The effect of silver nitrate on nasal septal cartilage

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Abstract

Epistaxis from the anterior septum is frequently treated with a topical application of silver nitrate, which cauterizes the bleeding vessel. However, this treatment causes a septal perforation in a small percentage of patients. We report our study of the histologic effect of topical silver nitrate on samples of septal tissue obtained from 11 patients. We found that 30 seconds of exposure allowed silver nitrate to penetrate to a depth of approximately 1 mm. Longer exposure (45 and 60 sec) resulted in no significant additional penetration. Similarly, the amount of silver nitrate deposition into the chondrocytic lacunae did not vary significantly with the length of exposure. On the other hand, the depth of deposition into the extracellular matrix was positively associated with the duration of exposure. We found no direct evidence that silver nitrate exerted any damaging effect on septal cartilage. Instead, the development of septal perforations in patients who receive topical silver nitrate may be attributable to necrosis of the septal cartilage following damage to the overlying perichondrium, from which it derives its blood supply.

Introduction

The prevalence of epistaxis is approximately 10%, and the age distribution is bimodal, peaking first in the late teens and early 20s and later at 45 to 65 years of age.1 In most cases, bleeding originates in the nasal septum, particularly at the anastomosis of the nasopalatine, greater palatine, and anterior ethmoid arteries (Little’s area, Kiesselbach’s area). This origin is particularly common in the younger patients. In some cases, bleeding originates in the lateral nasal wall.2

The use of topical agents in the nose is one of the most diverse treatment options. In The Paradise of Wisdom (ca. AD 850), Ali ibn Rabban al-Tabari published one of the earliest commentaries on the treatment of epistaxis with topical agents.3 Over the centuries, many substances have been used to treat epistaxis; two of the more unusual have been salt pork4 and tonsillar tissue.5

Topically applied silver nitrate has been shown to be particularly efficacious in halting epistaxis.6-9 The first description of silver nitrate for epistaxis in the modern literature was published by Littel in 1932,6 although its use undoubtedly predates that report.9

In an aqueous environment, topical silver nitrate acts as a strong oxidizing agent, stimulating the production of free radicals and cauterizing tissue. Today, it is usually administered via a stick applicator; because the anterior nasal septum is easily accessible, this method of delivery is particularly convenient.

Despite the fact that application of topical silver nitrate is widely used, few data have been published regarding its histologic effects on nasal structures. In particular, there is no objective indication as to how long it should be applied to achieve hemostasis without jeopardizing the perichondrial blood supply to the cartilage. The aim of our investigation was to identify the histologic effects of silver nitrate on nasal septal cartilage after different lengths of exposure.

Materials and methods

We obtained samples of nasal septal cartilage from 11 consenting patients who had undergone septoplasty for nasal obstruction. Each specimen was divided into three sections, and each section was placed into contact with an applicator that delivered a combination of 75% silver nitrate and 25% potassium nitrate (Bray Health & Leisure; Oxfordshire, England) for 30, 45, and 60 seconds. After each application, specimens were immediately washed with normal saline. They were then fixed in 10% formalin, and paraffin sections of each specimen were taken and stained with hematoxylin and eosin (H&E). The specimens were then assessed with the assessor blinded to the duration of silver nitrate contact. Each specimen was examined microscopically to establish:

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• the depth of silver nitrate penetration through the septal cartilage
• the presence and extent of silver nitrate deposition in both the chondrocytic lacunae and the extracellular matrix; the amount of deposition was graded as slight (≤2 deposits per lacuna), moderate (3 to 6 deposits), or heavy (≥7 deposits)
• the appearance of the chondrocytic nuclei

Results
Of a total of 32 evaluated specimens, 11, 10, and 11 were exposed to silver nitrate for 30, 45, and 60 seconds, respectively. Silver nitrate deposition was clearly identified in all specimens as a brown staining of the cartilage with a meniscus base. Although the brown staining appeared to be homogenous on light microscopy, golden-brown particulate deposits were also observed.

