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REVIEW

Infectious Diseases in the Head and Neck with Eosinophilia

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Abstract
Eosinophilia, defined as an elevated eosinophil count either in blood or tissue, has diverse implications for diagnosing and managing various diseases. Elevated eosinophil levels are often associated with conditions such as allergic reactions, autoimmune disorders, and specific infections. In the realm of head and neck pathology, eosinophilia can offer valuable insights into underlying infectious processes, which are often challenging to diagnose due to their overlap with other inflammatory and allergic conditions. This review describes the roles of blood and tissue eosinophilia in several infectious processes affecting the head and neck region. We focus on nine key conditions: allergic fungal rhinosinusitis, mycetoma, invasive fungal rhinosinusitis, rhinosporidiosis, baylisascariasis, toxocariasis, onchocerciasis, loiasis, and histoplasmosis. Allergic fungal rhinosinusitis, for example, is a hypersensitivity reaction to fungal antigens and is frequently associated with significant eosinophilic inflammation. Conversely, mycetoma, invasive fungal rhinosinusitis, and rhinosporidiosis may include eosinophils as part of a mixed inflammatory infiltrate. Histoplasmosis may also induce systemic eosinophilia as an atypical immune response to fungal infection. Additionally, baylisascariasis, toxocariasis, onchocerciasis, and loiasis are parasitic infections that often lead to systemic eosinophilia. By exploring these conditions, this review elucidates how identification of eosinophilia contributes to the diagnostic process. Understanding the association between eosinophilia and these infectious processes involving the head and neck is crucial for enhancing diagnostic accuracy, differentiating between similarly presenting conditions, and guiding effective treatment strategies.

Keywords Eosinophilia · Fungal sinusitis · Histoplasmosis · Rhinosinusitis

Introduction

Eosinophilia, characterized by an elevated number of eosinophils in the blood, tissues or organs, is commonly associated with allergic reactions, autoimmune diseases, and certain infections. Eosinophilia can be defined as either an increased peripheral eosinophil count in the blood (defined by absolute eosinophil count) or an increased number of eosinophils in the tissue as evaluated on histology. The absolute eosinophil count is the total WBC count multiplied by the percentage of eosinophils and normal is considered up to 500 cells/microL. Eosinophilia can indicate underlying infectious or inflammatory processes in the head and neck region. This describes how eosinophilia intersects with specific infectious conditions affecting the head and neck region. Understanding the relationship between eosinophilia and these infections is crucial for accurate diagnosis and effective treatment (Table 1).

This review provides a comprehensive examination of the role of eosinophilia in several infectious processes affecting the head and neck region. We focus on nine key conditions: allergic fungal rhinosinusitis, mycetoma, invasive fungal rhinosinusitis, histoplasmosis, rhinosporidiosis, baylisascariasis, toxocariasis, onchocerciasis, and loiasis and discuss the clinical and pathologic findings associated with each.

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Table 1 Summary of eosinophilia in the blood (eosinophil count) and tissue by disease process

Disease Process	Systemic/Blood AEC	Tissue Eosinophilia
Allergic fungal rhinosinusitis	+ + +	+ + +
Mycetoma	-	±
Invasive fungal rhinosinusitis	-	±
Histoplasmosis	±	±
Rhinosporidiosis	-	±
Bylisascariasis	+ + +	±
Toxocariasis	+ + +	-
Onchocerciasis	+ + +	±
Loiasis	+ + +	±

Non-Invasive Fungal Rhinosinusitis

Allergic Fungal Rhinosinusitis

Allergic fungal rhinosinusitis (AFRS) is an eosinophil-mediated hypersensitivity reaction to non-invasive fungal elements, with Aspergillus species being the most common triggers, followed by other dematiaceous fungi [1]. This condition typically affects the nasal cavity and paranasal sinuses and is observed equally in male and female patients. AFRS is most frequently seen in immunocompetent atopic children and young adults, particularly in warm and humid regions, such as the southern and southwestern United States.

The development of AFRS involves multiple immunologic pathways, primarily type I and type III hypersensitivity reactions. In type I hypersensitivity, the immune system produces allergen-specific IgE in response to various fungal antigens. Meanwhile, type III hypersensitivity is characterized by an increase in allergen-specific IgG, which forms immune complexes with fungal antigens. Recent studies suggest additional mechanisms involving superantigen and non-IgE-mediated pathways [2].

