Case report

Apnea test induced barotrauma in an obese subject with a short neck - A case report

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ABSTRACT

Death is an assured inevitability event for all living beings of this cosmos. Based on the American Academy of Neurology (AAN) recommendations, clinical diagnosis of Brain Death (BD) includes multiple sequences of tests. The Apnea Test (AT) is a more significant procedure in diagnosing BD. Unfortunately, complications during this procedure can occur more frequently than documented, and its incidence is limited to case reports. However, most critical care physicians and Cardio-respiratory therapists consider an AT a principal method to confirm BD as it provides decisive information about the definitive loss of brainstem function. Here, we describe the execution of the AT procedure that transpired into extensive subcutaneous emphysema, tension pneumothorax, and pneumoperitoneum followed by pulseless electrical activity due to direct airway perforation by the supplemental oxygen catheter. In addition, we do a literature review on airway complications incidence and suggest methods to avoid such fatal complications. Finally, further prospective studies are recommended to evaluate the barotrauma incidence and complication nature during AT.

Keywords: Brain death (BD); Apnea test (AT); Endotracheal tube (ETT); Body mass index (BMI).

INTRODUCTION

Death is an assured inevitability event for all living creations, from first-cell organisms to the last human of this cosmos. Globally, for centuries the concept of brain death has been controversial and the conception of a brain death in medical sciences has been developed in the last five decades. In 1980 American Medical Association depicted BD as "Irreversible cessation of all clinical circulatory and respiratory functions, or irreversible cessation of all clinical functions of the entire brain, including the brain stem (1). Hence, Irreversible traumatic or non-traumatic brain injury can cause BD in which brain and brain stem functions cease permanently. Therefore, the determination of BD with current accepted medical guidelines is vital. In the United States, the incidence of BD is about 2.06% annually (2). According to the Saudi organ transplant center annually 606 irreversible traumatic brain injury individuals are seen below 40 years old (3). The etiology of brain death is multifactorial, and it may happen from traumatic or non-traumatic events (medical conditions). Brain death is commonly seen because of intracerebral hemorrhage or infarct, aneurysmal subarachnoid hemorrhage, cardiac arrest, and severe head injury. However, any sudden abruption of cerebral perfusion associated with a rise of sudden intracranial pressure greater than mean arterial pressure can lead to acute severe brain injury, and this mechanism of brain injury can occur in two ways. Extracranial brain injury most commonly happens in post-cardiopulmonary arrest patients due to resuscitation delay causing blood flow cessation to the brain. Whereas intracranial brain injury typically happens in intracerebral hemorrhage and traumatic brain injury, causing an elevation in intracranial pressure and impaired oxygenation to the brain (4). In this context, the prime characteristics that illustrate the brain death of a person when on mechanical ventilation are an unresponsive coma, absent brainstem functions, and apnea. Hence, vigilant clinical examinations such as brain stem reflex assessment and apnea tests are vital. Clinically, the apnea test is the most common clinical method to confirm brain death.

The traditional strategy to accomplish an AT is to place the patient in a semi fowlers position followed by continuous monitoring of heart rate, blood pressure, and pulse oximeter. Once hemodynamic stability is maintained, the mechanical ventilator is discontinued and oxygen insufflation via a suction catheter or nasal cannula is introduced into the endotracheal tube (ETT) at the carina level for 8-10 minutes. Spontaneous respiratory motions are monitored cautiously throughout the procedure, 8 liters of O2 are delivered for 8-10 minutes, and PO_2, partial pressure of carbon dioxide (PaCO2), partial pressure of oxygen (PaO_2), and pH levels are measured at the end. Thereupon, the ventilator is connected again. Absence of respiratory motions and increase of arterial $PaCO_2$ of more than >60 mmHg or if there is any increase of $PaCO_2$ greater than 20-mmHg of standard baseline $PaCO_2$ concludes a positive apnea test which supports the BD diagnosis

