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Life-Threatening Laryngeal Oedema Secondary to Electrolytes Imbalance: Implications for an Otolaryngologist

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Abstract

Introduction: *Electrolytes are essential for normal functioning of every system in the body and their imbalance can have serious consequences. Some of the well known complications include cardiac arrhythmias, neuromuscular instability, myopathy, cerebral oedema, and osmotic demyelination which can prove fatal. However, less commonly known complication arising from electrolyte imbalance is stridor associated with vocal cord paralysis and / or laryngeal oedema. Known risk factors for electrolytes imbalance include old age, diabetes mellitus, diuretics, pure water hydration and gastrointestinal tract losses.*

Aims: *To raise awareness among Otolaryngologists about the importance of electrolyte imbalance that could potentially compromise the airway.*

Methods: *Two case reports with literature review*

Results: *A 60 year old Caucasian female developed proximal muscle weakness associated with a 2 weeks history of diarrhoea. She was found to have hypokalaemia of 1.7 and other electrolytes were deranged as well. She continued to deteriorate and required intubation and ventilation because of respiratory muscles involvement. She failed extubation trial and was noted to have severe endolaryngeal oedema. She required surgical tracheostomy. As the electrolytes were corrected, her muscular weakness and laryngeal oedema improved and she made full recovery.*

A 63 year old Caucasian male presented with stridor and confusion with a preceding 1 week history of sore throat and dysphagia. On examination he had severe laryngeal oedema that required emergency debulking and subsequently he was kept intubated and ventilated. He was found to have hyponatraemia of 112 that was gradually replaced with improvement in his laryngeal swelling.

Conclusions: *Stridor and laryngeal oedema can be the presenting features of deranged electrolytes. Such patients should be looked after in a multidisciplinary team setting involving the Endocrinologists, Intensivists and Otolaryngologists.*

Introduction

Electrolytes are essential for normal functioning of every system in the body and their imbalance can have serious consequences[1]. Some of the well-known complications include cardiac arrhythmias, neuromuscular weakness, and cerebral oedema which can be fatal[1,2,3]. However, a rare complication arising from electrolyte imbalance is vocal cord paralysis and/or laryngeal oedema. The aim of this article is to raise awareness among otolaryngologists, emergency physicians and anaesthetists regarding the potentially severe and life-threatening complication of laryngeal oedema as reported in this case series. Additionally, this article will briefly discuss those electrolytes which are most clinically relevant.

Case Reports

1st Case

A 60-year-old Caucasian female was admitted to the neurology ward with a 2-day history of gradual onset proximal muscle weakness, associated muscle ache and preceding 6-week history of diarrhea. Her muscle weakness progressed to a degree where she was unable to walk. There were no other neurological symptoms. She had a past medical history of a cerebrovascular accident 20 years ago with no residual weakness, hypertension and hypothyroidism. Her medications included bendroflumethiazide, amlodipine, atenolol, citalopram and thyroxine. On neurological assessment of motor power using the oxford scale[4], she had reduced proximal muscles power; hip 3/5, shoulder 3/5, brisk upper and lower limb reflexes with intact sensation, normal muscle tone and normal plantar responses. Cranial nerve examination was unremarkable. Oxygen saturation was 93% on room air and chest auscultation revealed bilateral basal crepitations. The rest of her vital signs were within normal parameters

Initial differentials included Guillain-Barre syndrome, motor neuron disease, myasthenia gravis and motor neuropathy. Blood tests revealed a potassium (K^+) of 2.1 mmol/L, sodium (Na^+) of 135 mmol/L, creatine kinase (CK) of 508 U/L, with a normal full blood count (FBC), glomerular filtration rate (eGFR), thyroid and liver function tests(LFT's, TFT's). Initial arterial blood gas (ABG) test revealed pH of 7.5, PCO2 of 5.9 kPa, a PO2 of 10.5 kPa, and oxygen saturation of 94% on 3 liters of oxygen. Electrocardiogram (ECG), showed flattened T-waves in lateral chest leads. A chest x-ray revealed a raised right hemi diaphragm.

Subsequently, a lumber puncture was performed, and analysis of the cerebrospinal fluid was unremarkable. This was after cross sectional imaging revealing no concerning features.

