Case Report

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Transient aphonia in a patient undergoing laparoscopic cholecystectomy: A case report

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ABSTRACT

This case report presents an otherwise healthy 36-year-old female patient who underwent laparoscopic cholecystectomy due to acute cholecystitis and postoperatively exhibited transient aphonia.

Key Words: *Laparoscopic cholecystectomy; transient aphonia; self-limiting aphonia; post-cholecystectomy aphonia*

INTRODUCTION

Laparoscopic cholecystectomy is widely accepted as the standard of care for treating cholecystitis in the acute setting [1]. Although considered a minor surgical procedure and associated with minimal systemic complications, administration of general anaesthesia may still lead to significant adverse events [2-5]. CO2 inflation, steep patient position and prolonged operative time may predispose to the emergence of neurological complications especially in prone patients [4,5]. Neurological disorders after surgery are not uncommon in high-risk patients [6]. However, only rarely have they been reported in the literature in young healthy patients undergoing abdominal surgery [7-10]. In this study, we report the case of a young patient presenting aphonia immediately after laparoscopic cholecystectomy.

CASE REPORT

A 36-year-old female patient was referred to our department after diagnosis of acute cholecystitis. Personal and family medical history were insignificant. After initial evaluation and appropriate supportive treatment, she was scheduled for a laparoscopic cholecystectomy. Routine preoperative lab tests, including complete blood count (CBC), liver function tests (LFTs), renal function, coagulation studies, electrolytes and lipid profile were obtained along with preoperative electrocardiogram (ECG).

In the operating room, a standard laparoscopic cholecystectomy was performed without any intraoperative complication. The procedure was performed under general anaesthesia. Anaesthetic regimen included propofol and fentanyl for induction and remifentanil used for maintenance, without any adverse reaction. Cholecystectomy was performed under a low intra-abdominal pressure of CO2 (10-11mmHg). Awakening and extubation were uneventful. Following extubation, it became obvious that the patient was unable to speak, although she could follow verbal commands. There was no deterioration of respiration, and all vital signs remained normal. Muscle tone was intact. Emergency neurological consultation was performed in the operating room. No other neurological deficit was observed other than the inability to speak. Emergency otorhinolaryngology evaluation with laryngoscopy revealed normal vocal cord movement. The patient was kept in the resuscitation room for monitoring and possible airway management in case of emergency. After 45 minutes, her voice started gradually to recover, with full recovery within three hours after extubation.

Postoperative course was otherwise insignificant. Prior to discharge the patient was re-evaluated by a neurologist and psychiatrist and scheduled for imaging (brain MRI, carotid and lumbar artery ultrasound) and detailed

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coagulation studies to rule out underlying causes of the event. The patient has been followed-up for the last four months. No similar symptoms have been exhibited during this time.

DISCUSSION

Transient postoperative aphonia after abdominal surgery is rare and limited case reports describe such episodes across the literature [2-5]. Differential diagnosis includes transient ischemic attack (TIA), conversion disorder, non-reversed pharmacological effect and vocal cord injury during airway management [2-5]. TIA is described as a focal neurological deficit lasting for minutes with full neurological recovery [6]. It can be attributed to transient hypoxia, hypotension or embolism and predisposes to ischaemic attack in the near future. Several surgical diseases predispose to the development of venous thrombosis. Prolonged immobilisation, inflammation or underlying neoplasm may precipitate the emergence of deep venous thrombosis, associated with ischemic brain attacks [7]. In our case of a 34-year-old healthy woman, with normal prior medical history and initial coagulation studies, the diagnosis of a TIA is deemed not likely. Furthermore, preoperative measures with prophylactic anticoagulants and compression socks were employed and no signs of deep vein thrombosis observed pre- or postoperatively.

