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RESEARCH

Case study: lessons from a laryngeal abscess

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This case report focuses on a patient who presented with respiratory distress and with a background history of asthma. The underlying pathology is explored, as well as the diagnostic error that could have resulted in serious complications.

Keywords: asthma, error, laryngeal abscess, squamous carcinoma

Introduction

This case serves as a stark reminder of the importance of a clinical examination, the wisdom in thinking further when initial treatment fails, and the dangers of laryngeal disease. Although not common, laryngeal abscesses may be more frequently encountered with the increasing incidence of tuberculosis and immunocompromised patients.

Case report

A 27-year-old woman, previously known to suffer from asthma, presented to a peripheral district hospital with a history of collapsing at home, and with difficulty breathing. While en route to the peripheral hospital, she suffered a generalised tonic-clonic seizure. The only other history noted at the time she presented was that she had taken some herbal medication for her asthma. Exactly what she had taken was never ascertained. On presentation she was noted to be tachypnoeic (60 breaths per minute) and tachycardic (126 beats per minute), with a silent chest and marked use of accessory respiratory muscles. She had a Glasgow Coma Scale (GCS) score of 10, with a normal blood pressure, no added heart sounds, and the rest of the examination was unremarkable. Initial blood tests showed a white cell count (WCC) of 17.1 x 10^9/l as the only significant abnormality. At the peripheral district hospital she received treatment, including hydrocortisone, nebulised beta-2 agonists, antibiotics, and a theophylline infusion. However, she continued to deteriorate. At this stage the attending doctors were considering only an acute asthma attack or herbal intoxication (organophosphate poisoning). An arterial blood gas at this point revealed a severe acidaemia with a pH of 7.0, pCO_2 that was very high and unrecordable, and a pO_2 of 20.0 kPa (FiO_2 unknown). The patient's condition improved temporarily, but after failing to respond sufficiently to treatment over a five-hour period, she suddenly deteriorated. This prompted the medical team to attempt intubation and mechanical ventilation. The referral letter to the receiving regional hospital noted that this intubation was difficult and an abnormal larynx was visualised during the procedure. Three doctors attempted intubation and eventually a size 5.5 endotracheal tube (ETT) was secured. After intubation the arterial blood gas (ABG) showed a profound respiratory acidosis with a pH 7.12, pCO_2 15.9 kPa, pO_2 6.3 kPa (FiO_2 unknown), bicarbonate of 25 mmol/L, and a base excess of 0.8 mmol/L.

No hypoxaemia or hypoglycaemia was documented during the inter-hospital transfer, although was seemingly possible. The patient arrived at the regional hospital emergency department in the early hours of the morning. Examination revealed a haemodynamically stable patient with a heart rate of 111, blood pressure of 139/80, respiratory rate of 18, oxygen saturation of 99% (FiO_2 of 0.4), and serum glucose of 7.4 mmol/L. The ABG showed the following: pH 7.519, pO_2 15.5 kPa, and pCO_2 6.4 kPa. She did not have any respiratory signs and on auscultation her chest was clear. She was sedated (morphine and midazolam infusions) with a GCS of 8T (M4E4VT). The correct placement of the ETT was confirmed and she required minimal ventilatory support (ventilator settings: pressure assist control mode, FiO_2 0.4, pressure support 6cmH_2O, PEEP 8cmH_2O). A decision was then made to keep her intubated until the day duty doctors arrived, despite the apparent rapid improvement in bronchospasm. The critical care team was consulted later that morning. It was noted at this stage that the rapid improvement and ventilator settings, together with the history of a difficult intubation and abnormal laryngoscopy, was not in keeping with her suggested life-threatening bronchospasm and alternative diagnoses were suggested. She was immediately transferred to theatre for an examination under anaesthesia.