Overnight, the patient's condition worsened and a repeat ABG showed type I respiratory failure and further deterioration in the serum Potassium (K^+ =1.71 mmol/L), accompanied by low blood calcium and magnesium. Her vital capacity was found to be 0.65 L, alongside a respiratory rate of 30/m with poor respiratory effort and a biphasic stridor. She was transferred to the intensive therapy unit (ITU) for intubation and further care. However, prior to that the ENT specialists were contacted to carry out a nasendoscopic laryngoscopy which revealed marked oedema of the supraglottis, bilateral oedematous arytenoids and false cords. The left true vocal cord (VC) was visible but due to laryngeal oedema the right true VC was not visible with clear subglottic narrowing. Intubation in ITU was then carried out with no difficulties.

The muscle weakness was presumed to be secondary to electrolyte imbalance. During her course of stay in intensive therapy unit (ITU) she developed Staphylococcus Aureus positive pneumonia which was successfully treated with flucloxacillin. As multiple attempts of extubation failed, direct laryngoscopy was carried out in theatre that showed persistent marked laryngeal oedema. For completion laryngeal biopsies were taken for histopathology and a tracheostomy was performed. The biopsies were subsequently reported to be benign. Her ventilatory support was gradually weaned off. Her serum electrolytes were continuously monitored and showed marked improvement with intra-venous electrolytes replacement. She was then transferred to the high dependency unit and finally to a medical ward. Successful de-cannulation was carried out on day 10 post insertion. As her serum electrolyte levels returned back to normal, the proximal muscles weakness and the laryngeal oedema both completely resolved.

2nd Case

A 63-year-old Caucasian male presented to the emergency department with stridor, difficulty in breathing and confusion, with a preceding 1-week history of a sore throat and dysphagia. With a background history of gastroesophageal reflux disease, chronic obstructive pulmonary disease,

hypercholesterolaemia, and alcohol abuse. On clinical examination, the patient had biphasic stridor on breathing with transmitted upper respiratory sounds on chest auscultation. His medications included salbutamol inhaler, tiotropium, carbocysteine, simvastatin and lansoprazole.

He was commenced on IV steroids, humidified oxygen and an adrenaline nebuliser. Initial serum investigations showed, a serum Na^+ level of 117 mmol/L. At this point his Lansoprazole was withheld and the patient was admitted for further work up. Later on, in the day the patient became more confused and developed an increased respiratory effort and rate. ABG was performed showing acidosis; PCO2 of 6.5 kPa, PO2 of 11 kPa and a base excess of 3.3 mmol/L. ENT advice was sought at this stage as the stridor had become more pronounced; and marked glottis oedema was noted on flexible nasendoscopy. After discussion between both the ENT specialist and anaesthetic team on call, the safest approach agreed was to carry out invasive ventilation followed by subsequent direct laryngoscopy in theatres. Intra operative findings included severe laryngeal oedema, associated with slough and unilateral VC polypoidal lesion. Debulking of the oedematous laryngeal lining was performed, a biopsy was taken and sent for histopathology which later revealed a benign pathology of no significance.

Post operatively patient was transferred to the ITU. During the first day in ITU, repeat serum investigation revealed, hyponatraemia of 112 mmol/L. Due to this finding, the patient was started on 1.8% NACL for Na^+ replacement according to local trust protocol and placed under strict monitoring. The target was to increase Na^+ by 0.5 -1 mmol/L/hour to achieve the desired level of 126 mmol/L. Further investigations were carried out to rule out the cause of the hyponatraemia. The significant past medical history of alcohol abuse placed this patient at a higher risk of central pontine myelinolysis. Due to this, greater attention was paid to Na^+ replacement. On day 6 of his ITU admission, the patient's Na^+ was 136 mmol/l and he was successfully extubated. The patient continued to make good recovery and was discharged from the hospital shortly after ITU discharge.

Discussion

Electrolyte imbalance associated laryngeal oedema is a novel medical condition with unexplained exact underlying pathogenesis in the current literature. Most common causes of laryngeal oedema include intubation, hereditary angioedema and pregnancy[5, 6, 7]. To our knowledge these are the first cases reporting electrolyte imbalance as a direct cause of laryngeal oedema. Contemporary literature mainly discusses metabolic stridor in the context of vocal cord paralysis, as opposed to laryngeal oedema[8,9].

