Granulomatous Disease in the Head and Neck: Developing a Differential Diagnosis

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Abbreviations: GD = granulomatous disease, GPA = granulomatosis with polyangiitis, IgG4 = immunoglobulin G4, LCH = Langerhans cell histiocytosis, SLE = systemic lupus erythematosus

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Introduction

Any process that results in granuloma formation in the body may be termed granulomatous (1,2). Histologically, granulomas are aggregates of mononuclear inflammatory cells or modified macrophages, which are usually surrounded by a rim of lymphocytes and often contain giant cells (2–4). Granulomas typically form to protect the host from persistent inflammatory stimuli, which if
The history of a GD is provided, the radiologist should be aware of the head and neck manifestations of the disease in order to perform a complete search for possible disease, as well as to distinguish these findings from other possible causes. Knowledge of the clinical and radiologic patterns of GDs in the head and neck will allow interpreting radiologists to provide a useful differential diagnosis, thus facilitating appropriate clinical management.

### Etiology

**Autoimmune GDs**

Autoimmune GDs with head and neck manifestations include GPA (formerly known as Wegener granulomatosis), Churg-Strauss syndrome, and Behçet disease. GPA is a rare, multisystem, necrotizing vasculitis whose classic triad includes necrotizing respiratory tract granulomas, small vessel vasculitis, and renal disease (6,7). Patients with GPA most commonly present with sinonasal disease in the form of chronic sinusitis, epistaxis, and rhinitis (6). Churg-Strauss syndrome, or allergic granulomatous angiitis, is characterized by asthma, nasal disease, systemic vasculitis, and eosinophilia (8,9). Patients with Churg-Strauss syndrome present with allergic rhinitis and nasal polyposis (9,10). Behçet disease is an autoimmune vasculitis that involves the central nervous system and also causes mucosal ulcerations, uveitis, and sensorineural hearing loss in the head and neck (11–13).

**Infectious GDs**

Infectious conditions that incite granuloma formation in the head and neck include tuberculosis, cat-scratch disease, syphilis, leprosy, and fungal infections. Tuberculosis occurs as a result of infection by *Mycobacterium tuberculosis* ongoing may produce locally inflammatory and destructive effects (1,5). Granuloma formation may be the primary result of a disease or a secondary disease association. Granulomatous diseases (GDs) may be autoimmune, infectious, idiopathic, or hereditary (1,4) (Table 1). GDs often are systemic processes with manifestations throughout the body. In the head and neck, GD processes have a wide range of clinical manifestations that may affect the orbits, sinonasal cavities, salivary glands, aerodigestive tract, temporal bone, skull base, or vascular structures.

Imaging findings of GD in the head and neck can appear nonspecific, with considerable overlap with imaging findings of other conditions, including malignancy. In such situations, it is important for the radiologist to raise the possibility of GD as an alternative diagnosis to malignancy to avoid inappropriate treatment. Without the benefit of a prior diagnosis of GD, laboratory findings, or suggestive clinical signs and symptoms, GDs may be difficult to distinguish from each other radiologically. However, certain imaging findings in the head and neck should prompt the radiologist to include GD in the differential diagnosis. When clinical history of a GD is provided, the radiologist should be aware of the head and neck manifestations of the disease in order to perform a complete search for possible disease, as well as to distinguish these findings from other possible causes. Knowledge of the clinical and radiologic patterns of GDs in the head and neck will allow interpreting radiologists to provide a useful differential diagnosis, thus facilitating appropriate clinical management.

