

# Naso - Gastric Tube (NGT) Syndrome in a patient with Polycythemia with Stroke in Neuro - ICU

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## Abstract

Nasogastric tube (NGT) insertion is a usual and routine procedure in intensive care units (ICUs) for enteral nutrition (EN) and administering oral medicines. These indwelling tubes are easy to place and usually safe, but rarely can cause serious complications. NGT syndrome is characterized by throat pain and bilateral vocal cord paralysis, which can cause upper airway obstruction in the form of stridor. It is life-threatening condition if not recognized early and treated on time. This is a case report of an elderly man with NGT syndrome and highlight its significance and management in ICUs.

**Keywords:** Nasogastric Tube, Vocal Cord Weakness, Tracheostomy, Percutaneous Endoscopic Gastrostomy

## Introduction

The nasogastric tube can produce sudden, life-threatening bilateral vocal cord paralysis and is often an unrecognized cause of this clinical entity. Although indwelling tubes are easy to place and safe, serious complications can occur. NGT syndrome is characterized by throat pain and bilateral vocal cord paralysis/paresis, which can cause upper airway obstruction (stridor). In this report, a rare case of NGT syndrome in Polycythemia with stroke is described.

## Case Report

An 84 -year-old man was admitted to Neuro-ICU with facial asymmetry and right sided weakness with giddiness since 3 days. Neurological deficit remained unchanged over 3 days. There was no history of headache, vomiting and convulsion. No history of altered sensorium or fall. Patient was a known case of polycythemia (Hb: 20.6 gm %). He had not been previously investigated for the same before admission. He was on tablet. aspirin (75 mg) for polycythemia and had occasional giddiness. He had Deep Vein Thrombosis (DVT) two years back and was treated conservatively with anticoagulant. There was no co-morbid condition like hypertension, diabetes and ischemic heart disease.

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## On Examination

His pulse was 70 /min regular and Blood pressure 140/90 mm of Hg, He was hemodynamically stable with SpO<sub>2</sub> 98 % at room air. Neurologically he had right upper motor neuron facial weakness and right hemi-paresis (MRC grade - upper limb power 3+ and Lower Limb power 4). Patient was fully conscious and oriented and was having difficulty in swallowing for solid as well as liquid. Hence Ryle's tube (Naso-gastric tube) was inserted for feeding as well as giving medicine and condom catheter (Urodrain) applied for urine drainage.

Investigations revealed Hb: 20.6 gm% ( Polycythemia), WBC 8400/mm<sup>3</sup>, Platelet 194k, Fasting blood sugar 99mg%, Liver function and renal function tests were normal, BT/ CT/PT/PTT were normal, PT/ INR-1.04, uric acid 5.9 and Lipid profile were normal. ECG showed LAHB, skiagram chest didn't reveal any abnormality. Hematological opinion sought and investigations were carried out as advised by him. Serum Erythropoetin level - 3.3 (N: 5.4 to 31 miu/ml), JAK 2 mutation was not detected. Ultrasound of Abdomen and Pelvis did not reveal any significant abnormality. MRI/ MRA/ DWI revealed wedge shape restricted diffusion defect in left corona radiate suggestive of acute middle cerebral arterial non-hemorrhagic infarct. MRA of brain revealed minor atherosclerotic plaque in internal carotid artery without significant block.

Therapeutic Phlebotomy for removal of (220 ml) blood done for 5 days as per hematologist's advised . Patient's Hb became 15.2 gm%. On 5<sup>th</sup> day of Ryle's tube insertion, patient developed sudden onset development of stridor.

Immediately High flow Oxygen started. Patient's oxygen saturation deteriorated. ENT consultant was obtained. Fiberoptic laryngoscopy revealed aretenoids as well as vocal cords edema (fig.1). Immediately Patient was intubated with C mack laryngoscope with smaller tube followed by tracheostomy. PEG (Percutaneous Endoscopic Gastrostomy) tube inserted on the same day for administration of drugs like aspirin and others in addition to feeding. After 5 days of tracheostomy, fiberoptic laryngoscopy repeated and revealed subsidence of aretenoid as well as vocal cord edema (fig.2). Patient was put on T piece trial which he tolerated well and after ABG report, tracheostomy tube was removed. After 10th day, oral test feed were given which he tolerated well and PEG tube also removed. Patient was discharged after treatment of minor infection at the PEG tube site.

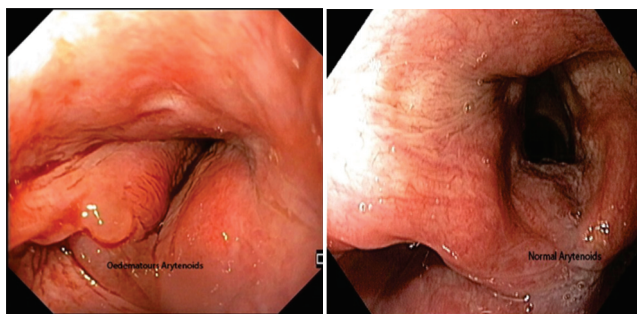


Fig. 1: Oedematous Arytenoids and vocal cords

Fig. 2: After 5 days of tracheostomy, laryngoscopy revealed normal arytenoids and vocal cords

## Discussion

Provision of nutritional support is a crucial component of ICU care and EN is preferred over parenteral nutrition. NGT insertion is simple and minimally invasive and is routinely carried out in critically ill patients. NGT-related complications are not as rare as commonly believed. Most complications are due to mechanical injury of the nose, esophagus, and stomach. Misplacement of the NGT into a pulmonary or intracranial region can also occur in patients with head injuries [1]. NGT syndrome was first named by Sofferman et al. to describe bilateral vocal cord paralysis developed in patients with an NGT in 1990 [2]. Diabetes mellitus and immunocompromised states have been suggested as risk factors for NGT syndrome. In our case, the patient did not have diabetes mellitus. Exact risk factors are not yet established. Brousseau et al [3] also estimated a range of NGT onset from 12 h after intubation to 2 wks after extubation. The time to recovery from respiratory symptoms and vocal cords dysfunction has been reported at 1 day to 3 months [4,5]. Diagnosis is established by bilateral vocal cord paralysis and ulceration of the post-cricoid region [3].

Three pathophysiologic mechanisms were proposed as the cause of NGT syndrome:

- 1) The dynamic and vertical movements of the larynx against the fixed NGT.
- 2) Compression of the cricoid by the NGT and the cervical spine.
- 3) Tonic contraction of the cricopharyngeus muscle pulling the NGT against the postcricoid.

These induce postcricoid esophageal ulceration and myositis of the posterior cricoarytenoid (PCA). As a result, vocal cord abduction is compromised. However; esophageal ulceration is not always present. The key treatment of NGT Syndrome is removal of the NGT or long intestinal tube because it can lead to rapid resolution of respiratory distress. If patient is having acute respiratory distress then tracheostomy and gastrostomy / PEG (Percutaneous Endoscopic Gastrostomy) to be done. Once vocal cord edema subsides, tracheostomy tube can be removed.

## Conclusion

NGT Syndrome should be considered in all patients who present with throat pain, hoarseness, shortness of breath or stridor after a nasogastric or a long intestinal intubation insertion. Early diagnosis can prevent catastrophe. NGT Syndrome requires prompt treatment, such as removal of the tube and tracheotomy, and close follow-up with Laryngoscopy / Bronchoscopy. If patients are diagnosed early, almost all of them will eventually recover.

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