A case of laryngeal oedema and concomitant hyponatraemia has been previously described by Wegmüller et al[10]. The authors report a case of 45-year-old healthy woman undergoing a hysteroscopic myomectomy who developed severe laryngeal oedema secondary to irrigating fluid over-absorption and resultant dilutional hyponatraemia. However, in our series laryngeal oedema developed in the presence of hyponatraemia without hypervolemia. This suggests that, electrolyte imbalance must have a major role in causing laryngeal oedema. Detailed below is a brief account of some of the electrolytes discussed in the case series:

Potassium

Potassium is predominantly found within intracellular fluid at a concentration of between 140 to 150 mmol/L[10]. Within extracellular fluid, potassium is found at a concentration of 3.5 to 5.0 mmol/L. Potassium has a key role in excitability of nerve and muscle cells[11]. A rapid change in potassium levels can result in life threatening consequences. Changes in serum pH can affect serum potassium concentration, and therefore should be considered when treating potassium abnormalities. Common dietary sources for potassium include milk, potatoes, cereal, meats, vegetables and fruits[12].

Sodium

Sodium is a major intravascular ion that is essential for cellular homeostasis and fluid balance[13, 14]. Plasma sodium concentration narrowly ranges from 135 to 145 mmol/L in healthy individuals[15]. Acute shifts in serum sodium concentration can result in free water shifts into or out of the vascular space[13]. An acute fall in serum sodium concentration will result in the transfer of water from the vascular space into the interstitial space. In contrast, an acute rise in serum sodium concentration will result in the transfer of water from the interstitial space to the vascular space. In terms of managing sodium disorders, the American Heart Association suggests gradual correction of sodium

concentration, as rapid correction has been found to be associated with central pontine myelinolysis and cerebral bleeds[13].

Magnesium

Magnesium is the fourth most abundant mineral within the human body[16]. Around 60% of magnesium is present in bone, 20% in skeletal muscle, 19% in other soft tissues, and 1% in extracellular fluid[17]. Normally, serum magnesium concentration ranges from 1.3 to 2.2 mmol/L[13]. However, it is important to note that serum magnesium concentration does not indicate total body magnesium stores, as extracellular magnesium is typically bound to serum albumin. Magnesium's main function involves regulating the movement of other electrolytes, such as potassium, calcium and sodium, into and out of cells. Due to this reason, serum magnesium concentration is linked to serum potassium, calcium and sodium concentrations.

Calcium

Calcium is the most abundant mineral found within the human body[13]. 50% of calcium within blood plasma are bound to proteins, mostly albumin, and the rest are active and free to carry out their biological functions[18]. Free serum calcium concentration ranges from 2.1 to 2.6 mmol/L and is regulated by 3 main hormones; parathyroid hormone, vitamin D, and calcitonin[19]. Calcium is involved in many different functions within the human body including enzymatic reactions, muscle contractions, platelet function, cardiac contractility, as well as bone strength and neuromuscular function[13]. Total serum calcium concentration is directly related to serum albumin concentration, however free serum calcium concentration is inversely related to serum albumin concentration. Due to this, in the presence of hypalbuminaemia total serum calcium concentration may appear low, however free serum calcium concentration may still be within the normal range. It is also important to note that calcium has the ability to antagonize both magnesium and potassium at the plasma membrane. Therefore, calcium can be utilized to treat the symptoms and signs of hypermagnesaemia and hyperkalaemia.

Conclusions

Deranged electrolytes including hyponatremia and hypokalemia can cause serious airway issues by inducing laryngeal oedema and impaired vocal cord mobility. It is important to promptly identify any abnormality in plasma electrolytes in patients presenting with stridor so that appropriate measures can be instituted in a timely fashion. Such patients should be looked after in a multidisciplinary team setting, involving the Endocrinologists, Intensive care specialists and Otolaryngologists.

References

[1] Balcij AK, Koksal O, Kose A, Armagan E, Ozdemir F, Inal T, Oner N. General characteristics of patients with electrolyte imbalance admitted to emergency department. World J Emerg Med [Internet]. 2013 [cited 2020 Jun 14]; 4(2); 113-116. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4129840/

[2] Knochel JP. Neuromuscular manifestations of electrolyte disorders. Am J Med [Internet]. 1982 Mar [cited 2020 Jun 14]; 72(3); 521-535. Available from: https://www.sciencedirect.com/science/article/pii/0002934382905228

[3] Diringer M. Neurological Manifestations of Major Electrolyte Abnormalities. Handb Clin Neurol [Internet]. 2017 [cited 2020 Jun 14]; 141; 705-713. Available from: https://pubmed.ncbi.nlm.nih.gov/28190443/

[4] Naqvi, U., 2019. Muscle strength grading. In Statpearls [Internet]. StatPearls Publishing.