### Table 1: GDs with Head and Neck Manifestations

<table>
<thead>
<tr>
<th>Autoimmune</th>
<th>GPA (Wegener granulomatosis)</th>
<th>Churg-Strauss syndrome</th>
<th>Behçet disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infectious</td>
<td>Tuberculosis</td>
<td>Cat-scratch disease</td>
<td>Syphilis</td>
</tr>
<tr>
<td></td>
<td>Leprosy</td>
<td>Fungal (blastomycosis, histoplasmosis)</td>
<td></td>
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<tr>
<td></td>
<td>Actinomycosis</td>
<td>Rhinoscleroma</td>
<td></td>
</tr>
<tr>
<td>Idiopathic</td>
<td>Sarcoidosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hereditary</td>
<td>Chronic GD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other diseases with secondary granulomatous manifestations</td>
<td>Relapsing polychondritis</td>
<td>LCH</td>
<td>SLE</td>
</tr>
<tr>
<td></td>
<td>Rheumatoid arthritis</td>
<td>Chemical exposure (eg, cocaine, talc, beryllium)</td>
<td></td>
</tr>
</tbody>
</table>

Note.—GPA = granulomatosis with polyangiitis, LCH = Langerhans cell histiocytosis, SLE = systemic lupus erythematosus.
Sarcoidosis in a 26-year-old woman who presented with a nasal mass. (a) Axial contrast-enhanced computed tomographic (CT) image shows mildly enhancing polypoid lesions (arrow) in the anterior nasal cavity along the nasal septum and opacification of the right maxillary sinus (*). (b) Axial T2-weighted magnetic resonance (MR) image shows soft-tissue lesions (arrow) with heterogeneous signal intensity filling the nasal cavities bilaterally, as well as right maxillary sinus opacification (*). Biopsy of the nasal polypoid lesion revealed noncaseating granulomas, consistent with sarcoidosis.

**Hereditary GD**

Chronic GD is a hereditary disorder, most commonly X chromosome–linked, that results in immune system compromise as well as granuloma formation in many organs (29). The disease is most commonly pediatric, although more adult cases are being identified (29). When chronic GD manifests in the head and neck, patients typically present with lymphadenopathy (29,30). Because patients are immunocompromised, atypical bacterial and fungal infections may cause sinonasal inflammation and abscess formation in the head and neck.

**Other Causes of Granuloma Formation**

Although the following diseases are not primarily granulomatous in nature, relapsing polychondritis, LCH, SLE, and rheumatoid arthritis may result in secondary granuloma formation as part of their pathologic manifestation. Relapsing polychondritis is thought to be due to an immunologic reaction to type II cartilage (31). In the head and neck, it affects auricular, nasal, and respiratory cartilage and causes orbital inflammation and aud iovestibular dysfunction (11,32). LCH is characterized by abnormal proliferation of histiocytes and predominantly affects children (33,34). Although LCH has classically been defined as a neoplastic process, some advocate its classification as an autoimmune process, given the immune-mediated pathophysiology associated with LCH (4,34). Patients with LCH may present with otitis media, mastoiditis,
bone lesions and soft-tissue masses in the head and neck, and intracranial extension of disease (33–36). SLE is caused by deposition of antibodies and immune complexes, and in the head and neck it may be associated with rash, lymphadenopathy, and laryngeal and nasal cartilage involvement (11).

Rheumatoid arthritis is an autoimmune disorder that results in chronic systemic inflammation. Treatment with antitumor necrosis factor therapies has improved outcomes in patients with rheumatoid arthritis but may result in increased susceptibility to granulomatous pathogens, including *M tuberculosis*, *H capsulatum*, and *Listeria monocytogenes* (37). Rheumatoid arthritis and SLE have also been associated with interstitial granulomatous dermatitis (38), an inflammatory lesion of the skin.

Chemicals such as cocaine, talc, and beryllium may produce pathologic granulomas as a sequela of inhalation or ingestion (4,26).