(5). AT can measure the lower brainstem functionality by increasing arterial carbon dioxide partial pressure ($PaCO_2$) level and simultaneously decreasing the Cerebrospinal fluid (CSF)-pH level at once maximal arousal of respiratory centers in the medulla oblongata is stimulated (6). Clinically the precise $PaCO_2$ level at which the stimulation of medullary chemoreceptors is unknown, while a $PaCO_2$ value of 60 mm Hg or greater than 20 mm Hg from an average baseline signifies an appropriate target level for assessing BD (7). However, AT is a crucial, complex, and time-consuming procedure. Nevertheless, complications during this procedure can happen, and its incidence is limited to case reports. However, most critical care physicians and cardio-respiratory therapists consider an apnea test (AT) as a principal method to confirm brain death (BD) as it provides decisive information about the definitive loss of brainstem function. Here, we describe the execution of the AT procedure, which transpired in pulseless electrical activity due to sudden surgical emphysema, tension pneumothorax, and pneumoperitoneum. In addition, we do a literature review concerning the incidence of airway complications and propose ways to avoid such severe complications.

Case report

A 56-year-old female is medically free from the pulmonary disease, reported to the emergency room with a clinical history of diabetes mellitus type-II and hypertension on irregular medication. On arrival, the patient had a decreased arousal of GCS 8/15. Her vitals: blood pressure 188/102 mmHg, heart rate 127/min, respiratory rate 43/min, temperature 37 °C, oxygen saturation was 68% on room air. Physical examination revealed a weight of 80 kg, a height of 160cm, a body mass index (BMI of 31.3 kg/m2 - obese class- I), a greater neck circumference of 39 cm, and mild jugular distension. In addition, the patient's relative narrated the patient's history of sudden onset of severe headache with period of loss of consciousness, nausea, and vomiting. Based on her current critical clinical status, the team obtained informed consent and decided to intubate her with ETT size 7.5 ID. Her initial airway assessment was virtually impossible with a glide scope since typical laryngeal landmarks could not be able to visualize due to her short neck with Mallam Pati scores of IV. Therefore, an on-call anesthesiologist intubated the patient safely with an endotracheal tube size of 7 mm with a boogie's assistance and connected to a mechanical ventilator for respiratory support. Chest Xray taken (Fig.1) and an urgent CT scan showed a large right frontal and temporal intracerebral hemorrhage and diagnosed as subarachnoid hemorrhage (SAH). Her condition deteriorated while in the CT room subsequently with desaturation to 76% even with mechanical ventilator and from the CT room the patient shifted to ICU. Upon ICU admission the patient was comatose, with an absent cough and gag reflex. Our team decided to wait and observe for the next 48 hours to exclude the likelihood of recovery. After 48 hours of observance, the patient was euthermic with stable cardiovascular functions, bilateral pulmonary rhonchi, and no endocrine, electrolyte, or acid-base disturbance. Nevertheless, there is no sign of responsiveness to loud verbal commands, eye-opening eye movement or motor response in response to noxious stimuli, pupillary reaction to bright light with 5mm fixed dilated, or facial muscle movement. In this situation, the intensive care team decided to confide whether the patient was dead by neurological standards. Repeated CT revealed CNS catastrophe. According to our hospital's BD and organ donation policy, our team notified the patient's surrogate in the decision to do the BD procedure to document the clinical assessment of brain stem reflexes and apnea test to exclude the possibility of recovery. The following clinical examinations conclude the brain death of our patient: Absent oculocephalic reflex, absent eye deviation when frozen water of 60 ccs irrigation in each external auditory, no spontaneous breaths or triggered breaths from the ventilator, no gag, and no cough reflex while during ETT suction or ETT manipulation. After the confirmation of irreversibility, unresponsiveness, coma, and the absence of brainstem reflexes. AT was recommended, and prior to AT, our team confined the temperature of our patient was > 36°C; pre-apnea test arterial blood gas analysis showed a pH of 7.43, a carbon dioxide (PCO_2) of 43 mmHg, and an oxygen tension (PO_2) of 144 mmHg. (Pre-oxygenation was performed for 5 minutes through 100% fraction of inspired oxygen before the ETT was disconnected from the ventilator). Subsequently, oxygen tubing was connected to a suction catheter and inserted into the ETT size 7. According to the AAN guidelines, AT was performed using a 12-french suction catheter inserted 10 cm down into the endotracheal tube to provide an oxygen flow of 10 L/min. In less than two minutes following time, oxygen saturation decreased rapidly to 50%, blood pressure to 70/40, bradycardia of 37 bpm, cyanosis, and rapidly expanding subcutaneous emphysema in the neck and chest wall resulted in AT termination, and the patient developed pulseless electrical activity within 2 minutes from AT termination, and CPR was initiated and reviewed within 30 seconds. The patient was hooked back to the ventilator, and increased peak airway pressure exceeded the mechanical ventilator alarm limits. Hence, manual tidal ventilation through a Bag valve mask (BVM) was initiated. An emergency chest film revealed barotrauma (Fig.1), extensive subcutaneous emphysema, left pneumothorax, and pneumo-peritoneum). Immediate chest tube insertion was done to treat pneumothorax with 28 French (Fr-size) chest drainage catheter (Fig.2) and the patient became stable. Apnea test was repeated to confirm brain death,