Laparoscopic surgery can be associated with increased neurological adverse outcomes owing to possible CO2 systemic absorption and subsequent effect on cerebral vessels [7,8]. This is more likely in prone patients with multiple comorbidities, extended operative time and high CO2 pressure. In this instance, surgery was completed in under 50 minutes of pneumoperitoneum with maintenance of "low-pressure" (10-11mmHg) throughout the operation. Furthermore, CO2 values based on intraoperative capnometry and postoperative arterial blood gas analysis was within normal range. Extreme positioning of the patient during the operation may be associated with postoperative deficits. Such cases have been reported after gynecological surgery, with extensive time in the Trendelenburg position [7-10]. This can be attributed to venous congestion and subsequent cerebral oedema [7-10]. However, in our case, as in the majority of laparoscopic cholecystectomy procedures, only a slight reverse Trendelenburg position was employed.

Anaesthesia drug effect is a possible cause of neurological deficit in the immediate postoperative period. Unreversed neuromuscular blockade can lead to muscle weakness, including vocal cord paralysis [11]. In addition to that, possible trauma during intubation or extubation may cause inability to talk normally [12]. In our case, however, muscle tone was uninfluenced and postoperative laryngoscopy excluded structural or functional damage to the vocal cords.

Psychogenic disorders may present postoperatively in various forms. Surgery, especially in the emergency setting, has been described as a major stressogenic event. Postoperative delirium may not be uncommon and can be expressed as speaking disability. Certain anaesthesia and muscle relaxation agents, including midazolam and scopolamine, have been proven to elicit such attacks, although they were not used in our case [10].

Regarding the management of a patient who develops aphonia immediately postoperatively, the investigation should be immediate. The primary goal should be to secure the patient's airway. This will be done by assessing the patient's level of consciousness and pO2. Immediate laryngoscopy to rule out injury or oedema of the vocal cords and other laryngeal structures is essential. At the same time, the patient's ability to breathe should be checked. This will be done by listening to the chest to check the patient's ventilation, as well as the reversal of anaesthetic drugs. Next, a check for neurological deficits, other than aphonia, will be performed. A CT scan of the brain at two to six hours is necessary to rule out a stroke and, if it does not reveal pathology, repeat it at 24 to 48 hours. The patient should remain under monitor observation to ensure respiratory and haemodynamic stability. Performing MRI and carotid triplex is optional after the patient's discharge, as is rechecking by a neurologist. Aphonia most often returns in the first postoperative hours, however, this may take longer. Despite the earlier belief that if there is no immediate reversal of the aphonia, it may be permanent, this is not documented [16,17]. Table 1 lists possible modes of potential causes of postoperative transient aphonia and associated pathophysiological mechanisms.

CONCLUSION

Transient aphonia is an extremely rare complication after surgery under general anaesthesia. Emergence of such symptoms should prompt appropriate measures in order to ensure appropriate patient support while assessing the possibility of reversible causes. Immediate supportive management according to standard protocols should prioritise assessing and securing airway patency, sufficient ventilation and haemodynamic stability. After initial assessment and support, detailed neurological, psychiatric and ENT assessment is mandatory in order to promptly identify possibly reversible causes, including ischaemic brain injury, in the acute phase.

In our case, following immediate supportive measures, urgent clinical assessment , imaging studies and labora-

TABLE 1. Potential causes of postoperative transient aphonia and associated pathophysiological mechanisms.

CAUSES	MECHANISM
Endotracheal intubation factors [14,15,16]	Vocal cord injury/oedema Improper endotracheal tube position
Anaesthesia-related factors [11]	Muscle relaxants related voice volume reduction Drug-induced vocal cord irritation
Postoperative allergic reaction [14,16]	Laryngeal oedema
Psychogenic aphonia [17]	Hyperfunctional type: characterised by a significant contraction of the vocal cords (less common) Hypofunctional type: vocal folds come close together but do not fully close (more common)

tory workup failed to reveal any abnormal findings that would explain the patient's symptoms. Following detailed neurological and psychiatric evaluation and laboratory follow-up, psychogenic disorder is considered the most likely cause of the symptoms exhibited by our patient [12]. The full recovery of voice function, the absence of other symptoms or abnormal findings of postoperative workup support this diagnosis.

Conflict of interest: *None*

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