[5] Wittekamp BHJ, NKA van Mook W, Tjan DHT, Zwaveling JH, Bergmans DCJJ. Clinical review: Post-extubation laryngeal edema and extubation failure in critically ill adult patients. Crit Care [Internet]. 2009 Dec [cited 2020 Jun 14]; 13(6); 233. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2811912/

[6] Farkas H. Management of upper airway edema caused by hereditary angioedema. Allergy Asthma Clin Immunol [Internet]. 2010 Jul [cited 2020 Jun 14]; 6(1); 19. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2920238/

[7] Procter AJM, White JB. Laryngeal oedema in pregnancy. Anaesth [Internet]. 1983 Feb [cited 2020 Jun 14]. DOI: 10.1111/j.1365-2044.1983.tb13948.x. Available from: https://pubmed.ncbi.nlm.nih.gov/6829893/

[8] Moralee SJ, Reilly PG. Metabolic stridor: bilateral vocal cord abductor paralysis secondary to hypokalaemia? J Laryngol Otol [Internet]. 1992 Jan [cited 2020 Jun 14]; 106(1); 56-57. Available from: https://pubmed.ncbi.nlm.nih.gov/1541894/

[9] Lye WC, Leong SO. Bilateral Vocal Cord Paralysis Secondary to Treatment of Severe Hypophosphatemia in a Continuous Ambulatory Peritoneal Dialysis Patient. Am J Kidney Dis [Internet]. 1994 Jan [cited 2020 Jun 14]; 23(1); 127-129. Available from: https://pubmed.ncbi.nlm.nih.gov/8285187/

[10] Wegmüller B, Hug K, Buenzil CM, Yuen B, Maggiorini M, Rudiger A. Life-Threatening Laryngeal Edema and Hyponatraemia during Hysteroscopy. Crit Care Res Pract [Internet]. 2011 Mar [cited 2020 Jun 14]; 2011; 140381. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3085301/

[11] Thier SO. Potassium Physiology. Am J Med [Internet]. 1986 Apr [cited 2020 May 23]; 80(4A); 3-7. Available from: https://pubmed.ncbi.nlm.nih.gov/3706350/

[12] Stone MS, Martyn L, Weaver CM. Potassium Intake, Bioavailability, Hypertension, and Glucose Control. Nutrients [Internet]. 2016 Jul [cited 2020 May 23]; 8(7); 444. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4963920/

[13] ECC Committee, Subcommittees and Task Forces of the American Heart Association. 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular care. Circulation [Internet]. 2005 Nov [cited 2020 May 23]; 112(24); IV-1 – IV-5. Available from: https://www.ahajournals.org/doi/full/10.1161/CIRCULATIONAHA.105.166550?url_ver=Z39.88-2003&rfr_id=ori:rid:crossref.org&rfr_dat=cr_pub%20%200pubmed

[14] Farquhar WB, Edwards DG, Jurkovitz CT, Weintraub WS. Dietary Sodium and Health: More Than Just Blood Pressure. J Am Coll Cardiol [Internet]. 2015 Mar [cited 2020 May 25]; 65(10); 1042-1050. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5098396/

[15] Reynolds RM, Padfield PL, Seckl JR. Disorders of sodium balance. BMJ [Internet]. 2006 Mar [cited 2020 May 25]; 332(7543); 702-705. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1410848/

[16] Alawi AMA, Majoni SW, Falhammar H. Magnesium and Human Health: Perspectives and Research Directions. Int J Endocrinol [Internet]. 2018 Apr [cited 2020 May 27]; 9041694. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5926493/

[17] Swaminathan R. Magnesium Metabolism and its Disorders. Clin Biochem Rev [Internet]. 2003 May [cited 2020 May 27]; 24(2); 47-66. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1855626/

[18] Lian IA, Asberg A. Should total calcium be adjusted for albumin? A retrospective observational study of laboratory data from central Norway. BMJ Open [Internet]. 2018 [cited 2020 May 29]; 8(4); e017703. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5892769/

[19] Fong J. Hypocalcemia. Can Fam Physician [Internet]. 2012 Feb [cited 2020 May 28]; 58(2); 158-162. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3279267/

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