Treatment of Hereditary Hemorrhagic Telangiectasia–Related Epistaxis

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KEYWORDS
- Hereditary hemorrhagic telangiectasia (HHT)
- Osler-Weber-Rendu
- Epistaxis
- Septodermoplasty
- Laser photocoagulation
- Young’s procedure
- Bevacizumab

KEY LEARNING POINTS
At the end of this article, the reader will:
- Know how to diagnose a patient with HHT and discover the key examination findings.
- Know what additional workup is required in a patient with HHT.
- Understand the Epistaxis Severity Score and how it is used.
- Know the nonsurgical options for treatment of mild or moderate epistaxis related to HHT.
- Know the surgical options available for treatment of HHT.
- Understand the role of Avastin (bevacizumab) in treatment of HHT.

Video content accompanies this article at http://www.oto.theclinics.com

INTRODUCTION
HHT is a rare, autosomal dominant disease with prevalence of 1:5000 characterized by formation of multiple mucocutaneous telangiectasias as well as formation of AVMs within the pulmonary, cerebral, and gastrointestinal vasculature. Patients are particularly prone to formation of telangiectasias within the sinonasal mucosa, and recurrent, spontaneous epistaxis is the most common symptom at time of presentation. More than half of patients with HHT will develop troublesome epistaxis by the third decade of life, and severity of epistaxis increases with age. More than 90% of
patients with HHT experience recurrent epistaxis at some point in life. Severity and frequency of epistaxis varies widely between patients, from mild, occasional epistaxis to severe, life-threatening nosebleeds. In general, severity of epistaxis increases with age.¹

**ETIOLOGY**

<table>
<thead>
<tr>
<th>What causes formation of telangiectasias and AVMs?</th>
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<tr>
<td>• Mutations in genes associated with transforming growth factor-beta (TGF-β) superfamily signaling pathway</td>
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<tr>
<td>• Dysregulation of vascular endothelial tissue remodeling results in weakened integrity of vessel wall leading to formation of telangiectasias and AVMs</td>
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<tr>
<td>• Three gene mutations have been identified: endoglin (ENG), activin receptor-like kinase (ACVRL1 or ALK1), and MADH4</td>
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Various members of the transforming growth factor (TGF)-β superfamily have been implicated in the pathogenesis of HHT. Remodeling of the vascular endothelium within mucosal vessels occurs in a dysregulated fashion, leading to loss of elasticity and dilation of arteriole-venule communications. As a result, fragile and thin-walled telangiectasias form within the nasal cavity in regions with high airflow prone to dryness or repeated mechanical trauma. Telangiectasias tend to congregate along the anterior septum, head of inferior turbinates, anterior lateral nasal wall, and anterior nasal floor. Recurrent and spontaneous epistaxis results from traumatic rupture of the ectatic vessel wall lacking contractile and elastic elements. Elevated plasma levels of vascular endothelial growth factor (VEGF) are present in patients with HHT, which has provided rationale for treatment with VEGF inhibitors in certain cases.²

Two distinct mutations account for 90% of cases of HHT: ENG mutation is known at HHT1, and ACVRL1 mutation is known as HHT2.³ Another gene, MADH4 (mothers against decapentaplegic homolog 4) has been implicated in both juvenile polyposis and a small proportion of cases of HHT.⁴ Recently, mutation in bone morphogenetic protein-9 (BMP9) has been described as resulting in a vascular anomaly syndrome with phenotypic similarity to HHT.⁵

Distinct phenotypic variations have been described in HHT1 and HHT2. Patients with HHT1 are more likely to present with epistaxis earlier in life as well as pulmonary AVMs. Patients with HHT2 are more likely to develop hepatic AVMs.⁶
The diagnosis of HHT is based on various clinical and physical examination criteria known as the Curacão criteria. The Curacão criteria (see Table 1) were established in 2000 by an expert consensus panel, consisting of 4 different clinical criteria: recurrent and spontaneous epistaxis, mucocutaneous telangiectasias, visceral AVMs, and family history. Three positive criteria are necessary for a “definite” diagnosis, whereas 2 criteria allow for “possible/suspected” diagnosis. Diagnosis is “unlikely” with only 1 criterion present.7

Genetic testing may also provide a definite diagnosis of HHT, and standard testing for ENG and ACVRL1 mutation is available. Current genetic testing may not be positive in all patients with HHT due to presence of an unrecognized genetic mutation. Routine genetic testing, therefore, is not recommended in patients with suspected HHT. Expert guidelines have established indications for testing in specific situations, such as prenatal screening, index cases within a family, and establishing or excluding a definite diagnosis in members with few symptoms in a family with known HHT.7