**Radiologic Manifestations**

**Sinonasal Cavity**
The most common GDs with clinical sinonasal manifestations are GPA and Churg-Strauss syndrome. Both of these entities commonly manifest with sinusitis and polyposis (6,8). Sinonasal sarcoidosis is rare and occurs in about 1% of patients who have systemic sarcoidosis (20). Sarcoid granulomas may cause nodular thickening of the nasal septum (Fig 1) (26,39). In patients with GPA, granuloma formation may lead to osteous erosion and progressive destruction of cartilage (40,41). Nasal septal perforation and the resultant collapse of nasal cartilage may produce a saddle-nose deformity in patients with GPA (6). In addition to its association with GPA, nasal septal perforation due to granulomatous pressure erosions and cartilage destruction is seen in sarcoidosis and relapsing polychondritis (31,41).
Other GD processes such as tuberculosis, syphilis, toxic exposure (eg, cocaine, beryllium), and even leprosy may produce nasal septal perforation and mucosal ulceration. Trauma (eg, iatrogenic, nose picking) and lymphoma are other important causes to consider (20,41,42).

Sinonasal GD most commonly manifests as sinusitis clinically and as mucosal thickening, polyposis, and bone sclerosis from chronic inflammation radiologically (Figs 2, 3). In GD, there is a predilection for maxillary and ethmoid sinus involvement, and the frontal sinuses are almost always spared (20). Nasal cavity involvement commonly precedes paranasal sinus disease in GD (20). In addition to sinus mucosal thickening, sinonasal findings that should prompt inclusion of GD in the differential diagnosis include osseous and cartilaginous erosion,
nasal septal perforation (Fig 4), and intraorbital extension of sinonasal disease (Fig 5). Sinonasal GD may spread intracranially, commonly extending through the cribriform plate (41).

**Orbit**

The most common GD processes that affect the orbit are sarcoidosis and GPA (43). Ophthalmic lesions are diagnosed in 25%–80% of sarcoidosis cases (26,28,43). Sarcoid involvement of the orbit most commonly manifests as uveitis, which cannot be diagnosed radiologically, and chronic dacryoadenitis due to lacrimal gland involvement, which can manifest at imaging as enlargement of the gland (28,44). Sarcoidosis may also involve the optic nerve or nerve sheath, sometimes causing a masslike appearance (43,44). Orbital involvement is seen in 18%–50% of patients with GPA (43), and manifestations include scleritis, lacrimal gland enlargement, and orbital masses. Nasolacrimal obstruction and eyelid fistula formation may also occur in GPA. Although ocular tuberculosis is rare and most commonly manifests as chorioretinitis and uveitis, chorionic masses may also be seen (14,15), with metastasis, melanoma, and other GDs as important differential diagnoses at imaging (14). Another differential consideration for an orbital mass is idiopathic orbital inflammation, an inflammatory condition that usually is a diagnosis of exclusion (45). Idiopathic orbital inflammation has been described as part of a spectrum of immunoglobulin G4 (IgG4)–related disease, a systemic inflammatory process that may also affect the thyroid, salivary and pituitary glands, lymph nodes, and sinonasal cavities in the head and neck (46).

Orbital soft-tissue masses in GD avidly enhance at imaging and frequently involve the extraocular muscles and lacrimal glands (Figs 6, 7) (Table 2). In these cases, lymphoma and idiopathic orbital inflammation are major differential considerations. Orbital masses may extend intracranially via osseous erosion or venous channels, resulting in meningeal nodularity, thickening, and enhancement. As a clinical pearl, granulomatous orbital apex masses are seen in GPA (Fig 8) and sarcoidosis (43), and these masses may also spread intracranially along skull base fissures or foramina. Of note, intracranial lesions are often seen separately from the orbital lesion. Therefore, a thorough imaging survey of the intracranial structures is extremely important when an orbital lesion is identified (Figs 7, 9).

