Petrous apex cholesterol granulomas: evolution and management

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Petrous apex cholesterol granulomas result from obstruction of the normal aeration of the petrous air cells and have traditionally been treated by drainage and stent placement via a transtemporal approach. The immediate results were quite satisfying, but recurrence rates as high as 60% have been reported in some series. The authors present their experience treating 14 patients with petrous apex cholesterol granulomas. An extended middle fossa approach and a petrosal approach were used for eight and two patients, respectively. All underwent complete removal of the granuloma and cyst wall followed by obliteration of the cavity with a pedicled strip of temporalis muscle. No recurrences were seen at a mean follow-up period of 3.8 years. Four patients who did not undergo surgery are being followed clinically and with serial magnetic resonance images. Additionally, the clinical and radiographic findings in this series give new insights into the origin and continued growth of these lesions and confirm what had been described previously only in experimental models. It is concluded that petrous apex cholesterol granulomas feature a continuum of both clinical and radiographic findings and radical removal via an extended middle fossa approach is advocated.

KEY WORDS • cholesterol granuloma • etiology • middle fossa • petrous apex

PETROUS apex cholesterol granulomas are the result of chronic obstruction of the normal aeration to the petrous air cells. The traditional treatment has been simple drainage via either a transtemporal or transspHENoidal route, followed by placement of a small silastic tube to reestablish aeration. The recurrence rate with these approaches has been reported to be as high as 60%, with several patients requiring multiple revisions. The series presented here represents a retrospective review of our case material, and we concede the inherent deficiencies and biases associated with such a review. With this caveat and based on our experience, we advocate complete excision of the lesion followed by obliteration of the cavity with vascularized tissue, and present our experience using this technique. This series covers a wide clinical spectrum from asymptomatic to large destructive lesions, as well as a continuum of radiographic characteristics.

Clinical Material and Methods

Between July 1987 and January 1996, 14 patients (nine females and five males) ranging in age from 15 to 69 years (mean age 39 years) were treated by the senior author (O.A.) for cholesterol granuloma of the petrous apex. Their clinical characteristics are presented in Table 1. Presenting symptoms and signs included headache (four patients), dysfunction of fifth to eighth cranial nerves (eight patients), trigeminal neuralgia (one patient), cerebrospinal fluid (CSF) leak (one patient), and one patient was asymptomatic. Ten patients underwent surgical treatment via either the petrosal or the extended middle fossa approach, with the goal of complete removal of the cyst and obliteration of the cavity. Operations were not performed in four cases because the patients were asymptomatic (one patient), minimally symptomatic (two), or not a surgical candidate (one). Follow-up evaluation was accomplished by direct patient visit (four cases), phone interview with the patient (seven cases), or through the referring physician (three cases). Outcome was based on the patient’s report for those symptoms that were subjective in nature (for example, headache or facial pain) and on physical examination findings for objective deficits (including CSF leakage, cranial nerve palsies, and sensory loss).

Illustrative Cases

This series represents a wide clinical spectrum of petrous apex cholesterol granulomas. We describe four cases to illustrate the various stages of this disorder.

Case 10

This 27-year-old man developed acute-onset diplopia secondary to a partial sixth cranial nerve palsy. A computerized tomography (CT) scan was obtained and demonstrated expansion of the ipsilateral petrous apex (Fig. 1 left). On magnetic resonance (MR) imaging the lesion had
a high signal on both T₁- and T₂-weighted images (Fig. 1 center and right). Resection was performed via an extended middle fossa approach and a cholesterol granuloma of the petrous apex was completely excised. The cavity was then obliterated by a pedicle strip of temporalis muscle. Immediately after surgery the patient’s sixth cranial nerve palsy improved and his symptoms had almost completely resolved at the 2-month follow-up examination.

**Case 14**

This 38-year-old woman developed mild left-sided facial numbness in the second division of the trigeminal nerve; this was evident on formal examination. A nonenhancing lesion filling the left petrous apex was revealed on MR and CT studies. The CT scan did not show any bone changes, however, and the opposite petrous apex was well aerated (Fig. 2A). The lesion demonstrated a high-intensity signal on T₁-weighted MR images (Fig. 2B) and an isointense signal on T₂-weighted images (Fig. 2C). Because of the mild symptoms and atypical appearance on MR imaging, another MR image was obtained 9 months later. The patient reported that during the 9-month period the facial numbness had diminished significantly, only to return 2 weeks prior to the most recent study. The repeated MR image showed a small increase in the size of the lesion as well as in the T₂-weighted signal intensity (Fig. 2D). Further observation and radiographic follow up have been recommended.

