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# Spontaneous Tonsillar Hemorrhage Presenting in an Elderly Patient: A Case Report, Implications and Literature Review

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## Abstract

Spontaneous tonsillar hemorrhage (STH) is a rare event, especially in the post-antibiotic era. The most common causes include acute and/or chronic tonsillitis with subsequent ulceration and tonsillar vessel erosion. Children and young adults are most commonly affected. We report a unique case of a 72 year old female presenting with STH in the absence of any history or clinical findings of acute or chronic tonsillitis. The patient underwent an emergent tonsillectomy due to difficulty of direct visualization of the site of hemorrhage at the bedside. Histopathological evaluation demonstrated acute and chronic inflammation of the removed tonsil with surface ulcerations extending into parenchymal vessels. To our knowledge, these histological findings in a patient without clinical tonsillitis have not yet been described in patients with STH. Differential diagnosis of STH should include infectious mononucleosis, measles, carotid pseudoaneurysm or other major vessel source, increased venous pressure from congestive heart failure, malignancy and coagulopathies. STH from a peripheral tonsil vessel can be successfully managed with local intervention, however, preference should be given toward early tonsillectomy. This case illustrates and discusses a rare and potentially life threatening presentation of tonsillar hemorrhage and its implication in the elderly.

## Keywords

tonsil; hemorrhage; ulcerations; tonsillitis

## Introduction

Infectious and inflammatory disease of the tonsils remains a significant cause of morbidity in the pediatric and young adult population [1,2]. Spontaneous tonsillar hemorrhage (STH) is a rare, but a serious complication of the tonsils that was occasionally reported in the pre-antibiotic era. These early reports were primarily associated with infectious mononucleosis [3], however fatal hemorrhage from major vessel erosion such as the carotid artery as a result of deep neck infections were also reported [1,4,5,6,7]. Since the advent of antibiotics, major vessel bleeding is extremely rare [4]. More recently, reported cases of STH have been associated with peripheral or supplying blood vessels from inflammation and erosion of tonsils secondary to acute and/or chronic tonsillitis [6].

The incidence of STH has been reported at approximately one percent of those affected with tonsillitis [4,8,9]. However, even with the relatively common presentation of acute and/or chronic tonsillitis in today's population, STH is a rarely reported event [10]. Children and young adults are most commonly affected, however, STH may also present in older adults and the elderly. Until this case

presentation, the oldest reported patient who suffered from STH was in their early sixties, and the cause being clinical acute tonsillitis [11]. It is important to note that this patient, as well as other reported patients presenting with STH secondary to tonsillitis have displayed recognizable signs and symptoms of tonsillitis including sore throat, tonsillar erythema, exudates or tonsillar crypts [11]. We describe a unique presentation of STH in a 72 year old female who underwent an emergent tonsillectomy with histopathological evidence of acute and chronic tonsillitis, who furthermore, had no history or physical exam findings of clinical tonsillitis.

## Case Presentation

A 72 year old Caucasian female presented to a local emergency department with a chief complaint of oral bleeding. A large bag of bloody paper towels accompanied the patient which were used to collect the bleeding on the way to the hospital, signifying that the bleeding had been persistent. She denied any recent trauma, sore throat, cough or fever. There was no prior history of bleeding from the oral cavity. She also denied any prior episodes of tonsillitis, tonsilloliths or strep throat. The patient did have a recent three-day admission at the hospital for acute exacerbation of congestive heart failure and was discharged one day prior to the onset of oral bleeding. During that admission, she was given subcutaneous enoxaparin, an anticoagulant, for deep vein thrombosis prophylaxis and was also started on a daily baby aspirin. She had been taking NSAIDs for chronic pain for many months, and denied any easy bruising or prior bleeding from medications.

On physical exam, tonsils were symmetric and 1+ in size, without erythema, crypts or exudates. No obvious tonsillar masses or gross ulcerations were observed. A blood clot was noted in the posterior-superior pole of the left tonsil. After the blood clot was suctioned at the bedside, continuous bleeding was detected originating from the posterior surface of the tonsil and draining inferiorly. The focal location could not be adequately identified due to the posterior location of the hemorrhage. A bedside flexible nasopharyngolaryngoscopy confirmed that the bleeding was originating from the tonsil parenchyma posteriorly and demonstrated no suspicious masses, lesions or ulcerations in the nasopharynx or oropharynx.

