

Case Series:

**Intraoral Drainage And Neck Exploration of Cervical abscess following Acute Supraglottitis:
A Case Series**

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Abstract

Acute supraglottitis or epiglottitis is regarded as one of the feared pediatric emergency. With the advance in immunization, it is now less frequently seen in the group however there is an increase incidence in adult. Abscess formation following an acute episode of supraglottitis is uncommon but carries a significant morbidity and mortality. We present three cases of acute supraglottitis complicated by deep neck abscess. All of the patients required incision and drainage under general anesthesia with two of them required both intraoral and external approaches to drain the abscess. All of the patients recovered well postoperatively.

Keywords: Supraglottitis; epiglottitis; cervical abscess

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Introduction

Acute supraglottitis or epiglottitis is defined as inflammation of the epiglottis with or without adjacent supraglottic structures, including aryepiglottic fold, arytenoids and occasionally uvula involvement.^{1,2} Acute supraglottitis was previously feared as a pediatric emergency but with the introduction of *Haemophilus influenzae* type b (Hib) vaccination, the incidence has markedly reduced by 90-98%.^{2,3} There was increasing trend among adult.^{4,5} Other studies showed a stable incidence.⁶ The epiglottic abscess and cervical abscess are infrequent sequelae of acute supraglottitis. The incidence of epiglottic abscess in adult is ranging from nil to 29%.⁴ Acute supraglottitis complicated with deep neck spaces abscess formation is a rare occurrence.

Case 1

A 55-year-old male with underlying diabetes mellitus on insulin treatment, presented with three days history of severe sore throat and odynophagia, associated with dysphagia to solid and fluid. The sore throat was so severe that he was unable to

swallow his saliva causing drooling and voice change. He also noticed a submental swelling since the symptoms started. He had no history of foreign body ingestion or any food allergy.

Upon examination at emergency department, he was lethargic looking, dehydrated and having drooling of saliva. There was no stridor and he was not tachypneic. He was unable to talk due to severe pain. He was afebrile, normotensive, not tachycardic and oxygen saturation was 98% under room air. Oropharynx examination was unremarkable. There was a submental swelling measured 3cm x 4cm, which was tender but not inflamed. Flexible nasopharyngolaryngoscopy (FNPLS) revealed a swollen base of tongue, vallecula, epiglottis and arytenoids with pus dribbling from base of tongue area (Figure 1 a). Vocal cords and laryngeal inlet were partially seen.

He was referred to anesthetist and was admitted to ICU for observation. Computed tomography (CT) scan of the neck demonstrated a nearly abscess formation at left supraglottic region. The patient was started on high dose intravenous (IV)

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Ceftazidime 2g every 8-hourly (TDS) and IV Dexamethasone 8mg TDS. The initial total white cells count was 19 and the C-reactive protein (CRP) was 145. However the symptoms and septic parameters were not improved after one-week antibiotic.

A repeat CT scan revealed abscess collection at left supraglottic region with extension into left parapharyngeal space (Figure 1 b). Intraoral drainage of abscess and neck exploration were performed. Intraoperatively there was thick pus collection at vallecula which most likely ruptured locule from the left supraglottic abscess with 4cc pus was drained intraorally. Neck exploration revealed abscess locule at left parapharyngeal space extending to left pretracheal space with 20cc of pus drained. The organism grew from the pus were *Pseudomonas aeruginosa* and *ESBL-producing Klebsiella pneumoniae*. The antibiotic was changed to IV Piperacillin/Tazobactam as per antibiotic sensitivity to both organisms. Postoperatively, patient had hospital acquired pneumoniae and had total admission of one month duration with full recovery.

Case 2

A 46-year-old male with underlying diabetes mellitus and hypertension, presented with three-day history of fever with severe sorethroat and odynophagia. He was unable to take orally and was having voice change. Clinically, he was sitting up but grimacing in pain everytime he tried to swallow his saliva. He was not having stridor and not tachypneic. He was febrile and the blood pressure was 156/83mmHg but he was not tachycardic and oxygen saturation on air was 98%. Oropharynx examination revealed inflamed tonsils and edematous uvula. FNPLS revealed edematous epiglottis, aryepiglottic folds and arytenoids with heavy secretions (Figure 2 a). Vocal cords and laryngeal inlet were partially seen.

