

## Anesthesia Fatality due to Bile Aspiration: Lessons to be Learnt

Nouman I Alvi\*

Department of Anesthesiology, Aga Khan University, Karachi, Pakistan

\*Corresponding author: Nouman I Alvi, Assistant Professor, Department of Anesthesiology, Aga Khan University, Stadium Road, Karachi, Pakistan, E-mail: nouman.alvi@aku.edu

Received date: May 16, 2016; Accepted date: June 03, 2016; Published date: June 06, 2016

Copyright: © 2016 Alvi NI. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

### Abstract

Anesthetic related fatality is rare in this time and age in young patients who are ASA II (American Society of Anesthesiologists). Our patient had a very unfortunate anesthetic mortality due to aspiration of bilious vomit. A posthumous diagnosis of post Endoscopic Retrograde Cholangio-pancreatography (ERCP) pancreatitis leading to ileus was missed before surgery. There is a brief literature search about perils of bile aspiration, post ERCP pancreatitis and why pancreatitis is a lethal combination with acute respiratory distress syndrome (ARDS).

**Keywords:** Aspiration; Bile; Pneumonia; Fatality; Post ERCP pancreatitis

### Introduction

Patient assessment for surgery and anesthesia can be very complex sometimes even in simple surgeries. We wish to share a case of a seemingly low risk man who in fact had pancreatitis which could not be preemptively picked despite successive assessments done by anesthesia and surgical teams. This led to anesthetic complications and fatality. The case highlights importance of recognizing underlying pancreatitis and impaired gastric emptying in patients coming for straightforward cases like elective cholecystectomy.

### Case Report

A 29-year ASA II male was anesthetized for elective cholecystectomy. Patient was assessed by both anesthesia and surgery teams on the morning of surgery. He denied any symptoms suggestive of on-going intestinal obstruction. He reported that he had been eating, drinking and passing flatus normally for the past several days. His amylase and lipase levels were 180 U/L and 220 U/L, Alk. Phos. was 220 U/L and direct Bilirubin was 1.2 mg/dL. Resting SpO<sub>2</sub> and Chest X-rays were not done.

He was induced with Propofol, Fentanyl and Atracurium. Moments after induction patient had spontaneous regurgitation of large amounts of bilious vomit. The oro-pharynx was suctioned and endo-tracheal tube (ET) was promptly introduced followed by IPPV (positive pressure ventilation) on ventilator. Subsequently patient developed desaturation and was hemodynamically unstable but stabilized with appropriate interventions. The surgery was abandoned and it was decided to shift the patient to ICU for further management. He was meanwhile ventilated and anesthetized.

Two hours later, at the time of transfer to (intensive care unit) ICU SpO<sub>2</sub> (oxygen saturation) had dropped to 90% on 100 FiO<sub>2</sub> (inspired fraction of oxygen). BP (blood pressure) was 140/90, HR (heart rate) was 125-138 per minute, Temp was 38.6 Celsius and bilateral white fluffy shadows had already developed on the Chest X-ray. A presumptive diagnosis of Acute Lung Injury (ALI) due to aspiration was made.

During the following days in ICU, the patient needed high vasopressor requirements due to fulminant sepsis. He developed acute respiratory distress syndrome (ARDS) and multi organ failure. He died on 4th postoperative day in the ICU. The patient's family denied a request for post mortem examination.

This case was declared a sentinel event. Our Department discussed this case in morbidity and mortality meeting. This was followed by a full, multi-disciplinary, root cause analysis (RCA). We identified remediable and fixed factors contributing to this extremely unfortunate incident. Areas marked for improvement were: better surgeon-anesthesiologist communication, taking history from patient who has been medicated with Midazolam, caution about patient with recent history of Endoscopic procedures, caution about patients who have in-situ foreign bodies in their alimentary tract and improved documentation about procedures done from outside hospitals.

