

# A Curious case of Vasovagal Shock following Transesophageal Endobronchial Ultrasound-Guided Needle Aspiration (EUS-B-NA)

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**Abstract.** Esophageal ultrasound-guided bronchoscopic needle aspiration (EUS-B-NA) is a relatively safe procedure with rare complications. A vasovagal attack after EUS-B-NA has not been reported to date. Usually benign and self-limiting, it can cause refractory bradycardia and sudden cardiac arrest. Timely intervention reduces morbidity and mortality. Here, we report a novel case of vasovagal attack after EUS-B-NA and was managed successfully. Management includes identifying the triggering event and keeping the patient in the Trendelenburg position. Atropine is reserved for refractory cases. ([www.actabiomedica.it](http://www.actabiomedica.it))

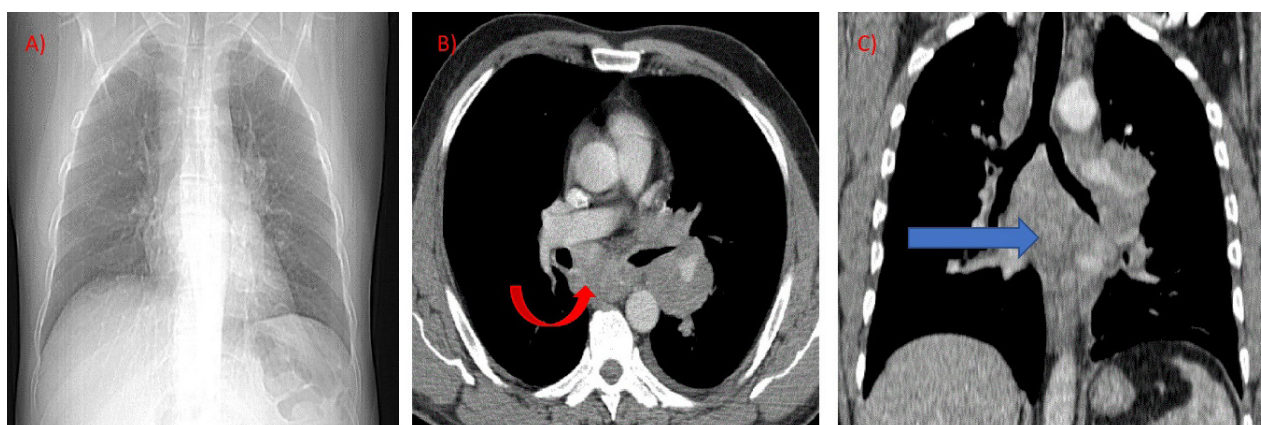
**Keywords:** EUS-B-NA, vasovagal, bradycardia, atropine

## Introduction

The endobronchial ultrasound (EBUS) scope is now being used by Pulmonologists in the esophagus for the sampling of mediastinal lesions (EUS-B-NA). It is increasingly utilized in the diagnosis of mediastinal lymphadenopathy and the staging of lung cancer. EUS-B-NA makes a more complete assessment of the mediastinum possible and increases diagnostic yield when added to endobronchial ultrasound-transbronchial needle aspiration (EBUS-TBNA) (1). Comparing with EBUS-TBNA, EUS-B-NA is usually a well-tolerated procedure under conscious sedation, requiring lower doses of sedatives and anaesthetics and is associated with less cough and oxygen desaturation (2). Therefore, EUS can be a better procedure in patients with poor cardiorespiratory function (3). It is safe and no serious complications have been reported yet (1). Here, we report a fatal complication after EUS-B-NA and was managed successfully.

## Case History

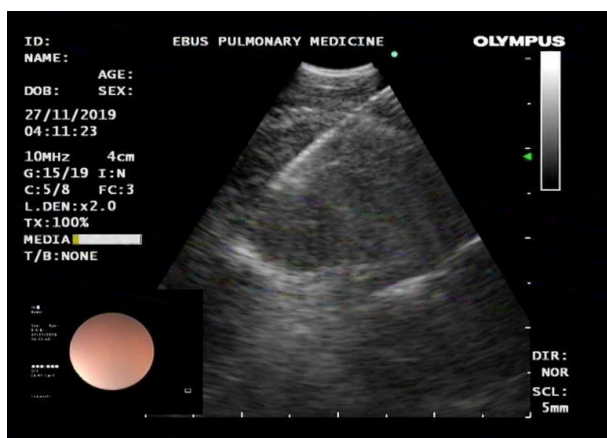
A 55-year-old male, ex-smoker with no previous co-morbidities was admitted to the hospital with complaints of progressive shortness of breath, cough with minimal expectoration, and chest discomfort for the last three months. Chest radiograph showed mediastinal widening (Figure 1A). Contrast-enhanced computerized tomography (CECT) of the thorax revealed a heterogenous mediastinal mass (Figure 1B and 1C). White light Bronchoscopy (WLB) and EBUS-TBNA were planned for the sampling of the lesion. The pre-procedural assessment was normal with stable vital parameters. Lignocaine sensitivity testing (intradermal injection) did not reveal any hypersensitivity and 10% lignocaine spray was used for oropharyngeal anaesthesia. WLB did not disclose any endobronchial abnormality. The patient had a high visual analogue score for cough (VAS- 70mm) during WLB, so a EUS-B-NA was performed with ease under minimal sedation (4). Sampling was done from the para esophageal lymph nodal station (station 8) (Figure 2).