which was uneventful. However, the patient died after 72 hours.

Fig.1: Anterior posterior chest radiograph showing ground glass opacity in both mid and lower zones of the lung peripheral predominance

Fig. 2: Anterior-posterior (AP-View) chest radiograph showing bilateral subcutaneous emphysema, left pneumothorax with intercostal drainage tube insertion, and pneumoperitoneum

DISCUSSION

Although the institute with state-of-the-art facilities had a good perspective to manage SAH, the death rate in SAH is still high. Nearly 10 % of SAH patients die before hospital arrival and 25 % of SAH patients die during the first 24 hours. The prime etiology of death is due to high ICP, acute hydrocephalus, re-bleeding, vasospasm, intracerebral hemorrhage, myocardial ischemia, cardiac arrhythmias, pulmonary oedema, and respiratory failure (8). Respiratory apnea signifies brain stem failure (9). The apnea test is the standard conventional technique utilized to support brain death diagnosis and identify potential organ donors. Serious complications during AT procedure may happen, facilitating poor organ perfusion and restricting organ retrieval for transplantation. Before AT procedure, our patient met the AAN prerequisite criteria, such as no spontaneous respirations, no evidence of residual paralytics, normothermia of > 36.5C, systolic blood pressure of > 100 mmHg, corrected diabetes insipidus, absence of severe acid-base, electrolyte, endocrine abnormality and PO2 of >200 mmHg after preoxygenation (10). Goudreau *et al.,* determined the frequency of AT complications and their predisposing factors through 145 Apnea test procedures in 121 patients. His study concluded that complications occurred during the AT procedures is 26%. In particular, Hypotension (24%) is the most significant complication that arises due to hypoxemia, and the most common predisposing factors are insufficient preoxygenation and acidosis (11). Also, investigators from another study reported complications of acidosis (68%), hypoxemia (23%), and Hypotension (12%) during the AT procedure in 129 patients (12). In a Canadian multicenter study, 77 BD patients were classified into two groups. Group one receives O_2 insufflation via a catheter positioned inside the ETT, and group two receives O_2 insufflation via an Airway maintenance breathing unit (AMBU) with a CPAP valve attached to the ETT; at the conclusive of the study, there was no significant disparity noticed in the PO2 decrease, PCO2 rise, or pH reduction between two groups (13). However, the American Academy of Neurology documented strategies to prevent or minimize hypoxemia in BD through guidelines of "Practice Parameters" recognized as apneic oxygenation, which comprises the delivery of high-flow oxygen into the ETT using a suction (oxygen) catheter placed at the carina level (14). Although high-flow O_2 insufflation (should be<10 L/min) can decrease the rate of CO2 accumulation and lengthen the test, it is also an important factor in determining the tracheal airway pressure, which can result in barotrauma (15). Thus, we decided to keep O2 insufflation at 8lpm through the catheter via ETT to prevent hypoxia and barotrauma. Despite this, we encountered immediate serious complications of surgical emphysema and pneumothorax, followed by Hypotension, bradycardia, and cardiac arrest in the next two minutes, which led to AT termination to provide immediate medical management. AT terminations due to complications may vary from 1.6 to 4.8% of BD patients. Su *et al.,* stated that among 550 BD patients, 50.7% of AT procedures were completed successfully, and 42.0% of AT procedures have been terminated due to hemodynamic instability (16). Similar to our case, GadBar *et al.,* documented a case with a history of intracerebral hemorrhage who developed tension pneumothorax, Hypotension, and bradycardia followed by cardiac arrest when an oxygen catheter at the carina level during the apnea test (17). A few studies limited to case reports reported the AT pitfalls and suggested a few clinical implications to regulate the procedural methods (18). Also, there is personal information on the occurrence of medical complications during pre-and post-AT. Nevertheless, prospective studies are lacking in this clinical background. The most common cause of iatrogenic barotrauma during mechanical ventilation is an underlying lung disease, whereas in our case the patient had no history of lung disease. Therefore, we presume that our patient's short neck with greater neck circumference, MallamPati score of 4, and position of