Diagnosis of acute supraglottitis was made and patient was started on IV Ceftazidime 1g TDS and IV Dexamethasone 8mg TDS. While he was observed at acute bay in general ward, the condition was not improved. We also noticed left submandibular swelling 3cm x 3cm in size, which was inflamed and tender on day four of IV antibiotic. Thus CT scan of the neck was performed which revealed left hypopharynx abscess extending from the level of C2 until C6, with extension into left supraglottic region and anteriorly involved strap muscles bilaterally (Figure 2 b).

Intraoral drainage and neck exploration were

performed. Intraorally, there was an abscess locule at left infratonsillar extending to left hypopharynx area with 5cc of pus drained. On neck exploration, there was pretracheal abscess with multiple locules of abscess found anterior to thyroid cartilage. Pus culture from intraoral was no growth and pus from neck grew *Staphylococcus aureus* which was sensitive to Ceftazidime. The patient had to undergo-exploration and wound debridement one week after the primary operation due to persistent pus discharge from wound. He also was intubated for 12 days due to poor weaning to ventilator. He recovered well and was discharged home after 29 days stay in hospital.

Case 3

A 52-year-old male with underlying diabetes mellitus on oral hypoglycemic agent, presented to emergency department with two-day history of fever with dysphagia and change of voice. He was unable to swallow his saliva and unable to lie flat due to feeling breathless. He also noticed a left neck swelling since the symptoms started. Clinically, he was sitting up, not tachypneic and no stridor. He was febrile and tachycardic with pulse rate of 140 bpm, but the blood pressure was normotensive and oxygen saturation was 96% under room air.

The oropharynx examination was unremarkable. There was left submandibular swelling 3cmx2cm which was firm and tender on palpation of the neck. FNPLS revealed edematous epiglottis covered with sloughy tissue (Figure 3 a). The rest of supraglottic structures were inflamed but not markedly edematous. Laryngeal inlet was partly seen. His initial TWC was 24.8 and the CRP was 198.3.

He was observed at acute bay in general ward with anesthetist review in ward and was started on IV Ceftazidime 1g TDS and IV Dexamethasone 8mg TDS. A repeated FNPLS after 2 days of antibiotic and steroid showed no improvement thus CT scan neck was performed. CT neck showed a swollen epiglottis with collection at pre-epiglottic space with minimal extension to left submandibular space (Figure 3 b). Direct laryngoscopy and intraoral drainage of abscess was performed. Intraoperatively, the lingual surface of epiglottis was bulging and fluctuant with pus 3cc drained after incision was made. Postoperatively, he was admitted to ICU and extubated after 24 hours. He was discharged well on day four post operation. The pus culture was no growth.

Discussion

Acute supraglottitis can result from infection, allergy or trauma, which causes inflammation of the supraglottic structures leading to edema and vascular engorgement. It can rapidly manifest as an acute upper airway obstruction. There are two forms of acute supraglottitis which are the classical paediatric disease caused by *Haemophilus influenzae* which presents with acute airway obstruction; while the adult form which is associated with oropharyngeal inflammation and is less likely to cause acute airway obstruction.⁹ In other words, the adult acute supraglottitis is a different entity to paediatric counterpart. In all of our cases, all of the patients were not in respiratory distress. The typical presentations of an acute supraglottitis are fever with severe sore throat, odynophagia and dysphagia (which can be out of proportion and contradicting with oropharynx examination findings), dysphonia and drooling of saliva.¹⁻⁵ More than 90% of patients have normal oral cavity and oropharyngeal examination.² Additionally, patient presented with acute supraglottitis symptom and also neck swelling or localized tenderness in the neck should raise a suspicion of an abscess formation.

The gold standard for diagnosis of acute supraglottitis requires FNPLS examination, which necessitates a referral to otorhinolaryngologist. The common precaution for acute supraglottitis in children was to avoid examination and stimulate the larynx further to avoid acute spasm and obstruction. This complication is especially high risk if indirect or rigid laryngoscopy is done. However, performing FNPLS was reported as a safe procedure in adult acute supraglottitis because the supraglottic structures can be viewed with precaution not to induce contact with the laryngeal structures, provided the patient is not in respiratory distress.¹⁻⁷ Relying only on the 'thumb sign' and 'vallecula sign' of the lateral neck radiograph might miss this potentially life threatening condition as the sensitivity of the lateral neck radiograph was only about 75%.^{1,2,4} If clinical suspicion of acute supraglottitis is high, even with negative lateral neck radiograph, the patient should be referred for laryngoscopic examination. Obtaining a radiograph in the availability of endoscopic assessment is a waste of valuable time in the management of this potentially critical airway emergency.¹ However, we suggest that, obtaining lateral neck radiograph in district hospital setting without otorhinolaryngologist may help in the initial diagnosis prior to referral.