**Figure 1A-C:** 1A. Chest radiograph showing mediastinal widening. 1B. Axial reformatted CT thorax image at the level of carina shows a large mediastinal mass (red arrow). 1C. Coronal reformatted CT image shows a heterogenous mediastinal mass extending below the carina into the paraesophageal LN station (blue arrow)

EUS-B-NA was done by the jabbing method. During the second pass, the patient suddenly developed bradycardia, hypotension, and loss of consciousness. The procedure was abandoned and EBUS scope was immediately withdrawn. Carotid pulsations were feeble, and peripheries became cold. Respiratory rate frequency was 12 breaths/minute. Peripheral oxygen saturation (Spo<sub>2</sub>) was not recordable. Direct laryngoscopy showed no evidence of laryngospasm but the airway was secured given the low Glasgow coma scale with an 8.5mm endotracheal tube. A vasovagal attack was suspected. He was kept in Trendelenburg position and started on intravenous (i.v) crystalloid bolus infusions. There was worsening bradycardia (heart

rate- 20beats/minute) followed by asystole. Atropine 1mg slow i.v bolus was administered and Advanced Cardiac Life Support (ACLS) algorithm was about to be initiated. However, his heart rate picked up immediately after atropine administration and he regained consciousness. After a few minutes, his vitals normalized. Continuous electrocardiogram monitoring during EUS-B-NA and the event showed only sinus bradycardia with no atrio-ventricular dissociation, or ST-T wave changes. He was extubated and kept under observation. Rapid on-site evaluation (ROSE) of FNA smears were diagnostic showing malignant cells. After an exhaustive workup for bradycardia, he was discharged from the hospital and asked to follow up with reports. The patient was advised to report if any future attacks of syncope occur.



**Figure 2.** EUS image depicting a heterogenous mass lesion with EBUS-TBNA needle within the mass at nodal station 8 (paraesophageal)

## Discussion

Esophageal ultrasound bronchoscopic fine needle aspiration EUS-B-NA is a novel, minimally invasive, and relatively safe procedure in which the endobronchial ultrasound EBUS scope is inserted into the esophagus for the sampling of peribronchial masses under real-time guidance. Apart from increasing the diagnostic yield, it is better tolerated by the patients and is extremely safe with no major adverse events. In a systematic review of combined EUS-B-NA and EBUS-TBNA for the mediastinal lymph nodal stag-

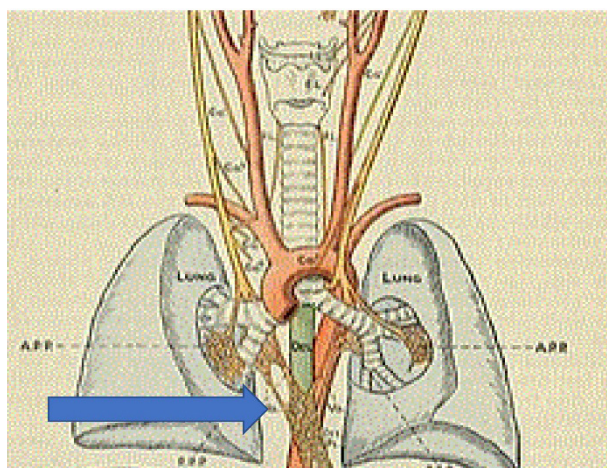
ing in lung cancer, severe complications were noted in only two patients (0.3%) which included lymph node abscess and pneumothorax (5). Other complications like haemorrhage, infection, and perforation of the esophagus have also been reported. Despite an extensive literature search, a vasovagal attack after EUS-B-NA has not been reported.