Patients with AFRS typically present with chronic nasal congestion, facial pain, nasal discharge, and nasal polyps. These symptoms can be severe and significantly impact the quality of life, potentially leading to complications from sinus obstruction. Many patients exhibit peripheral (blood) eosinophilia and increased IgE antibody levels, and serological tests for fungal antigens and specific IgE can be beneficial for diagnosis.

Imaging studies, particularly CT scans, reveal opacification and expansion of the nasal cavity and affected sinuses. On T2-weighted MRI, AFRS appears as marked hypointensity, often accompanied by thick, non-obstructive secretions that are hyperintense [3]. Macroscopically, the respiratory mucosa may present as edematous and polypoid, with thick, putty-like, foul-smelling secretions.

Histologic examination of biopsies from patients with AFRS shows characteristic inflammation composed predominantly of eosinophils, along with Charcot-Leyden crystal deposition, regardless of the fungal organism involved. Fungal hyphae and spores may also be observed, with the background containing necrotic debris from the respiratory epithelium and amorphous mucoid elements. Due to the variability in the number of fungal elements seen under light microscopy, special stains like Grocott-Gomori methenamine silver (GMS) or periodic acid–Schiff (PAS) are often used to highlight fungal hyphae effectively. (Fig. 1).

Mycetoma

Mycetoma, also known as fungal ball, is a form of non-invasive fungal rhinosinusitis characterized by a dense collection of fungal hyphae that forms a mass-like structure outside of the respiratory mucosa. It typically affects a single sinus, with the maxillary sinus most involved, followed by the ethmoid and sphenoid sinuses. Mycetoma is most frequently observed in middle-aged to elderly individuals, with a mean age of 64 years, and it has a noted female predominance [4].

The condition often develops slowly and may be asymptomatic in its early stage. When symptoms do occur, they resemble those of chronic rhinosinusitis and may include facial pain, rhinorrhea, nasal obstruction, nasal odor, and headache. Peripheral eosinophilia is often not seen.

The clinicopathologic diagnostic criteria proposed by deShazo include radiologic evidence of sinus opacification, the presence of mucoid or clay-like substance clinically or macroscopically, and microscopically identifiable dense hyphal aggregates located away from the mucosa, without evidence of tissue invasion [5].

While eosinophil-predominant inflammation in the tissue is not typically associated with mycetoma, it is not uncommon to find a mixture of eosinophils, histiocytes, and giant cells encasing the "fungal ball." (Fig. 2) This histologic feature is also known as the Splendore-Hoeppli phenomenon [6].

Invasive Fungal Rhinosinusitis

Invasive fungal rhinosinusitis (IFRS) is a rapidly progressing and potentially life-threatening fungal infection primarily affecting immunocompromised adults, including those with diabetes mellitus or undergoing immunosuppressive therapy. Although rare, cases have also been reported in the pediatric population [7].

IFRS is commonly caused by saprotrophic fungi, particularly those in the Zygomycetes class, such as Mucor and Rhizopus species, as well as Aspergillus species. Each of