### Table 2: Imaging Findings of Orbital Manifestations of GDs and Differential Diagnosis

<table>
<thead>
<tr>
<th>Imaging Finding</th>
<th>Commonly Associated GDs</th>
<th>Differential Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Orbital mass</td>
<td>GPA + SA + TB +</td>
<td>Lymphoma, IOI, IgG4-related disease, melanoma, metastasis</td>
</tr>
<tr>
<td>Orbital apex mass</td>
<td>GPA + SA + TB −</td>
<td>Lymphoma, IOI, Sjögren disease, IgG4-related disease</td>
</tr>
<tr>
<td>Lacrimal gland enlargement</td>
<td>GPA + SA + TB −</td>
<td>Lymphoma, IOI, Sjögren disease, IgG4-related disease</td>
</tr>
<tr>
<td>Extraocular muscle enlargement</td>
<td>GPA + SA + TB −</td>
<td>Lymphoma, thyroid orbitopathy, IOI, IgG4-related disease</td>
</tr>
<tr>
<td>Optic nerve or nerve sheath enlargement</td>
<td>GPA + SA + TB −</td>
<td>Lymphoma, meningioma, optic neuritis</td>
</tr>
</tbody>
</table>

Note.—Symbols in columns indicate the frequency with which a finding occurs, with “+” indicating a common finding and “−” an uncommon finding. IOI = idiopathic orbital inflammation, SA = sarcoidosis, TB = tuberculosis.
Figure 8. GPA in a 54-year-old man who presented with hemoptysis and diplopia. Axial T2-weighted (a) and contrast-enhanced fat-suppressed T1-weighted (b) MR images show bilateral lesions at the orbital apex (arrows) that demonstrate low T2 signal intensity in a and avid enhancement in b, with the left lesion larger than the right lesion. Note the abnormal flow voids in the left temporal region, findings consistent with a known arteriovenous malformation.

Temporal Bone and Skull Base
Some GDs may involve the osseous structures of the skull base and affect the meninges, cranial nerves, and other soft tissues (Table 3). GPA, tuberculosis, syphilis, and infection with nontuberculous mycobacteria may involve the temporal bone and cause otitis media and mastoiditis (19,40,47) (Fig 10), and late-stage syphilis may cause otic demineralization (19) with clinical symptoms of hearing dysfunction. Rarely, patients with Churg-Strauss syndrome may develop otitis media or sensorineural hearing loss, especially in late-stage disease (10). LCH often involves the temporal bone and manifests as otitis media and mastoiditis in children as a sequela of mastoid infiltration, which may be evident at imaging (Fig 11).

Sarcoidosis, GPA, tuberculosis, and syphilis can also affect the central skull base, manifesting as meningeal and cranial nerve inflammation (28,40,48). At imaging, meningeal inflammation may appear focal or diffuse and may show dural and leptomeningeal patterns of involvement. However, GD is most typically evident as leptomeningeal enhancement in the skull base or posterior fossa at imaging (Fig 12). Meningeal findings may be secondary to direct extension from the orbit, sinus, or skull base or due to hematogenous dissemination (26,48). Pituitary and hypothalamic involvement have been described in sarcoidosis, GPA, tuberculosis, and LCH, and patients may present with diabetes insipidus, hypogonadism, or hypoprolactinemia (28,40,49,50). At imaging, the pituitary gland and hypothalamus may show enlargement and heterogeneous or homogeneous avid enhancement (Fig 13).

Cranial Nerves
With regard to neural involvement, certain GDs are more commonly associated with cranial nerve pathologic conditions. In sarcoidosis, the facial nerve is most commonly involved and demonstrates enhancement or thickening at MR imaging (26). In fact, bilateral facial nerve palsy should prompt one to consider sarcoidosis as a possible cause (28,47). Although it is rare, the
Figure 10. GPA in a 56-year-old woman who presented with disturbed consciousness. (a) Axial T2-weighted MR image shows edema with sulcal effacement in the right temporal lobe (arrow). Note the opacification of the right mastoid air cells (arrowhead). (b) Axial contrast-enhanced T1-weighted MR image shows enhancement of the right mastoid air cells (arrow) and thickened dura (arrowhead).