**Case 8**

This 54-year-old woman presented with a 3-month history of transient “attacks” described as a dizzy sensation, unilateral facial numbness, and vertigo. The lesion was demonstrated by MRI to be a cholesterol granuloma filling the left petrous apex. Following resection, the symptoms improved and the patient has remained asymptomatic at the 2-month follow-up examination. Further observation and radiographic follow up have been recommended.

**TABLE 1**

**Clinical characteristics and follow-up results in 14 patients with petrous apex cholesterol granulomas***

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Preop Symptoms</th>
<th>Surgical Approach</th>
<th>Duration of Follow Up</th>
<th>Symptoms on Follow Up</th>
<th>Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>34, F</td>
<td>VIth, VIIth, &amp; VIIIth cranial nerve palsies</td>
<td>petrosal</td>
<td>8 yrs</td>
<td>improved</td>
<td>none</td>
</tr>
<tr>
<td>2</td>
<td>15, M</td>
<td>headache, VIth cranial nerve palsy</td>
<td>petrosal</td>
<td>7 yrs</td>
<td>asymptomatic</td>
<td>postop CSF leak</td>
</tr>
<tr>
<td>3</td>
<td>59, M</td>
<td>trigeminal neuralgia for 2 yrs</td>
<td>EMF</td>
<td>8 yrs</td>
<td>asymptomatic</td>
<td>none</td>
</tr>
<tr>
<td>4</td>
<td>18, M</td>
<td>headache, vertigo</td>
<td>EMF</td>
<td>5 yrs</td>
<td>asymptomatic</td>
<td>none</td>
</tr>
<tr>
<td>5</td>
<td>16, F</td>
<td>VIth cranial nerve palsy</td>
<td>EMF</td>
<td>36 mos</td>
<td>asymptomatic</td>
<td>developed seizures 2 yrs postop none</td>
</tr>
<tr>
<td>6</td>
<td>48, F</td>
<td>headache, VIth cranial nerve palsy</td>
<td>EMF</td>
<td>35 mos</td>
<td>asymptomatic</td>
<td>none</td>
</tr>
<tr>
<td>7</td>
<td>69, F</td>
<td>CSF leak</td>
<td>EMF</td>
<td>31 mos</td>
<td>mild V₃ paresthesias</td>
<td>none</td>
</tr>
<tr>
<td>8</td>
<td>54, F</td>
<td>seizures</td>
<td>EMF</td>
<td>15 mos</td>
<td>asymptomatic</td>
<td>none</td>
</tr>
<tr>
<td>9</td>
<td>45, M</td>
<td>VIth cranial nerve palsy</td>
<td>EMF</td>
<td>6 mos</td>
<td>asymptomatic</td>
<td>none</td>
</tr>
<tr>
<td>10</td>
<td>27, M</td>
<td>VIth cranial nerve palsy</td>
<td>EMF</td>
<td>2 mos</td>
<td>improved</td>
<td>none</td>
</tr>
<tr>
<td>11</td>
<td>41, F</td>
<td>headache, facial numbness</td>
<td>none</td>
<td>54 mos</td>
<td>no change</td>
<td>NA</td>
</tr>
<tr>
<td>12</td>
<td>55, F</td>
<td>VIth, VIIth, &amp; VIIIth cranial nerve palsies</td>
<td>none</td>
<td>17 mos</td>
<td>contra lateral VIIth cranial nerve palsy</td>
<td>NA</td>
</tr>
<tr>
<td>13</td>
<td>33, F</td>
<td>asymptomatic</td>
<td>none</td>
<td>1 yr</td>
<td>asymptomatic</td>
<td>NA</td>
</tr>
<tr>
<td>14</td>
<td>38, F</td>
<td>V₂ numbness</td>
<td>none</td>
<td>9 mos</td>
<td>numbness resolved, then recurred</td>
<td>NA</td>
</tr>
</tbody>
</table>