In theatre she underwent a gas induction, received short-acting opioids, and a non-depolarising muscle relaxant was administered after visualisation of the supra-glottic area. This was achieved with a C-Mac® video laryngoscope (Karl Storz, Tuttlingen, Germany). The theatre had been prepared for a more difficult airway situation, with other intubating aids, a jet-ventilator system, and the neck was prepared for an emergency tracheostomy with an ENT surgeon scrubbed and ready with a theatre nursing sister present. On examination a left supraglottic laryngeal abscess draining pus from the left aryepiglottic fold was noted. Granulation tissue was seen over the left false and true cords. There was no retropharyngeal or parapharyngeal abscess seen. There was also no abnormality seen in the subglottic area or trachea, although significant oedema was noted in the region of the abscess. The pus from the abscess was drained. We then decided to remove the ETT to view the trachea. The trachea was normal on bronchoscopy and the patient could not breathe due to the laryngeal oedema. Thus the decision was taken to insert a size 6 tracheostomy. She was transferred to the intensive care unit where she recovered well on intravenous antibiotics. Within days she was well enough to be transferred back to a general ward. There were no obvious sequelae noted as a consequence of the seizure.

Further investigations revealed that she was HIV negative, glucose monitoring remained normal, and her ANA and rheumatoid factors were within normal limits. Tuberculosis could not be identified or cultured and syphilis serology was normal. The laryngeal biopsies confirmed a well-differentiated keratinising squamous carcinoma with stromal invasion. The oedema of the patient's larynx took three weeks to settle before allowing a clinical staging of her laryngeal carcinoma. On re-examination with a flexible scope, her left hemi-larynx appeared fixed with obvious tumour invasion of her entire true cord, ventricle and extension onto her false cord. The difference between visualising an oedematous larynx due to an abscess versus visualising the same larynx three weeks later with a tumour was remarkable. Whilst the larynx was in the infected stage, there were almost no clear clinical signs of the tumour. This highlights the need to perform biopsies of infected tissue. There were no palpable cervical nodes. A CT scan of her neck and chest confirmed no obvious metastasis to her lymph nodes or pulmonary tissue. She was staged as a T3N0M0 squamous cell carcinoma of the larynx. She underwent a total laryngectomy three weeks later. Perioperatively, she did not develop any complications. She has gone through postoperative chemoradiation of six weeks' duration. A second-stage speaking-valve has been inserted and she is currently undergoing speech therapy with reasonable results. She is now nine months postoperative and has not shown any clinical signs of tumour recurrence.

Discussion

An acute laryngeal abscess is rare in the post-antibiotic era. It is a serious condition characterised by rapid deterioration in the size of the laryngeal inlet necessitating urgent airway management. In the pre-antibiotic era, primary laryngeal abscess formation was as a result of a preceding event of catarrhal inflammation, overexertion of laryngeal muscles, trauma due to foreign bodies or accidental cauterisation with alkalis or acids.[1] It was postulated that the abscess develops from an acute submucous laryngitis that immediately involves the perichondrium.[2] The perichondrium separates from the cartilage with abscess formation. As the inflammatory process continues, it either compromises the airway or extends laterally to develop a superficial subcutaneous abscess or fistula.

Secondary laryngeal abscess formation was also described in the pre-antibiotic era as an extension of an infection from the nasopharynx, tonsil or peritonsillar space, or as a complication of measles, scarlet fever, erysipelas, tuberculosis or syphilis.[2]

In the post-antibiotic era, the aetiology of laryngeal abscess formation seems to have changed. The aetiological event is now associated with recent airway manipulation[3,4] and laryngeal malignancies.[5,6] The cases of primary laryngeal infection in the post-antibiotic era are now associated with immunocompromised patients, or, rarely, still as a result of acute sinusitis or tuberculosis.[7–10]

Our case highlights the association between malignancies and laryngeal abscess formation. Marked oedema, erythema and pus draining from a supralaryngeal cavity were the hallmarks of presentation rather than the typical appearance of a laryngeal tumour. Multiple, blind biopsies are recommended in all cases of laryngeal abscess formation. A high clinical suspicion of an underlying malignancy should always be associated with patients with no underlying aetiological factors, as this is now the most common reason why, in the current post-antibiotic era, patients would develop a laryngeal abscess.