### EVALUATION

**What are characteristic examination findings in patients with HHT?**

- Multiple mucocutaneous telangiectasias involving skin (face, fingertips), lips, oral cavity (hard palate), tongue, and sinonasal cavity
- Differential diagnosis includes CREST syndrome (calcinosis, Raynaud disease, esophageal dysmotility, sclerodactyly, and telangiectasia), essential telangiectasia, ataxia-telangiectasia
- Telangiectasias vary in morphology from small, flattened, and stellate lesions to large, raised conglomerate lesions
- Crusting and dryness of nasal mucosa as well as septal perforation are common findings in patients with history of multiple nasal procedures for treatment of epistaxis
- Iron deficiency in conjunction with low ferritin and elevated transferrin is characteristic in patients with moderate to severe epistaxis

### Table 1

<table>
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<th>Criteria</th>
<th>Description</th>
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<tr>
<td>Epistaxis</td>
<td>Spontaneous and recurrent</td>
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<tr>
<td>Telangiectases</td>
<td>Multiple, at characteristic sites: lips, oral cavity, fingers, nose</td>
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<tr>
<td>Visceral lesions</td>
<td>Gastrointestinal telangiectasia, pulmonary, hepatic, cerebral, or spinal arteriovenous malformations</td>
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<tr>
<td>Family history</td>
<td>A first-degree relative with HHT according to these criteria</td>
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Patients with HHT will present to the otolaryngologist with a history of recurrent, spontaneous epistaxis. Some patients will identify particular triggers, such as dietary factors, emotional state, and seasonal variations. Other complaints include sequelae of recurrent epistaxis or prior treatments, such as crusting, foul odor, nasal obstruction, and septal perforation. In addition to the nasal cavity, mucosal telangiectasias involving the lips, hard palate, and tongue are characteristic, and patients may request treatment for these lesions as well. Multiple cutaneous telangiectasias are commonly seen on the skin of the face, ears, and fingertips, and referral to the dermatologist or facial plastic surgeon may be helpful in patients seeking treatment for these lesions. In a patient presenting with intranasal telangiectasias without a prior diagnosis of HHT, other conditions such as CREST syndrome, essential telangiectasia, and ataxia-telangiectasia syndrome should be considered in the differential diagnosis.

A complete examination of the upper aerodigestive tract should be performed during the initial evaluation of the patient with HHT. Anterior rhinoscopy and nasal endoscopy may detect presence of telangiectasias. Nasal endoscopy is performed in a careful, atraumatic manner to prevent bleeding. Patients with a history of severe, high-volume epistaxis may wish to defer nasal endoscopy in the office. Intranasal crusting and blood clots are typically present overlying larger telangiectasias, and debridement, if indicated, should be performed carefully to prevent bleeding. Nasal crusting is particularly prevalent in patients with a history of septodermoplasty. Cotton balls soaked with oxymetazoline or equivalent topical decongestant are a useful adjunct during the examination. Nasal endoscopy is useful to evaluate for septal perforation, scarring, and more posterior telangiectasias that may not be evident on anterior rhinoscopy.

Intranasal telangiectasias vary widely with regard to morphology and number of lesions present. Mahoney and Shapshay described 3 distinct vessel patterns associated with nasal telangiectasias in HHT: small, punctate isolated telangiectasias (type I) (Fig. 1), diffuse interconnecting lesions with “feeder” vessels (type II) (Fig. 2), and large, solitary AVM (type III). Type I lesions are more likely associated with mild

Fig. 1. Endoscopic view of left nasal cavity and left middle turbinate in patient with Shapshay type I (small, stellate) telangiectasias.
epistaxis, whereas type II and type III lesions are often associated with moderate or severe epistaxis. Morphologic characterization and quantifying number of lesions may be useful for treatment planning. For example, smaller punctate type I lesions tend to respond well to laser treatment, whereas larger, raised type II and III lesions are better addressed with coblation or electrocautery. Additionally, patients with fewer, predominantly anterior lesions are better candidates for in-office treatment as compared with patients with more numerous, more posterior telangiectasias.