* EMF = endovascular flow. NA = not applicable.
associated with nausea and followed by a period of staring that lasted as long as 15 minutes. The interictal examination was unremarkable, and her electroencephalogram was interpreted as normal. An MR study demonstrated a small lesion in the right petrous apex that had a low-intensity signal on T₁-weighted images (Fig. 3 left), a bright signal on T₂-weighted images (Fig. 3 center), and no enhancement with gadolinium administration. A CT scan revealed erosion of the petrous apex and the medial wall of the carotid canal (Fig. 3 right). Internal carotid angiography was normal. Although this was not believed to be responsible for her “attacks,” we were concerned that this lesion might be either a chordoma or low-grade chordosarcoma. She underwent surgery via an extended middle fossa approach to the lesion. On entering the eroded petrous apex, subacute hemorrhage and thickened mucosa were found. This material was removed, and the cavity was obliterated with a strip of vascularized temporalis muscle. She has continued to suffer the transient attacks, but has remained otherwise asymptomatic and showed no evidence of recurrence at her 15-month follow-up review.

Case 7

This 69-year-old woman presented with a long history of CSF rhinorrhea. Radiographic evaluation with CT scanning, CT scanning following metrizamide-enhanced cisternography, and MR imaging all demonstrated erosion of the petrous apex by a cystic lesion that was radiographically consistent with a cholesterol granuloma (Fig. 4). The patient developed a bacterial meningitis that was treated with appropriate antibiotic medications, and 2 weeks later underwent surgery via an extended middle fossa approach. She has remained without CSF leakage or recurrence of the granuloma at 31 months of follow up.

Operative Techniques

In two cases, we resected the lesion via the petrosal approach, as previously described. This approach was used earlier in the series and we have since used a more direct extradural approach. In eight cases, we used an extended middle fossa approach, which has the following advantages: 1) a direct yet shallow working distance to the petrous apex with a minimum of extradural temporal lobe elevation; 2) extradural dissection; and 3) early exposure and control of the petrous portion of the internal carotid artery.

For the extended middle fossa approach, the patient is placed supine with a roll under the ipsilateral shoulder, and the head is rotated to keep the zygoma nearly horizontal. A preauricular curvilinear incision is made. The zygoma is dissected in a subperiosteal fashion to spare the frontalis branches of the facial nerve. The zygomatic arch is sectioned at its most anterior and posterior ends, thus allowing downward displacement of the arch along with

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**Fig. 2.** Case 14. Initial CT scan showing no bone changes (A), T₁- and T₂-weighted MR images (B and C) displaying high-intensity and isointense signals, respectively, and a repeated MR image 9 months later (D) showing a slight change in the character of the lesion.

**Fig. 3.** Case 8. Axial T₁- (left) and T₂-weighted (center) images displaying low-intensity and bright signals, respectively. The CT scan (right) reveals erosion of the petrous apex and the medial wall of the carotid canal.
the temporalis muscle. This maneuver allows exposure along the middle fossa floor with less than 1 cm of temporal lobe elevation. A limited craniotomy abutting the floor of the middle fossa is performed. The temporal lobe is supported extradurally with a malleable retractor, and the dissection is continued medially until the middle meningeal artery is identified at the foramen spinosum. This artery is coagulated and sharply sectioned. The foramen ovale is then identified anteriorly and the greater superficial petrosal nerve is identified medially. The lesser superficial petrosal nerve may be seen lateral to the greater superficial petrosal nerve (Fig. 5A). The greater superficial petrosal nerve is sharply dissected from its dural attachment and preserved; however, in some cases this nerve must be divided to avoid traction injury to the facial nerve. More medially, the petrous portion of the internal carotid artery can be seen, and, depending on the size of the petrous apex cholesterol granuloma, may be deviated superolaterally. Care is taken in exposing this region because the bone over the carotid canal is often dehiscent. Once the petrous apex cholesterol granuloma is identified and entered, the fluid is drained and the soft tissues, including the pseudocapsule, are completely excised (Fig. 5B). Obliterating the resultant cavity with fat fragments makes interpreting the follow-up MR images arduous because of the difficulty in distinguishing the signal intensities of fat versus postoperative fluid collections. Therefore, we fashion a pedicled strip of temporalis muscle that can be used to obliterate the petrous apex cholesterol granuloma cavity (Fig. 5C). This permits postoperative MR studies that are easy to read, with the muscle flap giving a consistently hypointense signal on T1-weighted images.