the endotracheal tube (deep) at the level of carina was the main cause of immediate surgical emphysema and pneumothorax, and it became noticeable less than minutes after suction catheter (oxygen) insertion in ETT. Furthermore, the explanation for this complication might be that the suction catheter went beyond the distal end of the ETT. Thereby, distal trachea or the left main bronchus perforation had happened. Also, the deep ETT insertion and routine ETT suctioning might traumatize our patient's airways, thus causing them to perforate during AT by the blunt suction catheter. A few case reports were published with identical complications. However, this complication mechanism varies from the other published case reports. In the two cases, Bar-Joseph *et al*. stated that in their two cases bradycardia and hypotension incident occurred after 1-2 minutes of oxygen diffusion, and they concluded that tension pneumothorax developed as the result of enormous air trapping outside a cannula that was obstructing the airway (19). Similarly, Marks and Zisfein also encountered subcutaneous emphysema and thoracic inflation while examining the apneic oxygenation diffusion method to prevent hypoxemia (20). Likewise, Saposnik *et al.,* demonstrated that after 2 minutes, oxygen diffusion during apneic oxygenation resulted in pneumothorax and pneumoperitoneum headed by cardiac arrest (21). In contrast, in our case onset of subcutaneous emphysema happens less than minutes after insertion of the oxygen cannula, which makes this mechanism significantly different from other studies. To our acquaintance, this is the first written report on a short neck with greater neck circumference associated with surgical emphysema and pneumothorax during AT. The pathophysiological of this complication is uncertain. However, two potential possibilities might predispose to this complication, such as

1. Size of the endotracheal tube
2. Depth of the endotracheal tube
3. The duration gap between brain death diagnosis and apnea test

In addition, from our experience, we strongly suggest that a short neck with complex airway anomaly associated with greater neck circumference is a potentially high-risk factor for barotrauma. We also strongly suggest an inspection of a recent chest x-ray for ETT level from carina and endotracheal tube level from incisor before the AT procedure, and we also recommend performing ECG before AT. Finally, we strongly discourage apneic oxygenation by inserting a suction (oxygen) catheter through ETT. In contrast, T-piece and continuous positive airway pressure (CPAP) apneic oxygenation methods can be Safer and more effective methods of providing oxygen insufflation. Moreover, author Levesque et al. supported our statement that using a T-piece with a CPAP valve was more influential in minimizing the lessening of PO2 than the conventional O_2 catheter method (22).

CONCLUSION

The apnea test is a complex procedure. Despite the recommended guidelines, complications may occur. Therefore, AT providers should be cautious and skilled. Prospective studies are recommended for these groups of patients to address their safety guidelines and demonstrate the frequency of complications occurring during AT procedures.

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CONFLICT OF INTEREST

Nil.

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