The consensus showed that the patient had developed post ERCP (Endoscopic Retrograde Cholangio-pancreatography) pancreatitis that was missed before scheduling of surgery. He had undergone an ERCP and Duodenal Stent removal under monitored anesthesia care (MAC) sedation three or four days in a different hospital which he had not informed to his primary team. In hindsight, he suffered with ileus and impaired gastric emptying despite denying symptoms of intestinal obstruction or ileus. There is a possibility that he also had aspiration pneumonia already; he had had two separate ERCP's done under MAC (Monitored Anesthetic Care), when a secure airway was not used. This was done in a different hospital which was not shared earlier.

### Discussion

Aspiration is the commonest cause of anesthesia related fatality [1,2]. Recent literature has highlighted that patient selection; assessment of risk factors for aspiration is the best technique to mitigate risks of aspiration [1,2]. The pre-operative assessment and induction should ideally be carried out by the senior most anesthetists [1-3]. Rapid sequence induction (RSI) is the standard of care and should be actively considered in patients who have delayed gastric emptying time [1-3].

However it is primarily the robust assessment and patient selection which really saves the patient and not merely the rapid sequence induction (RSI).

Our findings mirror the situation in other parts of the world e.g. in NAP4 [3] (4th National Audit Project of the Royal College of Anaesthetists, UK) study and other literature [1,2]. Importance of pre-operative assessment and updated medical history is highlighted. Anticipation of aspiration is perhaps the best protection against aspiration. Patient selection and risk assessment should be stringent. Rapid sequence induction is still the universal practice of choice in any situations where GI motility is altered directly or indirectly [1,3].

The debate of early or delayed cholecystectomy in patients with pancreatitis is unsettled and surgical colleagues should carefully review each patient.

We believe, in our instance, the aspiration of bilious vomit while having concurrent post-ERCP pancreatitis and aspiration pneumonia, acted as a compounding factor, leading to acute worsening of sepsis. This ensuing vicious cycle, resulted in a rapidly progressive multi organ failure and death.

Our patient was particularly unfortunate in the sense that the aspirated content consisted primarily of bile.

Bile acid is an alkaline juice which contains salts and chenodeoxycholic and taurocholic acid. Bile acid and bile salts have been incriminated in causing mucosal injury to esophagus, larynx [4], epiglottis and lung parenchyma. They have been found to be associated with neonatal respiratory distress syndrome [5], bile pneumonia [5], alveolitis, bronchiolitis obliterans [6,7], and exaggerated injury in ventilator associated pneumonias [8,9]. There is understandably a paucity of studies done on humans but experimental studies conducted on rats [10], rabbits [11] and pigs [12], have demonstrated that bile acids cause inflammation and cytotoxic effects on histologic examinations. Similarly, broncho-alveolar lavage studies have found a positive correlation between increased bile acid levels and inflammatory markers in blood, increased neutrophilia [13], inflammation in alveoli and ventilator associated pneumonias [9]. Likewise visceral inflammation and injury of lungs, larynxes were found to be related to Bile Juice reflux independently of acid reflux in gastroesophageal reflux diseases [6].

Bile aspiration as opposed to acid aspiration has much worse prognosis. Bile, being an alkaline agent causes pulmonary damage, which is far more damaging than acid or other gastric juices. Bile acid has repeatedly been shown to cause direct pulmonary damage. The mechanisms have been oxidative stress, damage to endothelial cells, Type 1 and 2 pneumocytes, depletion of surfactant and damage to phospholipids [4-6]. This aspiration leads to chemical pneumonitis that turns into a fulminant ARDS carrying a much higher mortality.

Post-ERCP pancreatitis is the commonest complication of ERCP [14,15]. It should be considered in all patients coming for cholecystectomy, being done after any ERCP. It can lead to a worsening of an already grave and silent condition. Anesthesia management needs to be improvised because patient is high risk for aspiration. Rapid sequence intubation should be the standard practice [1-7].