![Image](https://example.com/image)

**Fig. 4.** Case 7. Metrizamide-enhanced CT scan demonstrating erosion of the petrous apex by a cystic lesion that was radiographically consistent with a cholesterol granuloma.

**Fig. 5.** A: View of the middle fossa showing the foramen ovale, third division of the trigeminal nerve, foramen spinosum, middle meningeal artery, and the lesser (lateral) and greater (medial) superficial petrosal nerves. The cholesterol granuloma is seen posterior to the third division of the trigeminal nerve and medial to the petrous carotid artery. B: Drawing showing resection of cholesterol granuloma from the petrous apex. C: Drawing showing obliteration of the cavity by means of a pedicled strip of temporalis muscle.

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A total of 10 patients underwent surgical procedures, two via a petrosal approach and eight via an extended middle fossa approach. All 10 patients were treated with radical removal of the cyst followed by obliteration of the cavity. Follow-up evaluations (mean 3.8 years, range 2 months–8 years) have shown no clinical evidence of recurrence. In four of the 10 surgically treated patients, radiographic follow-up has demonstrated no recurrence. Postoperatively, seven of 10 patients are clinically asymptomatic. Of the remaining three, the patient in Case 1 had marked improvement in her cranial nerve palsies; the patient in Case 7, who had presented with a CSF leak from a large erosive petrous apex cholesterol granuloma, has had no recurrence of CSF leakage and complains of only mild paresthesias in the third division of the trigeminal nerve; and the patient in Case 10 still has a mild sixth cranial nerve palsy that is improving. There was one postoperative CSF leak, which required reexploration of the surgical site and repair of a dural defect. One patient who has remained clinically asymptomatic developed a seizure disorder 2 years postsurgery that is well controlled by antiepileptic medications. There were no other postoperative complications.

The four patients who were treated nonsurgically have been followed, both clinically and with serial MR images, for an average of 23 months (9–54 months). One has remained asymptomatic, one has had no change in her mild symptomaticology, one had attained resolution of her second division trigeminal nerve numbness followed by recurrent numbness 9 months later, and one has developed a contralateral seventh cranial nerve palsy unrelated to her petrous apex cholesterol granuloma.

Discussion

Cholesterol granulomas are benign expansive lesions most commonly found in the middle ear or mastoid region in association with some type of inflammatory ear disease.2,9,10,12,24–27,31,33,35,36 Petrous apex cholesterol granulomas, however, have been described less often and in many cases have been erroneously grouped with epidermoid tumors, which are etiologically and histologically distinct from cholesterol granulomas.

Etiology and Experimental Models

Meyer (as quoted in Amedee, et al.4) is credited with providing the first description, in 1893, of a cholesterol granuloma in the peritoneum. One year later, Manasse (as quoted in Amedee, et al.4) described cholesterol crystals surrounded by foreign body giant cells and granulations in the external auditory canal and middle ear. Since then, there have been many reports in the literature of petrous apex cholesterol granulomas, but a clear understanding of the nature of this entity has been difficult to achieve because of the numerous terms used to describe it, as well as its grouping in many reports with other histologically distinct lesions, particularly epidermoid cysts.3,8,11,13,18,19,23,28,34 These lesions have been labeled over the years as cholesterol cysts, unicameral cysts, chocolate cysts, and xanthomas of the temporal bone.8,16,22,29,32