A careful history with attention to family history as well as symptoms of visceral AVMs should be performed. A history of stroke, heart failure, and GI bleeding warrant further investigation. The Curacao criteria expert guidelines recommend screening for pulmonary AVMs and cerebrovascular malformations in all patients with HHT at time of diagnosis. Bubble echocardiogram is a screening test recommended every 5 to 10 years following initial screening to rule out formation of new AVMs. If positive, bubble echocardiogram is followed by chest computed tomography (CT) scan to fully characterize size and number of AVMs. Patients with pulmonary AVMs should be referred to a pulmonologist, interventional radiologist, or thoracic surgeon for

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<th>What should be included in the initial evaluation in patients with HHT?</th>
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<tr>
<td>• Nasal endoscopy performed atraumatically, with careful debridement of crusts so as to characterize number and size of telangiectasias</td>
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<tr>
<td>• The Epistaxis Severity Score is a normalized, validated scoring tool used to quantify severity of epistaxis</td>
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<tr>
<td>• Patients with HHT should undergo pulmonary AVM screening with bubble echocardiogram at time of diagnosis and every 5 to 10 years thereafter. Brain MRI is indicated at time of diagnosis. Gastrointestinal (GI) endoscopy is indicated in patients with anemia disproportionate to degree of epistaxis</td>
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<tr>
<td>• Complete blood count and type and cross are recommended before any surgical intervention. Patients with unknown pulmonary AVM status undergoing surgery should undergo bubble echocardiogram screening before surgery</td>
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Fig. 2. Endoscopic view of right nasal cavity and middle turbinate in a patient exhibiting Shapshay type II (large, diffuse, interconnecting) telangiectasias.
treatment. Brain MRI is recommended at time of diagnosis, and presence of cerebrovascular malformations warrants referral to neurosurgery. Routine GI endoscopy is not indicated; however, patients with a history of GI bleeding and/or anemia disproportionate to epistaxis severity should undergo GI endoscopy for diagnosis and treatment of GI AVMs. The wide range of organ systems potentially affected in patients with HHT underlines the need for a multidisciplinary approach.

The Epistaxis Severity Score (ESS) is a validated, normalized scoring tool composed of questions related to nosebleed frequency, severity, presence of anemia, need for medical treatment, and need for blood transfusion. This is the only validated patient questionnaire for HHT-related epistaxis, and is a useful tool for evaluating treatment success as well as following nosebleed severity over time.

Severity of HHT-related epistaxis ranges from mild, occasional nasal bleeding to severe, life-threatening epistaxis. Mild nosebleeds are primarily a quality-of-life issue, whereas moderate bleeding may be associated with anemia or iron deficiency. Microcytic iron deficiency anemia with elevated transferrin and low ferritin is characteristic of moderate or severe HHT-related epistaxis. Expert guidelines recommend annual hemoglobin and hematocrit screening in patients older than 35 years.

Laboratory testing before surgical intervention should include a complete blood count with type and screen or type and crossmatch. Patients should be questioned regarding need for recent blood transfusions, and prepared for possible transfusion during surgery. Patients with unknown pulmonary AVM status should be screened with bubble echocardiogram before surgery. Patients with a positive or unknown history of pulmonary AVM should receive antibiotic prophylaxis before any procedure. Additionally, intravenous filters should be considered in patients with a positive or unknown history of pulmonary AVMs to prevent air embolism.

**MANAGEMENT**

<table>
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<tr>
<th>What nonsurgical treatments are available for patients with HHT-related epistaxis?</th>
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<tr>
<td>• Nosebleed prevention is the mainstay of therapy for any patient with HHT-related epistaxis</td>
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<tr>
<td>• Nonmedicinal prevention measures include avoidance of triggers, use of nasal emollients, proper nasal hygiene, and avoidance of blood thinning medications if possible</td>
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<tr>
<td>• Pressure tamponade and use of topical spray decongestants are useful for nosebleed treatment</td>
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<tr>
<td>• Nondissolvable nasal packing should be avoided in most cases</td>
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<tr>
<td>• Topical medical treatments include bevacizumab (Avastin), estriol, tranexamic acid, thalidomide, and timolol</td>
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Treatment of HHT-related epistaxis includes nasal hygiene and avoidance of triggers. Regardless of treatment modality (with exception of nasal closure), nosebleeds will continue. In most patients, treatment goals include decreasing the frequency, volume, and severity of epistaxis and to improve quality of life. Less commonly, treatment is pursued to relieve severe anemia or prevent life-threatening epistaxis. There are few randomized, prospective studies of treatment modalities for HHT-related epistaxis. Most reports are retrospective case series or based on expert opinion. No standard treatment algorithm is available; however, a graduated treatment strategy beginning
with conservative topical therapies progressing to more invasive surgical treatments is recommended. Table 2 summarizes the treatment options with associated level of evidence.