The vasovagal attack is triggered by anxiety, emotion, pain, and, irritation of the vagus nerve (6). In our case, it is believed to be triggered by iatrogenic stimulation/irritation of the vagus nerve/esophageal plexus during EUS-B-NA due to its anatomical location and proximity with the mediastinal mass. The right and left vagus nerves descend posteriorly in the thorax to their respective pulmonary hila, ramify to form the esophageal plexus just anterior to the esophagus (7) (Figure 3). Ward et al. in their case report attributed vagal nerve irritation as the trigger for the vasovagal attack (8). Similar observations were also made by Fashola and colleagues (6). Further, the hospital anxiety and depression score (HADS) for anxiety evaluation prior to the procedure was 4 (not anxious) and the VAS for pain assessment during EUS-B-NA was 1 on a scale of 10 (no pain), thereby, ruling out anxiety and/or pain as the possible triggers for the vasovagal event (9). The pathophysiology of a vasovagal attack includes bradycardia and vasodilatation decreasing cerebral blood

flow (10). Bradycardia is due to an increase in cardioinhibitory impulses via the vagus nerve to the Sino Atrial (SA) node. Usually self-limiting, occasionally leads to asystole (11). Timely diagnosis and intervention are required

Apart from a vasovagal attack, other factors like electrolyte abnormalities, hypothyroidism, drugs, toxins, and cardiac illness can also predispose to bradycardia. Drug history and toxins were negative in our case. Serum electrolyte levels and thyroid function tests were within normal limits. There was no past history of recurrent syncope or any pre-existing cardiac illness. Chest skiagram radiograph and transthoracic ultrasound did not reveal any pneumothorax and/or pneumomediastinum. Electrocardiogram (ECG) showed sinus rhythm and Doppler echocardiography failed to disclose any abnormality. Procedure-related causes for bradycardia and sudden cardiac arrest include laryngospasm, severe hypoxemia and the use of drugs (sedatives, analgesics, and anaesthetics) for procedural sedation (12). Direct laryngoscopy ruled out laryngospasm and there was no fall in spo2 during the procedure. Midazolam (I.V) was used for sedation and topical lignocaine for local anaesthesia (2ml of 2% lignocaine transtracheal and 2-3 actuations of 10% lignocaine topical spray). Midazolam is a relatively safe short-acting benzodiazepine, known to cause tachycardia and heart rate variability due to vagal tone reduction (drug-induced vagolysis) (13). Lignocaine sensitivity testing, done prior to the procedure did not reveal hypersensitivity. The cumulative dose of lignocaine administered during the procedure was 120mg (1ml of 2% lignocaine equals approximately 20mg + each actuation of 10% lignocaine equals 10mg) which was less than the 3-5mg/kg I.V dose (180-300mg, weight of the patient- 60kg) or 9.6mg/kg topical dose usually required to cause any systemic toxicity (14,15). Besides, the first symptom of Lignocaine toxicity is usually neurological (numbness of the mouth and tongue followed by muscular twitching), and bradycardia with cardiac depression occur only at extremely high doses (16).

Finally, Management of a vasovagal reaction includes keeping the patient in Trendelenburg position (17). The hemodynamic effects of the Trendelenburg position include an increase in cardiac output, systemic



**Figure 3.** Formation of esophageal plexus by the right and left vagus nerve anterior to the esophagus. Adopted from Cunningham text book of Anatomy.1914. Nervous system pg-792 [[File: Cunningham&#039; s Text-book of anatomy (1914) (20628833868).jpg|thumb|Cunningham&#039; s Text-book of anatomy (1914) (20628833868)]] (Wikimedia Commons)



vascular resistance, right atrial pressure due to an increase in venous return (18). The increase in cardiac output helps to counter the depressor vasodilatory response of the vasovagal attack (19). However, not all cases respond to Trendelenburg position and Atropine may be required for refractory cases (11). Atropine antagonizes reflex bradycardia and asystole caused by vagal stimulation due to its vagolytic action (20).

## Conclusion

Although EUS-B-NA is considered an extremely safe procedure for Pulmonologists, our case report highlights a rare life-threatening complication. Due to the anatomy and distribution of vagus nerve and its branches, there could be an increased risk of vasovagal episodes during EUS especially during the sampling of lymph nodal stations 8 and 9. Irrespective of the trigger, early recognition confers better prognosis and survival.

**Conflicts of interest:** Each author declares that he or she has no commercial associations (e.g. consultancies, stock ownership, equity interest, patent/licensing arrangement etc.) that might pose a conflict of interest in connection with the submitted article.

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