Ideally, a patient diagnosed with acute supraglottitis is monitored under anaesthetist care in acute bay of general ward, or preferably in the ICU, due to impending airway compromise. Airway intervention is still controversial, but some studies suggest clinical criteria to predict the eventual airway outcome. Friedman et al classified the respiratory distress into 4 stages (Table 1),⁸ and most studies suggest that artificial airway intervention is taken for Friedman II to IV.^{9,11} Another study classified patient into high risk and low risk group to decide on airway intervention. Patient with diabetes mellitus, immunosuppression, and visualization of less than 50% of glottic aperture, stridor, drooling and onset of symptom less than 24 hours are under high risk group and need more vigilance in airway management.¹ Katori and Tsukuda utilize scope classification (SC) to predict airway management of patients with acute supraglottitis (Table 2).¹⁰ They found that symptoms of stridor and muffled voice, severe epiglottic swelling (SC III), extension to arytenoids (SC B), rapid clinical course of less than 24 hours and diabetes mellitus are the factors that significantly associated with airway intervention. As for Katori and Tsukuda, their preferred airway intervention for the above criteria is tracheostomy under local anesthesia without attempting intubation to avoid worsening epiglottic edema.

Our first two cases met the criteria of SC II with SC B, while the third case had SC II with SC A. All of the three cases were in Friedman stage I of respiratory distress. All of the patients were intubated using glide scope by anaesthetist (with fiber optic intubation trolley and rigid bronchoscopy guided intubation trolley standby) in operation theatre prior to general anesthesia for operative procedure and not for airway intervention. The intubation for Case 1 and Case 2 were difficult where the airway only can be secured after third attempt by a senior anaesthetist. The intubation for Case 3 patient was relatively easy whereby he was able to be intubated after first attempt by a registrar. In our center, we still prefer intubation as the first step for airway intervention. We suggest a close observation for patient with SC II and below with SC A or B in Friedman I. While a difficult intubation procedure (glide scope, fiber optic intubation and rigid bronchoscopy guided intubation) is suggested when securing airway for surgical intervention for patients with SC II and above with SC A or B in Friedman I. Additionally, an emergency airway

intervention using a difficult intubation procedure with emergency tracheostomy on standby should be done in patients with SC III and above with SC A or B in Friedman II to IV.

Resolution of symptom and improvement of supraglottic edema on scope findings are two most important clinical evidences of response to the treatment given. We performed FNPLS once every 2 days to monitor the supraglottic edema although some authors suggest daily inspection.² We also found benefits in serial evaluation of CRP and TWC to monitor progress of the patient and predict any sequelae or complications. As in our cases, the poor clinical response to antibiotic with rising TWC and CRP has raised the suspicion of abscess formation. These clinical, scope and biochemical evaluations are the reliable predictors and guide to surgeon whether the particular patient need further evaluation with CT scanning or benefits with conservative management. A prospective study in South Korea suggested that CT should be done in all patients with a diagnosis of supraglottitis for early diagnosis of an epiglottic abscess.¹² However, in our setting, CT neck is used as a diagnostic tool of epiglottic or cervical abscess only when the patient did not improved clinically and/or endoscopic monitoring, after

trial of high dose broad spectrum antibiotic and worsening biochemical findings that an abscess formation is suspected.

As we learned from this case series, patient with supraglottitis with epiglottic abscess should undergo drainage of abscess intraorally with or without neck exploration depending on the extension of the abscess formation under general anesthesia. There are some reports on needle aspiration of an epiglottic abscess under local anesthesia and also conservative management with IV antibiotic without surgical intervention, both with good clinical outcome.^{13,14}

Conclusions

Diagnosis of adult acute supraglottitis requires high index of suspicion in patient that presented with severe sore throat and odynophagia with negative oropharynx examination. Prompt recognition of this potentially fatal condition leads to early management of airway compromise and improve the overall outcome of the patient. Sequelae of acute supraglottitis such as abscess formation should be suspected in acute supraglottitis patients that fail to improve with IV antibiotic and steroid.