Prevention of nasal dryness and crusting is essential in all patients with HHT. Various topical nasal emollients are available to maintain nasal humidification. Many commercial products with a strong safety profile are available for frequent, daily use. In general, saline gels formulated with hyaluronic acid are more effective than saline mist sprays at maintaining a high level of nasal hydration. Several other emollient products have been proposed for daily use in patients with HHT, including mupirocin ointment, petroleum jelly, and tranexamic acid gel. A recent cohort study demonstrated a significant improvement in ESS using compounded topical sesame/rose geranium oil in patients with HHT. Nasal saline irrigations may be effective at preventing crusting and dryness, but should be administered carefully in an atraumatic manner as they may be a trigger for nosebleeds in some patients.

Bevacizumab (Avastin, Roche/Genentech, San Francisco, CA) is a monoclonal antibody inhibitor of the VEGF-A receptor that has been proposed as a preventive treatment modality for HHT-related epistaxis. Several case series have demonstrated efficacy with this treatment modality. Limitations include cost and lack of insurance coverage due to off-label use. When used topically, benefits of treatment generally extend for 2 weeks following cessation of use. The largest published case series to date evaluated treatment of 52 patients with HHT with topical and/or injected bevacizumab in conjunction with potassium-titanyl phosphate (KTP) laser photocoagulation. Significant improvement in ESS was noted up to 46 months after treatment. Submucosal injection of bevacizumab in the region of the cartilaginous septum was associated with a high risk of septal perforation in this series.

Studies evaluating treatment for HHT-related epistaxis using estrogen and selective estrogen receptor modifiers such as raloxifene have demonstrated some efficacy. Two prospective, randomized studies evaluating raloxifene treatment in patients with HHT demonstrated improvement in severity and frequency of nosebleeds, in addition to improvement in hemoglobin levels in a subset of patients. Topical estriol therapy has also proven effective in reducing HHT-related epistaxis. Patients will often report worse epistaxis following menopause, and hormonal replacement should be considered in those patients not at increased risk of breast or endometrial cancer.

Recently, topical timolol therapy (0.5% ophthalmic solution, 1 drop each nostril 3 times a day) has been described as a successful treatment for HHT-related epistaxis, although evidence is limited to a single case report.

<table>
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<tr>
<th>What surgical interventions are available for HHT-related epistaxis?</th>
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<tr>
<td>- Surgical treatment should be tailored to the individual patient and severity of epistaxis</td>
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<td>- In-office treatments useful for mild to moderate epistaxis include injection sclerotherapy or bipolar electrocautery</td>
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<tr>
<td>- Use of silver nitrate cauterity should be avoided in patients with HHT</td>
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<tr>
<td>- Cauterization treatment options in the operating room (OR) for moderate to severe epistaxis include laser photocoagulation, electrocautery, and electrosurgical plasma coagulation (coblation)</td>
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<td>- More aggressive surgical options include septodermoplasty and nasal closure (Young procedure) for cases of moderately severe or severe epistaxis</td>
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<tr>
<td>Therapy</td>
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<tr>
<td>Estrogen/antiestrogen therapy</td>
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<tr>
<td>Topical estrogen</td>
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<tr>
<td>Bevacizumab (topical application)</td>
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<td>Bevacizumab (injection)</td>
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<tr>
<td>Injection sclerotherapy</td>
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<td>Bipolar cautery</td>
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<td>Treatment</td>
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<td>----------------------------</td>
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<tr>
<td>KTP laser</td>
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<tr>
<td>Nd:YAG laser</td>
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<tr>
<td>Coblation</td>
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<tr>
<td>Septodermoplasty</td>
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<td>Young procedure</td>
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In-office treatment has gained in popularity in recent years following initial descriptions of intranasal injection sclerotherapy. Intranasal sclerotherapy is useful for mild or mild to moderate cases of HHT-related epistaxis. One of the primary advantages of in-office treatment includes avoidance of general anesthesia, which is a significant benefit in patients suffering from pulmonary or cardiac sequelae of HHT. Disadvantages of in-office treatment include diminished access to more posterior lesions and decreased ability to control higher volume bleeding in an awake patient. Boyer and colleagues provided the first description of in-office injection sclerotherapy using sodium tetradecyl sulfate (Sotradecol, AngioDynamics, Latham, NY). A large European case series describes use of polidocanol injection sclerotherapy with equal efficacy. The sclerosant is diluted in a 4:1 fashion with air and foamed by vigorously passing the solution between 2 Luer lock syringes. Following application of local anesthetic, up to 3 mL foamed sclerosant is injected peripherally into telangiectasias in a submucosal fashion. Generally, benefits of injection sclerotherapy will last up to 4 months.