Examination of the 11 specimens exposed for 30 seconds showed that the average penetration of silver nitrate was approximately halfway through the depth of the cartilage; the average depth of penetration was not significantly different in the specimens that had been exposed for 45 and 60 seconds (figure 1). Specifically:

• Among the 11 specimens exposed for 30 seconds, the mean depth of penetration was 0.94 mm (range: 0.4 to 2.0).
• Among the 10 specimens exposed for 45 seconds, the mean depth was 1.07 mm (range: 0.6 to 1.7).
• Among the 11 specimens exposed for 60 seconds, the mean depth was 1.05 mm (range: 0.5 to 1.5).

Figure 1. Photographs show the depth of silver nitrate penetration in representative specimens at 30 seconds (A), 45 seconds (B), and 60 seconds (C) (original magnification ×4).

Figure 2. Photomicrographs show the deposition of silver nitrate in a representative chondrocytic lacuna (A) and an extracellular matrix (B) (original magnification ×40).
Besides the homogenous brown staining of cartilage by silver nitrate, particulate deposits were identified in the chondrocytic lacunae and extracellular matrix (figure 2). In the former, deposits were seen in all 32 specimens; on average, the degree of deposition was moderate. No differences were seen in the pattern or amount of deposits in the lacunae in all specimens.

By contrast, particulate deposits in the extracellular matrix could be localized to the superficial one-third, the middle one-third (two-thirds deep), and the full thickness of the stained area. The depth of extracellular matrix deposits after 30 seconds of exposure was evenly split between the superficial one-third and the middle one-third; with longer exposure, almost all deposits settled beyond the superficial one-third (table 1).

The intensity of the extracellular matrix deposits was primarily slight or moderate (table 2). There was a slight trend toward an increase in the amount of deposition with prolonged exposure.

In all specimens, chondrocytic nuclei exhibited smudging, but no difference was seen in the numbers of affected chondrocytes.

Perichondrial tissue that contained blood vessels was identified in one specimen from the 30-second group and in two each from the 45- and 60-second groups. Brown staining was observed in the blood vessels, but given the limited number of such samples, further assessment was not possible. The depth of penetration was similar in those sections of cartilage that were covered by perichondrium, suggesting that the perichondrium does not act as a barrier to silver nitrate penetration.

**Discussion**

To achieve hemostasis of a bleeding nasal septal vessel, only the mucosa requires cautery. Nevertheless, the underlying perichondrium and septal cartilage are also affected by cautery, which might result in a septal perforation. Younger and Blokmanis found that nasal cautery was the causal factor in 7% of septal perforations. Very few nasal septa exposed to silver nitrate cautery become perforated. Other complications of silver nitrate application to septal cartilage include hypersensitivity reactions, asymptomatic argyremia, and tattooing secondary to silver deposition within the mucosa.

In our study, the mean depth of silver nitrate penetration was approximately half the thickness of the specimens regardless of the duration of exposure. Notably, the presence of perichondrium did not appear to act as a barrier to this process. Therefore, it appears that exposure for even fewer than 30 seconds would result in significant deposition of silver nitrate.

We found no evidence that silver nitrate deposition in itself compromises cartilage viability. Particulate deposits were observed in chondrocytic lacunae. Given the widespread use of topical silver nitrate for epistaxis and the infrequency of septal perforation, this treatment may not have a long-term effect on chondrocyte viability. However, it is possible that the smudging of the chondrocytic nuclei that we observed represented early damage to the chondrocytes. In light of this and the depth of penetration, it would appear to be sensible to pay heed to the widely practiced precaution of avoiding bilateral cautery over the same area.

Our study was carried out on nonviable cartilage denuded of its mucosa and, in most cases, its perichondrium. The development of septal perforation may depend on the extent of injury to the perichondrium from which the blood supply to the underlying cartilage is obtained. In addition, local factors such as the presence of atherosclerosis of the nasal vasculature may be important. We plan further studies with perfused septal cartilage in order to investigate the dynamic and long-term effects of silver nitrate application.
These studies will include an investigation of the long-term effects on the structure and function of cartilage associated with the tendency (although small) of extracellular matrix deposition to increase in intensity and depth according to the duration of silver nitrate exposure.

References
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