Several investigators have established in animal models that the development and continued “growth” of cholesterol granulomas involves ventilatory obstruction of normally aerated bone.5,20,30 This obstruction leads to absorption of air and the development of negative pressure within the air cells, causing mucosal engorgement and hemorrhage. The cholesterol crystals that result from the red cell degradation incite a typical foreign body reaction leading to further hemorrhage and inflammation. Ojala30 was able to produce histological changes similar to those found in cholesterol granulomas by obstructing the ventilation to the normally aerated chicken humerus. Within 2 weeks, this study demonstrated cholesterol crystals surrounded by giant cells, as well as edematous granulation tissue. In 1982, Hiraide, et al.30 conducted an elegant study in which they obstructed the pharyngeal orifice of the eustachian tube in 10 squirrel monkeys and examined the temporal bones of each monkey at 1, 3, or 6 months. These researchers found a progression of histological changes beginning at 1 month with the appearance of thickened mucous membranes and a few macrophages, leukocytes, and red cells, followed at 3 months by a collection of lipid droplets and birefringent cholesterol crystals surrounded by macrophages and foreign body giant cells. By 6 months, well-developed cholesterol granulomas had formed in the mastoid region, complete with cholesterol crystals surrounded by fibroblasts, hemosiderin pigment cells, and foreign body giant cells.

The progression from mucosal engorgement and hemorrhage within a blocked air cell to a cholesterol granuloma with cholesterol crystals and foreign body giant cells has been clearly demonstrated only in experimental models. In our series, we were able to observe a continuum of radiographic findings, symptoms, and histological findings ranging from small asymptomatic lesions found incidentally (Case 8) to enormous erosive lesions occupying both the extra- and intradural compartments (Case 7).

Three of our patients who had only mild symptoms exhibited atypical lesions on MR imaging. These lesions were located in the petrous apex and lacked the typical bright signal on T1- and T2-weighted imaging. In fact, one patient had a lesion with a low-intensity signal on T1- and a high-intensity signal on T2-weighted imaging. She underwent exploratory surgery because of the atypical nature of the lesion and our concern that it was a neoplasm. At the time of surgery mucosal thickening and subacute hemorrhage were discovered. This was most likely the earliest stage in the formation of a petrous apex cholesterol granuloma. Because the typical signal characteristics are a combination of chronic hemorrhagic byproducts, cholesterol crystals, and proteinaceous debris, the MR image may display any combination of T1- and T2-weighted signal characteristics during the early stages of these lesions.

Our belief is that there is not only a clinical continuum but a radiographic progression associated with petrous apex cholesterol granulomas. We found that some petrous apex cholesterol granulomas will not exhibit the characteristic MR signatures because they have not reached the mature stage. Case 14 is an excellent example: serial MR images demonstrated a petrous apex lesion with high-intensity T1- and isointense T2-weighted signal character-
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istics associated with a well-aerated contralateral petrous apex. Clinically this patient exhibited second division trigeminal nerve numbness that resolved but then suddenly returned. The recurrence of the numbness was probably related to a second hemorrhage and slight expansion of the cavity. This correlated with the changes seen on the repeated MR study.

Treatment Protocols

Patients who are asymptomatic or minimally symptomatic can be safely followed with serial neurological examination and MR imaging as we are doing in four of our patients. For patients with significant neurological findings or large compressive lesions, surgery is the only treatment available at this time. Using the petrosal approach initially and the extended middle fossa approach subsequently, we have been able to remove the entire granuloma and cyst wall safely and to prevent recurrence by obliterating the cavity with vascularized tissue. With this technique, we have been able to achieve a 0% recurrence rate in an average follow-up period of more than 3 years.

The traditionally recommended treatment for petrous apex cholesterol granulomas has been drainage without removal of the cyst wall, followed by restoration of aeration via a silastic tube to one of the other open temporal bone air cells. This has been accomplished most often via an intrapetrous route through or under the temporal bone structures or transsphenoidally if the lesion abuts the sphenoid sinus. These approaches are quite demanding technically, even for surgeons familiar with temporal bone anatomy, place the facial nerve and cochlear function at undue risk, provide limited access to the pathology through a deep and restrictive operative field, and result in a relatively high rate of recurrence, leading to multiple revisions in some patients.

Brodkey, et al., recently reported a series of 17 cases of petrous apex cholesterol granuloma. Eleven were drained via a transmastoid–infralabyrinthine, transcanal–infra- cochlear, or translabyrinthine approach. Although they did not experience any recurrences during a follow-up period of 29.5 months, 44% of the patients with good preoperative hearing (tested by audiogram) had postoperative hearing loss despite being treated with a hearing preservation approach.

