–4.
16. Diagnostic and statistical manual of mental disorders: DSM-IV. 4th ed. Washington DC: American Psychiatric Association; 1994.
17. Ross CA, Joshi S, Currie R. Dissociative experiences in the general population: a factor analysis. Hosp Community Psychiatry. 1991;42:297–301.