Table 3: Imaging Findings of Temporal Bone or Skull Base Manifestations of GDs and Differential Diagnosis

<table>
<thead>
<tr>
<th>Imaging Finding</th>
<th>Commonly Associated GDs</th>
<th>Differential Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Middle ear or mastoid air cell opacification</td>
<td>GPA: +, SA: −, CSS: +, TB: +, Syphilis: +, Leprosy: −</td>
<td>Bacterial or viral infection, postsurgical effect, LCH</td>
</tr>
<tr>
<td>Osseous demineralization or erosion</td>
<td>GPA: +, SA: −, CSS: −, TB: +, Syphilis: +, Leprosy: +</td>
<td>Cholesteatoma, LCH</td>
</tr>
<tr>
<td>Cranial nerve thickening and enhancement</td>
<td>GPA: +, SA: −, CSS: −, TB: +, Syphilis: −, Leprosy: −</td>
<td>Neoplasm, infection (eg, Lyme disease)</td>
</tr>
<tr>
<td>Pituitary and hypothalamic involvement</td>
<td>GPA: −, SA: +, CSS: −, TB: +, Syphilis: −, Leprosy: −</td>
<td>LCH, lymphocytic hypophysitis, neoplasm (eg, metastasis, lymphoma), IgG4-related disease</td>
</tr>
</tbody>
</table>

Note.—Symbols in columns indicate the frequency with which a finding occurs, with “+” indicating a common finding and “−” an uncommon finding. CSS = Churg-Strauss syndrome, SA = sarcoidosis, TB = tuberculosis.

Vascular Involvement

There are GDs shown histopathologically to cause vasculitis, such as Churg-Strauss syndrome,
Figure 11. LCH in a 1-year-old boy who presented with recurrent otitis media. (a) Axial contrast-enhanced CT image shows a heterogeneously enhancing lesion centered in the right mastoid process (arrow). (b) Axial CT image (bone window) shows osseous destruction in the mastoid process of the right temporal bone (arrow). The right mastoid air cells and tympanic cavity are completely opacified (arrowhead). Partial opacification of the left mastoid air cells and tympanic cavity is also seen.

Aerodigestive Tract
The majority of pharyngeal GDs involve the mucosa. Sarcoidosis may cause nodular or diffuse infiltration of supraglottic structures, a finding seen in about 6% of patients (Fig 17) (56). Pharyngeal involvement is rare in patients with GPA and may be attributed to postnasal drip or even fistulas from sinonasal disease, resulting in pharyngitis (26). Pharyngeal tuberculosis manifests as oropharyngeal nodules and ulceration and parapharyngeal and retropharyngeal abscesses (14). Mucosal lesions resulting from tuberculosis are often clinically indistinguishable from malignancy (14).

Mucosal and submucosal involvement also predominates in GD of the larynx and trachea and may progress to fibrosis, scarring, and calcification. Subglottic stenosis is the most characteristic laryngeal lesion in GPA and occurs in 16%–23% of cases as a result of inflammation and fibrosis (6,57,58). Relapsing polychondritis most frequently causes increased tracheobronchial wall density or calcification, as well as...
Figure 13. LCH in a 43-year-old man who presented with diabetes insipidus. Sagittal nonenhanced (a) and contrast-enhanced (b) T1-weighted MR images show a bulbous homogeneously enhancing mass involving the hypothalamus and pituitary stalk (arrow). The posterior bright spot of the pituitary gland is not seen (arrowhead in a).

Figure 14. GPA in a 67-year-old man who presented with multiple cranial nerve impairments. (a, b) Axial T1-weighted (a) and T2-weighted (b) MR images demonstrate soft-tissue thickening in the nasopharynx (arrows) and opacification of the mastoid air cells bilaterally. (c) Axial contrast-enhanced fat-suppressed T1-weighted MR image shows abnormal diffuse enhancement of the mucosa, torus tubarius, eustachian tubes (arrows), and longus colli muscles (☆) that extends to the jugular foramina bilaterally (arrowheads).
Figure 15. Sarcoidosis in a 53-year-old man who presented with a pulsatile mass in the left side of the neck. Axial contrast-enhanced T1-weighted MR image shows a well-demarcated enhancing mass surrounding the left common carotid artery (arrow). Biopsy of the mass revealed sarcoidosis.