Monopolar electrocautery and silver nitrate cautery have been associated with increased risk of septal perforation. Monopolar suction electrocautery may be used prudently in the OR when other modalities fail to achieve hemostasis (Video 1). In general, silver nitrate cautery should be avoided in patients with HHT as it is relatively imprecise and has a tendency to worsen epistaxis severity.

Endoscopic intranasal laser photocoagulation has been a mainstay of operative treatment for HHT-related epistaxis since it was first described nearly 30 years ago. Both the KTP and neodymium-doped yttrium aluminum garnet (Nd:YAG) lasers have been described for this use. The advantages of this method include precise coagulation of vessels with relatively minimal depth of thermal injury. Disadvantages of laser treatment include cost and lack of availability at some centers. Additionally, eye protection is required by all OR personnel, and special precautions, such as moist eye pads and wet towel drapes, are necessary during use. Several retrospective series have described success with this treatment modality, with decrease in nosebleed frequency and severity for approximately 1 to 2 years following treatment. In general, patients with smaller, punctate telangiectasias fare well with laser photocoagulation, whereas those with larger, conglomerate lesions are better treated with other modalities, such as coblation or septodermoplasty. Lesions are treated in a “rosette” fashion, starting at the periphery of the lesion so as to address feeding vessels, then proceeding inward. Laser photocoagulation is relatively ineffective in situations with active bleeding (Video 2).

Bipolar electrocautery and electrosurgical plasma coagulation (coblation) are both effective surgical treatments for HHT-related epistaxis. Bipolar is generally preferred over monopolar electrocautery due to improved precision and decreased depth of thermal injury. Several bipolar forceps designed for endoscopic nasal surgery are available commercially, such as the Landolt, Wormald, and Stammberger instrument designs. The coblation wand (PROcise EZ and EXcise PDX wands; ArthroCare ENT, Austin, TX) combines irrigating bipolar function along with coblation function, which is useful for ablation of large, conglomerate telangiectasias. The coblation wand is particularly effective during active bleeding (Fig. 3, Video 3). A recent randomized, prospective trial demonstrated similar efficacy comparing coblation treatment with KTP laser photocoagulation.

Septodermoplasty is an effective treatment for patients with HHT with moderate to severe epistaxis, or in those patients who prefer to avoid repeat surgical cautery treatments. This technique involves removal of the superficial layer of septal mucosa while carefully preserving the underlying perichondrium. This may be accomplished using curettage or with the microdebrider. A split-thickness skin graft is positioned over the denuded septal mucosa, and nasal packing is left in place for several days to prevent disruption of the graft (Fig. 4). Some investigators describe staged unilateral...
procedures; bilateral procedure may be performed safely in one session. In patients with prominent telangiectasias along the nasal floor, inferior turbinates, and lateral nasal wall, the graft may be extended to cover the nasal floor and lateral nasal wall after removal of the inferior turbinates. A handful of case series have described success with this technique as measured by improved quality of life and decreased need for blood transfusion in previously transfusion-dependent patients with HHT. Patients should expect a significant reduction in epistaxis for at least 2 years, at which point neovascularization with formation of new telangiectasias within and around the graft starts to occur. Invariably, patients experience loss of normal mucociliary function in the region of the graft with resulting crusting and foul odor, and a daily nasal hygiene regimen may be necessary in these patients.

Fig. 3. The coblation wand positioned between the inferior turbinate and septum as the surgeon prepares to coblate a bulky, raised telangiectasia on the nasal septum.

Fig. 4. Endoscopic view of the left nasal cavity in a patient undergoing septodermoplasty procedure for HHT-related epistaxis. The split-thickness skin graft has been positioned in place over the left septum using a quilting Vicryl Rapide suture. The skin graft is distinguished by its relatively pale color.
Complete closure of the nasal vestibule, also known as the Young procedure, is used in patients with severe, transfusion-dependent or life-threatening epistaxis. This technique achieves complete nasal closure by raising mucocutaneous flaps through an endonasal or alotomy approach. This procedure results in obligatory mouth breathing and loss of olfaction. Although patients are often reluctant to lose the ability to breathe through their nose, this can be an effective and potentially life-saving procedure in cases in which prior treatment with cautery and/or septodermoplasty has been unsuccessful. This technique also may be performed in a unilateral fashion, and closure can be reversed at any time if the patient desires. Several small case series have described success with this technique when other less aggressive treatments have failed. Ting and colleagues described a case of severe life-threatening epistaxis in a patient with HHT following the Young procedure that required reversal of nasal closure and endovascular treatment.