In 1989, Thedinger, et al., reported on 10 patients who were treated with drainage and silastic tube placement. Six patients (60%) experienced recurrence of their lesions, with five requiring two or more procedures. One patient in their series required six drainage procedures. In all cases, they attributed the recurrence to closure of the fistulous tract or obstruction of the silastic stent. In 1991, Giddings, et al., reported on two cases treated by drainage alone, one with recurrence requiring reoperation. In 1990, Altschuler, et al., reported two recurrences in four patients who underwent operation via a retro- or a transmastoid approach. The high incidence of recurrence is not surprising when one keeps in mind the nature of the lesion being treated. When the thickened mucosa, which is the source of the repeated hemorrhages, is not completely excised, it is highly unlikely that a small silastic tube will remain patent in the face of the thick breakdown products of a cholesterol granuloma.

Histological Findings

The histopathological nature of cholesterol granuloma is well described and consists of an inflammatory granulation tissue matrix containing various sizes of cholesterol crystals surrounded by multinucleated giant cells (Fig. 6). No special stains or immunohistochemical studies are necessary to diagnose these lesions. If detected during the very early stages, only chronic hemorrhage without the inflammatory reaction will be found (see for example Case 8).

Radiographic Findings

Prior to the advent of MR imaging, distinguishing a cholesterol granuloma from a cholesteatoma or mucocoele of the petrous apex was at times extremely difficult. Even with the availability of MR imaging, differentiating these lesions, particularly in the early stages, can be problematic. The appearance of a petrous apex cholesterol granuloma on CT scans is that of a smooth margined nonenhancing mass located in the anteromedial portion of the petrous apex (Fig. 1 left). The density of the mass will be close to that of brain tissue and typically the opposite petrous apex will be well pneumatized. Whereas the CT characteristics help distinguish cholesterol granulomas from other lesions of the petrous apex, their appearance on MR images is most often diagnostic. Mature cholesterol granulomas demonstrate high signal intensities on both T1- and T2-weighted images, which, when combined with their lack of enhancement or only meager peripheral enhancement with gadolinium, are almost pathognomonic for this entity (Fig. 1 center and right). For petrous apex cholesterol granulomas in the earlier stages of development, CT scans may show only mild erosion at the petrous apex, and the MR images may not display the signal intensities seen with older, more developed lesions.

Fig. 6. Photomicrograph showing a typical cholesterol granuloma. Note cholesterol clefts (open arrow) and multinucleated giant cells (black arrow). H & E, original magnification × 10.
Clinical Presentation

As is the case with any lesion of the skull base, the presenting signs and symptoms of petrous apex cholesterol granulomas depend on the location and extent of the tumor. Smaller lesions may be found incidentally during radiographic evaluation for unrelated complaints or may be discovered during the evaluation of nonspecific complaints such as headache or dizziness; in six of our patients radiographic studies were obtained because of related or minor symptomatology. As these lesions progress, they cause compressive dysfunction of local cranial nerves (for example, trochlear, trigeminal, and abducens) leading to facial pain and/or numbness or diplopia. Larger lesions with more involvement of the temporal bone may lead to unilateral hearing loss or facial weakness. Extremely large lesions can erode intradurally and patients may present because of CSF leak (as in Case 7), cerebellopontine angle mass lesions, or chemical meningitis from spilling of the cyst contents within the subarachnoid space. Continued clinical and experimental studies of the pathophysiology of these lesions will likely reveal that once the repeating cycle of blockage, hemorrhage, and inflammation has begun, it will continue; however, the time course of this progression may vary greatly among individuals. We are currently following four patients with diagnoses of petrous apex cholesterol granuloma on MR imaging who have remained stable with a mean follow-up period of 23 months.

Conclusions

Cholesterol granulomas of the petrous apex are distinct lesions resulting from blockage of the normal aeration of the petrous apex air cells, which in turn leads to a repeating cycle of mucosal engorgement, hemorrhage, and granuloma formation.

The clinical and radiographic findings vary according to the age of the lesion and range from subacute hemorrhage within the air cells to well-formed granuloma and erosion of the petrous apex.

We strongly advocate the extended middle fossa approach because it provides a safe, shallow, extradural exposure of the petrous apex and enables the surgeon to remove the granuloma totally and to obliterate the cavity with vascularized tissue.

Drainage by either fenestration or silastic tube placement is inadequate and prone to failure because of the pathophysiological nature of the granuloma and consistency of its contents.

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