Figure 16. GPA in a 41-year-old woman who presented with worsening left-sided mixed hearing loss. (a) Axial contrast-enhanced T1-weighted MR image shows a large defect in the left nasal cavity and ethmoid sinus (*), with destruction of the clivus. Diffuse enhancement of the mastoid air cells (arrow) is due to inflammation secondary to eustachian tube obstruction. (b) Axial contrast-enhanced CT image shows extensive destruction of the nasal cavity, including a large nasal septal defect (*) and osseous defects involving the lateral wall of the sphenoid sinus, clivus, and left petrous apex. Note the encasement of the basilar artery (arrow). (c) Three-dimensional volume-rendered image from MR angiography shows severe narrowing of the basilar artery (arrow).
smooth or nodular airway wall thickening with cartilage destruction (27,32) (Fig 18). It tends to spare the posterior tracheobronchial membrane, an imaging finding that can help differentiate it from other diseases such as GPA and amyloidosis. Sarcoidosis that manifests in the larynx is rare and may cause diffuse laryngeal thickening, small nodular lesions, or localized infiltrative lesions that are mostly submucosal (56,59). Tuberculosis in the larynx and trachea is also rare and may cause narrowing or obstruction due to edema or chronic fibrosis (59) (Fig 19). Laryngeal histoplasmosis and blastomycosis manifest as ulcerated mucosal lesions, especially in their disseminated and chronic forms, and may be misdiagnosed as malignancy at direct inspection (22,26,60).

In pharyngeal GD, mucosal hyperenhancement may be evident at imaging, although this finding is nonspecific, and palatal erosion may be seen (Table 3). Glottic and paraglottic nodularity or thickening may also be visualized at imaging. When imaging the larynx, radiologists can look for subglottic stenosis as soft tissue medial to the cricoid cartilage, a finding that always is abnormal (Fig 20a). Tracheobronchial narrowing,
cartilage thickening, and calcification may also be seen to affect the airways at imaging (Fig 20b). Aerodigestive tract GD manifestations that include glottic or paraglottic thickening, ulcerated lesions, and airway narrowing may also characterize neoplasia. As such, malignancy should be at the forefront in the differential diagnosis.

Oral Cavity and Buccal or Masticator Space
Cervicofacial actinomycosis is a granulomatous infection that occasionally causes head and neck mass lesions. Often related to poor dental hygiene or dental manipulation, these lesions occur in the oral cavity and masticator space (25,61). These masses are often misdiagnosed as malignancy because they have a subacute clinical course and are difficult to diagnose microbiologically (25). Radiologic findings of actinomycosis include an enhancing soft-tissue mass with a hypoattenuating center and surrounding inflammatory changes (25,26) (Fig 21). Regional lymphadenopathy is uncommon, a finding that may help distinguish the lesion from malignancy (25,61). Tuberculosis is another granulomatous differential consideration when a mass lesion is seen in the masticator space. The tuberculous mass may result from coalescent necrotic lymphadenitis and can extend across fascial planes in the head and neck. Tuberculosis
may also cause oral or tongue ulcerations associated with adjacent tonsillar enlargement (14). Oral cavity mucosal inflammation and ulceration and palatal osteonecrosis may be seen in GPA, although these findings are rare (6).