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Figure 1 (a) Case 1 scope finding showed swollen valleculla, epiglottis and arytenoids with pooling of saliva



Figure 1 (b) Case 1 CT scan neck abscess collection at left supraglottic region with extension into left parapharyngeal space



Figure 2 (a) Case 2 scope finding showed edematous epiglottis, aryepiglottic folds and arytenoids into left supraglottic region

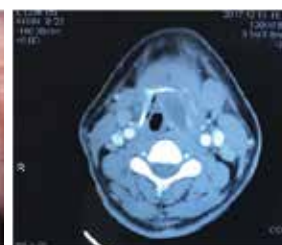


Figure 2 (b) Case 2 CT neck finding left hypopharynx abscess with extension into left supraglottic region.



Figure 3 (a) Case 3 scope finding showed edematous epiglottis covered with slough



Figure 3 (b) Case 3 CT neck finding swollen epiglottis with collection at pre-epiglottic space extending to left submandibular space

Table 1 The Friedmann classification of respiratory distress in acute supraglottitis

Stage I	No respiratory complaints Respiratory rate <20/min
Stage II	Subjective respiratory complaint Respiratory rate >20/min
Stage III	Moderate respiratory distress Stridor, retractions, perioral cyanosis PCO ₂ >45mm Hg Respiratory rate >30/min
Stage IV	Severe respiratory distress Stridor, retractions, cyanosis, delirium, decreased consciousness Respiratory arrest

PCO₂, Arterial carbon dioxide pressure

Table 2 Katori and Tsukuda grading on scope findings

Grade	Findings
SC I (slight swelling)	Minimal swelling of epiglottis and the entire length of the vocal folds can be seen on scope.
SC II (moderate swelling)	The epiglottis swelled moderately and more than half of the posterior vocal folds can be seen on scope.
SC III (severe swelling)	The epiglottis swelled severely and only less than half of the posterior vocal folds can be seen on scope.
SC A	No arytenoids swelling
SC B	Swelling involving arytenoids

References

1. Riffat F, Jefferson N, Bari N, McGuinness J. Acute supraglottitis in Adults. *annals of Otolaryngology, Rhinology & Laryngology*. 2011;120(5):296-299.
 2. Glynn F, Fenton JE. Diagnosis and management of supraglottitis (Epiglottitis). *Current Infectious Disease Reports*. 2008;10:200-204.
 3. Gulfred LA, Lyhne D, Becker BC. Acute Epiglottitis: epidemiology, clinical presentation, management and outcome. *The Journal of Laryngology & Otology*. 2008; 122:818-823.
 4. Ng HL, Sin LM, Li MF, Que TL. Anandaciva S. Acute Epiglottitis in adults: a retrospective review of 106 patients in Hong Kong. *Emergency Medical Journal*. 2008;25:253-255.
 5. Berger G, Landau T, Berger S, et al. The rising incidence of adult acute epiglottitis an epiglottic abscess. *American Journal of Otolaryngology*. 2003; 24(6): 374-383.
 6. Hafidh MA, Sheahan P, Keooh I et al. Acute Epiglottitis in adults: a recent experience with 10 cases. *The Journal of Laryngology & Otology*. 2006;120:310-313.
 7. Ira M, Preyra I, Fernandes CM et al. Adult epiglottitis: a five-year retrospective chart review in major urban centre. *Journal of Canadian Association of Emergency Physician*. 2005;7(6): 387-391.
 8. Friedman M, Toriumi DM, Grybauskas V, Applebaum EL. A plea for uniformity in the staging and management of adult epiglottitis. *Ear Nose Throat Journal*. 1988;67:873-870.
 9. Mayo-Smith MF, Spinale JW, Donskey CJ, Yukawa M, Li RH, Schiffman FJ. Acute epiglottitis: An 18-year experience in Rhode Island. *Chest* 1995;108:1640-1647.
 10. Katori H, Tsukuda M. Acute epiglottitis: analysis of factors associated with airway intervention. *The Journal of Laryngology and Otology*. 2005;119(12):967-973.
 11. Chan K, Pand Y, Tan K. Acute epiglottitis in the tropics: is it an adult disease? *The Journal of Laryngology and Otology*. 2001;115 (9): 715-718.
 12. Lee Y, Kim T, Eun Y. Routine computerised tomography in patients with acute supraglottitis for the diagnosis of epiglottic abscess: is it necessary? - a prospective, multicentre study. *Clinical Otolaryngology*. 2013;38(2):142-147.
 13. Hindy J, Novoa R, Slovik Y, Puterman M, Joshua B. Epiglottic abscess as a complication of acute epiglottitis. *American Journal of Otolaryngology*. 2013;34(4):362-365.
 14. Gillett D, Eynon-Lewis N. Supraglottitis and abscess formation. *The Journal of Laryngology & Otology*. 2010;125(01):99-102.
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