It is imperative to prevent these patients from getting MAC (monitored anesthetic care) or procedural sedation without a secure

airway e.g. cuffed tracheal tube when they come for ERCP and other related procedures.

## Conclusion

While aspiration and risk of impaired gastric motility as anesthetic risk are well known risk factors, this case was an unforgettable learning opportunity for us. It reminded us how a patient can fall through the safety net despite many checks and balances. Both anesthesia and surgery with their highly skilled staff were unable to pick up on the clues and misled by innocuous findings. Our patient highlighted for us the importance of dynamic and repeated assessments of patients before surgery and anesthesia. It also highlighted the dilemma about the uncertainty of timing of cholecystectomy in patients with pancreatitis. This fatality reminds of the rare but grave complications like post-ERCP pancreatitis that can make a bad situation worse.

## References

1. Nason KS (2015) Acute Intraoperative Pulmonary Aspiration. *Thorac Surg Clin* 25: 301-307.
2. Abdulla S (2013) Pulmonary aspiration in perioperative medicine. *Acta Anaesthesiol Belg* 64: 1-13.
3. Cook TM, Woodall N, Harper J, Benger J (2011) Major complications of airway management in the UK: results of the Fourth National Audit Project of the Royal College of Anaesthetists and the Difficult Airway Society. Part 2: intensive care and emergency departments. *Br J Anaesth* 106: 632-642.
4. Sasaki CT, Marotta J, Hundal J, Chow J, Eisen RN (2005) Bile-induced laryngitis: is there a basis in evidence? *Ann Otol Rhinol Laryngol* 114: 192-197.
5. Zecca E, Costa S, Lauriola V, Vento G, Papacci P, et al. (2004) Bile acid pneumonia: a "new" form of neonatal respiratory distress syndrome? *Pediatrics* 114: 269-272.
6. Sweet MP, Patti MG, Hoopes C, Hays SR, Golden JA (2009) Gastro-oesophageal reflux and aspiration in patients with advanced lung disease. *Thorax* 64: 167-173.
7. D'Ovidio F, Mura M, Tsang M, Waddell TK, Hutcheon MA, et al. (2005) Bile acid aspiration and the development of bronchiolitis obliterans after lung transplantation. *J Thorac Cardiovasc Surg* 129: 1144-1152.
8. Tsai HC, Lin FC, Chen YC, Chang SC (2012) The role of total bile acid in oral secretions in ventilator-associated pneumonia. *J Crit Care* 27: 526.
9. Wu YC, Hsu PK, Su KC, Liu LY, Tsai CC, et al. (2009) Bile acid aspiration in suspected ventilator-associated pneumonia. *Chest* 136: 118-124.
10. Yu L, Ding Y, Huang T, Huang X (2014) Effect of Bile Acid on Fetal Lung in Rat Model of Intrahepatic Cholestasis of Pregnancy. *International Journal of Endocrinology* 2014: 308274.
11. Henderson RD, Fung K, Cullen JB, Milne EN, Marryatt G (1975) Bile aspiration: an experimental study in rabbits. *Can J Surg* 18: 64-69.
12. Porembka DT, Kier A, Sehlhorst S, Boyce S, Orłowski JP, et al. (1993) The pathophysiologic changes following bile aspiration in a porcine lung model. *Chest* 104: 919-924.
13. Zecca E, De Luca D, Baroni S, Vento G, Tiberi E, et al. (2008) Bile acid-induced lung injury in newborn infants: a bronchoalveolar lavage fluid study. *Pediatrics* 121: e146-149.
14. Ding X, Zhang F, Wang Y (2015) Risk factors for post-ERCP pancreatitis: A systematic review and meta-analysis. *Surgeon* 13: 218-229.
15. Thaker AM, Mosko JD, Berzin TM (2015) Post-endoscopic retrograde cholangiopancreatography pancreatitis. *Gastroenterol Rep (Oxf)* 3: 32-40.