**Salivary Glands**

Sarcoidosis is the classic GD that affects the salivary glands, and 6%–30% of patients with sarcoidosis show parotid gland involvement (28,62). At CT and MR imaging, the parotid glands in patients with sarcoidosis may show multiple benign-appearing, noncavitating, intraparotid masses (“foamy parotids”) or diffuse glandular enlargement (Fig 17). Intense radio-tracer uptake in the salivary glands at gallium 67 scintigraphy (the “panda” sign) is also characteristic of sarcoidosis (28,51). In patients with tuberculosis, parotid gland involvement predominates (Fig 22), but submandibular gland disease may also be seen at imaging (62). GPA may occasionally involve the salivary glands and manifest as heterogeneous enlargement and hyperenhancement at imaging (6,40).

**Lymph Nodes**

In patients with GD, the lymph nodes have a varied appearance and tend to show a predilection for the posterior triangle and jugular chain (14,28). Cat-scratch disease tends to cause...
Figure 23. Chronic granulomatous disease in a 1-year-old girl who presented with neck swelling. Axial contrast-enhanced CT image shows bilateral necrotic lymphadenopathy (arrows).

Figure 24. Tuberculosis in a 27-year-old man who presented with left-sided neck swelling. Coronal contrast-enhanced CT image shows multiple rim-enhancing low-attenuation lymph nodes in the left jugular and spinal accessory chains (arrows).

regional adenopathy, which can be enhancing or necrotic at imaging (16,17). Chronic granulomatous disease most often manifests in the head and neck as cervical lymphadenopathy (29,30). This may be a suppurative adenitis from a microbial infection, which manifests at imaging as necrotic lymphadenitis (Fig 23), or it may be lymphadenopathy from chronic granulomatous infiltration, which appears more homogeneous at imaging. Syphilis, leprosy, and SLE may have associated cervical lymphadenopathy, although such adenopathy is without distinguishing features (11,26,63). Lymphadenopathy is the most common manifestation of tuberculosis in the head and neck (14,18). Tuberculous adenitis classically appears at imaging as low-attenuation or necrotic lymph nodes (Fig 24), but it may also show avid enhancement at earlier stages (14,15,18). One-third of patients with sarcoidosis present with cervical lymphadenopathy (28), which at imaging tends to be homogeneous in attenuation, signal intensity, and enhancement. Intraparotid lymph nodes are commonly involved (18). Note that calcification in cervical lymph nodes with tuberculosis and sarcoidosis involvement may not be seen as commonly as calcification in the mediastinum (17).

Summary

GD in the head and neck has extensive and varied manifestations. The challenge that radiologists face is to narrow down the differential considerations to the most probable causes on the basis of imaging findings. Although many imaging findings in the head and neck are not specific for one disease entity, this article has discussed imaging findings in multiple head and neck organ systems that should raise suspicion for a GD process. The ability to recognize manifestations of GD at imaging allows radiologists to provide useful recommendations to the referring clinician and leads to appropriate clinical management.

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Pages 1243–1244
In addition to its association with GPA, nasal septal perforation due to granulomatous pressure erosions and cartilage destruction is seen in sarcoidosis and relapsing polychondritis. Other GD processes such as tuberculosis, syphilis, toxic exposure (eg, cocaine, beryllium), and even leprosy may produce nasal septal perforation and mucosal ulceration. Trauma (eg, iatrogenic, nose picking) and lymphoma are other important causes to consider.

Pages 1244–1245
In addition to sinus mucosal thickening, sinonasal findings that should prompt inclusion of GD in the differential diagnosis include osseous and cartilaginous erosion, nasal septal perforation, and intraorbital extension of sinonasal disease. Sinonasal GD may spread intracranially, commonly extending through the cribriform plate.

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Page 1246
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Pages 1251–1252
When imaging the larynx, radiologists can look for subglottic stenosis as soft tissue medial to the cricoid cartilage, a finding that always is abnormal. Tracheobronchial narrowing, cartilage thickening, and calcification may also be seen to affect the airways at imaging. Aerodigestive tract GD manifestations that include glottic or paraglottic thickening, ulcerated lesions, and airway narrowing may also characterize neoplasia. As such, malignancy should be at the forefront in the differential diagnosis.