Due to the severity and duration of the hemorrhage, as well as the posterior location of the bleeding which could not be directly visualized and managed at the bedside, a decision was made to perform an emergent tonsillectomy in a controlled setting in the operating room. Intraoperatively, active bleeding was noted to be originating from the left tonsil at its posterior-superior location. Tonsillectomy using electrocautery was successfully performed without complications or significant intraoperative blood loss. The patient was admitted for monitoring of any further bleeding and was discharged home the following day. One week later at her follow-up appointment there was no recurrence of oral bleeding.

Histopathological specimen of the involved tonsil tissue measuring 3.5 x 2.1 x 1.2 centimeters demonstrated lymphoid hyperplasia and acute and chronic inflammation. Epithelial ulcers were present which extended into small parenchymal vessels, however, larger vessels deep to the tonsil parenchyma appeared uninvolved (Figures 1,2,3). No malignancy was identified.

## Discussion

Spontaneous tonsillar hemorrhage was defined by Griffies*et al.* in 1988 as continuous bleeding for

greater than one hour, or bleeding greater than 250 mL irrespective of duration [4,9,12]. Fortunately, serious cases of life-threatening bleeding are rare [13]. In several reports, the incidence of STH in patients with acute or chronic tonsillitis is about one percent [4,8,9]. STH has never been described occurring in a patient in their 70's; majority of the reported cases of STH have been in children and young adults [1,14]. Not only was our patient much older than the typically described patient presenting with STH, she also didn't present with signs or symptoms such as sore throat, halitosis or tender lymphadenopathy which would indicate tonsillitis. Additionally, she had no history of tonsillitis or exam findings such as tonsilliths or tonsillar crypts which would suggest chronic inflammation. It would be possible to overlook the most common cause of STH, that being acute or chronic tonsillitis, if it presents in a patient with no signs, symptoms or history of tonsillitis, especially in a patient outside the expected age range.

It's speculated that tonsillitis may progress to STH as a results of microscopic and macroscopic ulcerations in the epithelial layer and subsequent extension into and rupture of the prominent tonsillar vasculature [4,14]. Acute inflammatory response is also thought to increase blood flow to the tonsils with secondary edema and vascular congestion [2]. Studies using xenon-131 clearance technique have shown significantly increased blood flow to inflamed tonsils [1]. The increased vascularity is anticipated to be the contributing factor in hemorrhagic tonsillitis [1]. Figures 2 and 3 demonstrate increased vascularity of the tonsillar tissue surrounding the ulceration craters, which suggests tonsillitis in our patient. Furthermore, histological examination in previous reported cases of STH secondary to tonsillitis have demonstrated tonsillar lymphoid hyperplasia and surface ulcerations involving underlying vasculature [8,10,12]. This also coincides with the histological findings in our patient, however, what clinically separates our patient from other reports was the absence of signs and symptoms which would allow the clinician to suspect tonsillitis as an underlying contributing factor. Based on these findings, acute and/or chronic tonsillitis, at least on a histopathological level, may be present in patients with no clinical complaints, signs or history of tonsillitis. This raises questions about the implications of histological tonsillar inflammation in patients with no clinical evidence of tonsillitis. Does histological tonsillar inflammation in a patient with normal physical examination represent a clinically irrelevant process which is inherent to all normal tonsils, or does it signify an underlying disease process? As part of a system of mucosa-associated lymphoid tissue (MALT) found in the lungs, nasal cavity, gastrointestinal tract, salivary glands and skin, underlying tonsillar inflammation could potentially indicate inflammation of these associated systems [15]. This association has not been studied in depth.