Septal perforation is one of the most common complications of surgical treatment for HHT-related epistaxis, and should be avoided if possible (Fig. 5). Injury to opposing sides of the cartilaginous septum deep to the perichondrium can result in a perforation. Septal perforation causes a disruption of laminar nasal airflow with subsequent dryness and crusting. This results in large telangiectasias at the posterior aspect of the

Fig. 5. Endoscopic view of the right nasal cavity in a patient with a large septal perforation resulting from multiple prior surgical treatments for HHT-related epistaxis.
perforation, and treatment of these lesions can enlarge the perforation. Collapse of the nasal dorsum with saddle nose deformity may occur from large septal perforations. Careful surgical technique while avoiding injury to opposing sides of the septum, and avoidance of excessive use of monopolar cauter y or silver nitrate cauter y will help prevent this complication. Submucosal injection of bevacizumab following surgical cauter y increases risk of perforation. A staged procedure should be considered when large, prominent telangiectasias are present on opposing areas of the anterior nasal septum.

Scarring is an expected outcome of most surgical cauter y procedures. Scarring results in loss of mucociliary clearance and function, leading to crusting and mucostasis. Postoperative nasal hygiene and nasal debridement may help prevent adverse scarring. Scarring with formation of synechiae may result in obstruction of nasal airflow or outflow of sinonasal secretions, and this may be corrected in the office or during subsequent procedures.

Some patients may experience a worsening in epistaxis severity following treatment. In these cases, alternative treatment modalities should be considered. It is not uncommon for patients to experience increased epistaxis during the initial 2 weeks after surgery, and a regimen of aggressive nasal humidification along with a course of oral antibiotics may help speed healing in these cases. Use of bevacizumab in conjunction with surgical cauter y may increase success rates in patients in whom standard treatments have failed to garner benefit.

**SUMMARY**

Several medical and surgical treatments are available for HHT-related epistaxis. Severity of epistaxis varies widely among patients with HHT, from mild, annoying nosebleeds to severe, transfusion-dependent and potentially life-threatening epistaxis. Treatment should be tailored to the individual patient, and careful presurgical counseling is helpful to establish realistic expectations of surgical treatments. Generally, a graduated treatment approach is successful, starting with topical therapies and progressing to more invasive surgical procedures.

The HHT Foundation, now known as Cure HHT, is a valuable online resource for patients and their families ([curehht.org](http://curehht.org)).

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**Post-Test Questions (Correct answers are in italics)**

1. Mutations in these 2 genes are responsible for most cases of HHT
   a. **ACVRL1 and Endoglin (ENG)**
   b. Endoglin (ENG) and MADH4
   c. TGF-beta and VEGF
   d. HHT1 and HHT2

2. The ____ criteria are used to make a clinical diagnosis of HHT
   a. HHT Foundation
   b. ESS
   c. Curacão
   d. Faughnan

3. In addition to intranasal telangiectasias, patients with HHT are also at risk for AVMs involving these organ systems
   a. Pulmonary
   b. Gastrointestinal
   c. CNS
   d. **All of the above**
4. Which of the following methods of cautery are not generally recommended for treatment of HHT-related epistaxis?
   a. KTP laser  
   b. Silver nitrate  
   c. Bipolar  
   d. Coblation

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.otc.2016.02.010.

Supplementary PDF slides related to this article can be found online at http://www.oto.theclinics.com/.

REFERENCES


SUGGESTED READINGS


This article provides the first description of in-office Sotradecol sclerotherapy treatment for HHT-related epistaxis, which has become a popular in-office treatment option for HHT patients over the past 5 years.


This article summarizes the expert consensus guidelines for diagnosis and HHT and provides recommendations for screening and other diagnostic studies.


This article provides a good summary of both septodermoplasty and KTP laser treatment for HHT, and also provides some outcomes data.


This article summarizes the Epistaxis Severity Score, which is a validated questionnaire that is, useful for quantifying severity of nosebleeds in patients with HHT.


This article provides the results of a large survey sent to patients with HHT inquiring about various dietary and lifestyle triggers for epistaxis.