Regarding the possibility of infection spreading to the mediastinum, this typically spreads via the retropharyngeal space. In this particular case this area did not show any signs of sepsis clinically. The larynx has no fascial planes that connect with the mediastinum. Thus a deep neck space infection in this patient was not an issue. The patient's WCC peaked at 17.1×10^9/l and normalised within 48 h, with the highest C-reactive protein measured as 58.

Asthma vs. laryngeal abscess

This case also highlights the need for a laryngeal examination for patients diagnosed with an acute asthma attack refractory to medical management. This particular patient presented with signs and symptoms that could have been explained by bronchospasm, except that the larynx and not the small airways were involved, and once again the importance of the clinical examination is highlighted. Clinical signs and symptoms can help to differentiate between laryngeal constriction and bronchospasm. However, the difference between stridor and a chest wheeze can sometimes be difficult to distinguish in a noisy environment. The difficult intubation, especially one requiring a smaller sized lumen endotracheal tube, should also have been viewed with suspicion. Any failure of response to the protocolised treatment of asthma should prompt the analysis of the original decision pathway.

In addition, the diagnostic error that occurred in this case might have been avoided, but again demonstrates the vulnerability of all doctors to this type of error. The process of making a diagnosis includes gathering data, interpretation, and verifying the data and conclusion derived. Each of these steps is prone to error and requires understanding, skill and experience to minimise this risk. As has been described elsewhere, failures in perception, aberrant reasoning, biases and other cognitive states, such as affect, have all been identified as possible contributors to diagnostic error.[11]

Addressing the areas that may lead to diagnostic error is not particularly well instructed in pre-graduate or undergraduate years. However, skills and workshops helping medical personnel identify areas of weakness that make them prone to error, and then providing guidance on how to build strategies to avoid mistakes, should probably be incorporated into current medical school and postgraduate training.

The initial treatment algorithm of this non-responding 'asthmatic' patient should have included continuous nebulisation, and other modalities such as magnesium, ipratropium bromide, ketamine, intravenous salbutamol and eventually an adrenaline infusion. It is unclear whether these were considered or simply not available at the peripheral hospital. In addition, according to the 2013 South African Guideline for the management of acute asthma in adults, it should be noted that antibiotics should only be given in cases of suspected infection, and aminophylline infusions fail after continuous nebulisations, ipratropium bromide, hydrocortisone and magnesium sulphate. In addition, one can argue that the severity of the patient's presentation should have prompted initiation of an earlier referral.

An additional danger to consider in such patients with reactive airways is the intubation and ventilation. In this case it seems unavoidable due to suddenly decompensating. However, the act of placing an ETT may provoke reactive airways, and significant skill and pharmacological knowledge may be necessary to rescue such situations. Inappropriate ventilator settings can also worsen

the situation, with common errors leading to 'stacking' of breaths, increasing plateau pressures and a worsening respiratory acidosis.

Conclusion

Acute laryngeal abscesses are considered infrequent in the current post-antibiotic era. However, immunocompromised patients and those with tuberculosis are more susceptible. With this in mind, we may be encountering this pathology more often in South Africa. Potentially serious airway risk exists and expeditious management needs to be performed. In addition, this case report highlights the dangers of diagnostic error where an unexplained improvement in a patient's condition was not evaluated and considered in the initial diagnosis.

Learning points

1. Consider all possibilities for respiratory distress. Severe asthma that either responds out of proportion to the initial presentation and therapy, or does not respond, should have the diagnosis reconsidered.
2. Acute laryngeal abscess is a potentially life-threatening condition that may be seen more frequently in the future. The present day aetiology is strongly associated with laryngeal malignancy, highlighting the need for blind biopsies.
3. A good understanding, and strategies to decrease the risk for diagnostic errors, should be introduced as mandatory undergraduate and postgraduate training.

Supplementary material

Supplementary material for this article can be accessed at http://dx.doi.org.10.1080/22201181.2015.1068533

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