It's important to consider that the prophylactic enoxaparin and baby aspirin administered a couple days prior to the onset of STH may have increased the risk hemorrhage in this patient. The elimination half-life of subcutaneous enoxaparin is reported to be approximately 3-4 hours and would be expected to have cleared the patient's system from the time she was last administered the dose to the onset of her oral hemorrhage approximately 36 hours later [16]. If there is concern for residual systemic enoxaparin or toxicity, the anticoagulant activity of enoxaparin can be monitored by measuring anti-factor Xa assay. Given that the platelet count and coagulation studies were normal (Table 1), question arises whether the patient also had an undiagnosed acquired or inherited platelet disorder which affected the function of the platelets. Being aware that acute and chronic tonsillitis presents with increased vascularity to the tonsil tissue which may further be complicated by surface ulcerations, unrecognized functional platelet disorders may lead to a scenario suitable for tonsillar hemorrhage.

Therefore, the etiology of tonsillar hemorrhage may include coagulopathies such as von Willebrand disease, factor IX deficiency, idiopathic thrombocytopenic purpura, as well as a result of coagulopathic medications.

Another possible cause of spontaneous tonsillar hemorrhage to consider in this patient is secondary to increased venous pressure as a result of congestive heart failure. Palatine tonsils drain into the local pharyngeal venous plexus which ultimately drains into the internal jugular vein via the lingual and pharyngeal veins. It's known that internal jugular vein distension occurs as a result of increased venous pressure in patients with congestive heart failure [17]. Although the correlation between congestive heart failure and spontaneous tonsillar hemorrhage has not been reported in the literature, it's plausible that increased venous pressure in the internal jugular vein can lead to blood engorgement in the pharyngeal venous plexus and induce tonsillar bleeding, especially in a patient who has histological evidence of tonsillar ulcerations in proximity of parenchymal vasculature. Differential diagnosis of STH should also include infectious mononucleosis, measles, carotid pseudoaneurysm or other major vessel source, and malignancy [7,10,11]. A thorough history and physical examinations as well as laboratory evaluation are therefore necessary to determine the underlying cause of STH.

The standard treatment modality of STH has yet to be defined secondary to the rarity of STH and the paucity of published reports. Shock and airway concerns, if present, must be addressed by the appropriate resuscitative measures, and the origin of the hemorrhage identified [5]. Management of STH may initially consist of local control including silver nitrate cautery or adrenaline soaked pledgets if the source of the bleeding is easily identified and access is adequate [1,6,9]. However, the blood supply to the tonsils is not directly visible on exam, therefore unless the bleeding is from a visible surface vessel, localization requires removal of the tonsil [10]. In the presence of a pulsatile tonsillar mass, peritonsillar abscess or other deep space neck infections, arteriography is recommended to rule out major vessel erosion [5,11]. Consideration should be given toward early tonsillectomy due to the potential life-threatening nature of a bleeding tonsil and risk of recurrence. Recurrent STH after conservative measures were taken have been reported, concluding that tonsillectomy is the definitive mode of treatment [12]. Complications of STH in an elderly patient may include blood-loss anemia leading to hypovolemic shock, respiratory failure secondary to aspiration, and ischemic stroke. These are significant events in patients who may already have several comorbid conditions or poor functional status. In this case, emergent tonsillectomy was performed as a means of controlling the acute hemorrhage which was not directly visualized at the bedside. The benefits of tonsillectomy outweighed the risk of possible exsanguination and aspiration, and deemed to be a safe and effective treatment option.

## Conclusion

This case illustrates a unique presentation of a rare but potentially dangerous complication of tonsillitis. Further reports of interest include determining the effects of coagulopathic medications and platelet function in patients with tonsillitis to determine risk factors for tonsillar hemorrhage. Examining the clinical importance of 'subclinical' tonsillitis and the possible association with a systemic MALT-associated inflammation may also be of interest.

## Figures

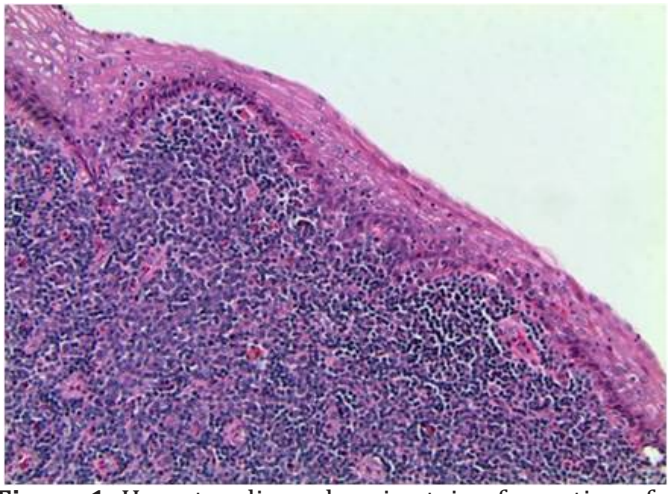

**Figure 1:** Hematoxylin and eosin stain of a section of the left tonsil showing normal stratified squamous epithelium without ulcerations.

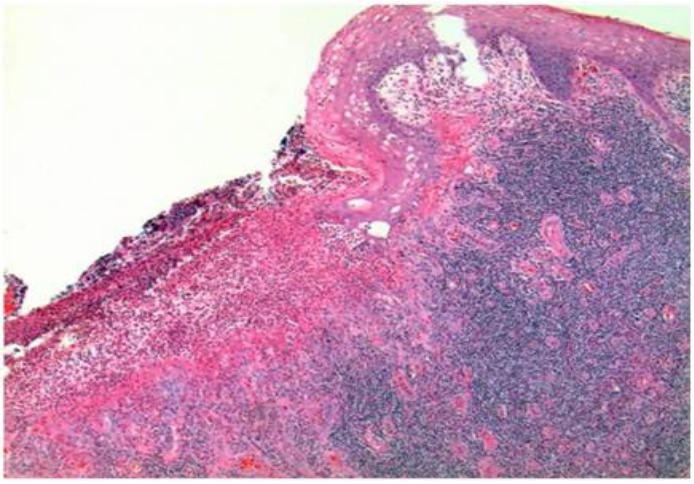

**Figure 2:** Hematoxylin and eosin stain of a section of the left tonsil showing an ulcer crater involving the epithelium and extending into the parenchyma of the tonsil. Note absence of stratified squamous epithelium at the ulcer site.

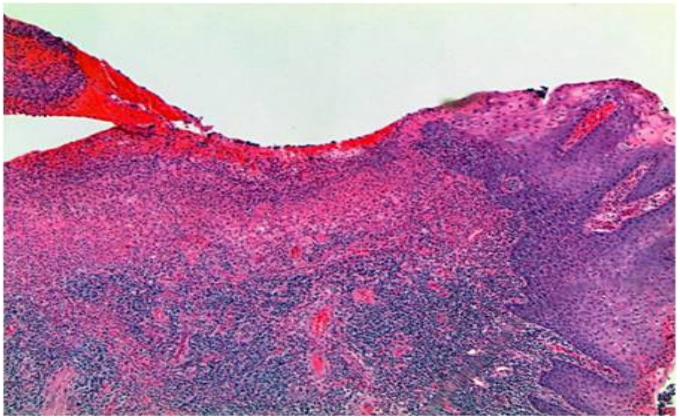

**Figure 3:** Hematoxylin and eosin stain of a section of the left tonsil showing an ulcer crater involving the epithelium and extending into the parenchyma of the tonsil. Note increased vascularity (pink/red).

## Table

**Table 1. Blood count and coagulation profile**

| Blood Parameter | Patient | Reference Range |
|---|---|---|
| White Blood Cells ($10^9$/L) | 8.1 | 4.3 - 11.0 |
| Hemoglobin (g/dL) | 9.5 | 11.6 - 15.9 |
| Hematocrit (%) | 30.6 | 35.7 - 47.8 |
| Platelet Count ($10^3$ mL) | 270 | 170 - 422 |
| Prothrombin Time (s) | 15.7 | 10.5 - 15.7 |
| Partial Thromboplastin Time (s) | 26.3 | 20.6 - 39.2 |
| International Normalized Ratio (INR) | 1.2 | 0.7 to 1.3 |

**Table 1:** Summary of the laboratory results for the patient. Low hemoglobin and hematocrit were noted but platelet count and coagulation profile were normal. Blood differential and chemistry profiles were also within normal limits.

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