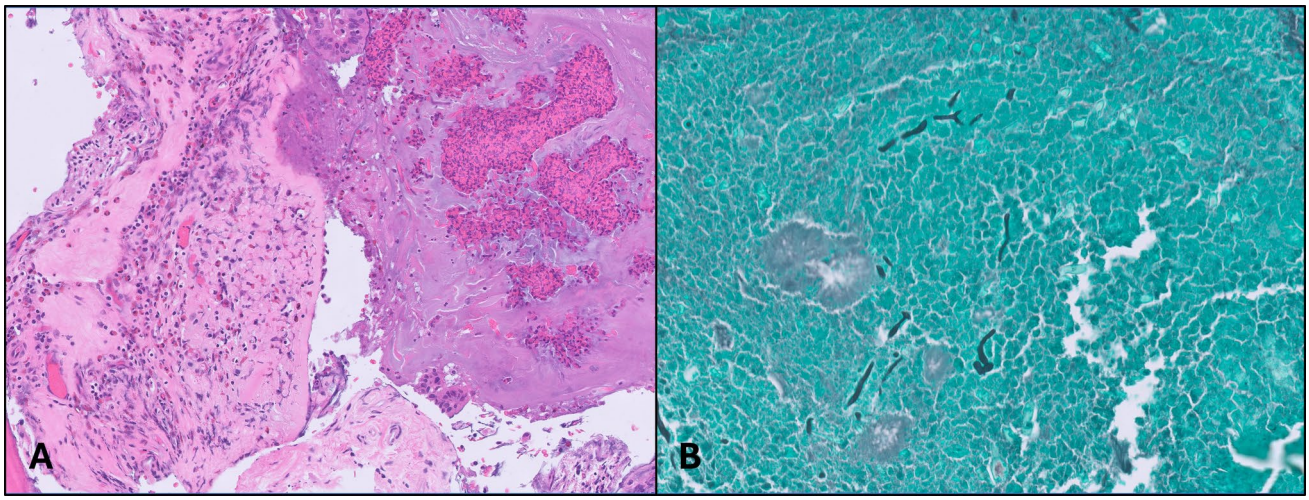

Fig. 1 Allergic fungal rhinosinusitis. **A** Stromal eosinophilia and allergic mucin with Charcot-Leyden crystals, H&E 20x. **B** GMS showing fungal hyphae, GMS 40x

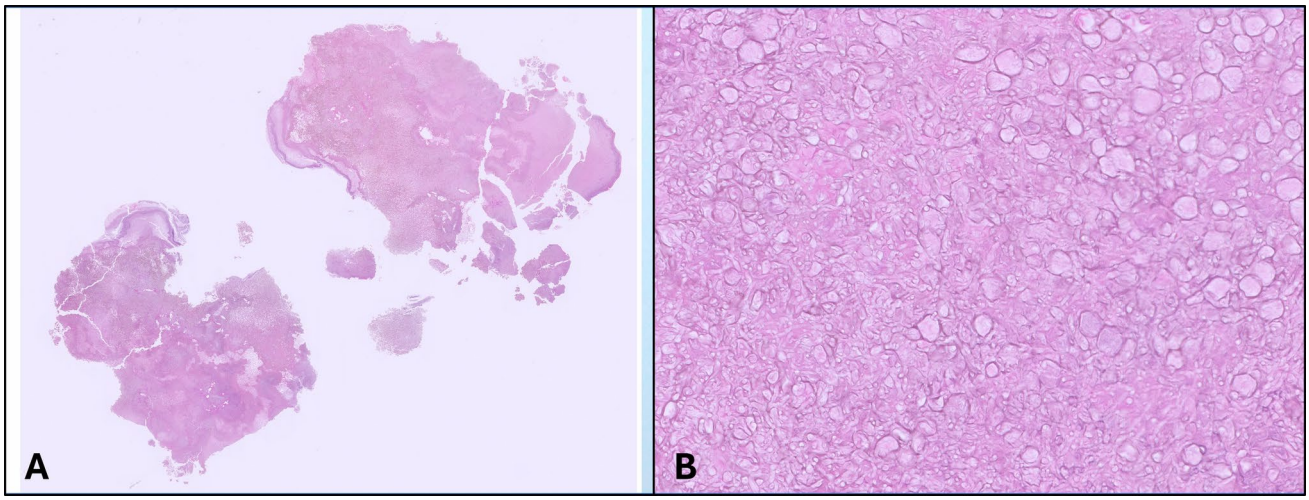

Fig. 2 Mycetoma. **A** Fragments of fungal hyphae, H&E 0.5x. **B** Fragments of fungal hyphae, H&E 40x

these fungi has a distinct hyphal morphology. The infection typically begins in the sinonasal tract, often through prior colonization or inhalation of the spores, and quickly spreads to nearby structures, including the eyes, brain, hard palate, and cavernous sinus [1].

Patients with IFRS often present with symptoms resembling a cold, such as sinonasal congestion with blood-tinged discharge, facial swelling, and the presence of black-crusted necrotic tissue. The infection can advance rapidly, leading to severe complications like orbital and cerebral involvement. Orbital symptoms may include proptosis, ptosis, ophthalmoplegia, or vision loss, while cerebral involvement can result in cranial nerve palsy or altered mental status. Peripheral eosinophilia is often not seen.

Diagnosis relies on a high index of clinical suspicion, supported by imaging studies (e.g., CT or MRI) that reveal mucosal thickening or space-occupying lesions accompanied by bone and soft tissue destruction. Direct examination of tissue samples from affected areas is also critical [8]. Grossly, the involved tissue typically appears hemorrhagic and dark. Immediate histologic evaluation can be done with intraoperative frozen section analysis, which reveals fungal hyphae that are angiotropic (involving blood vessels) or located near the vessels inside the soft tissue. Eosinophils, when present in the tissue, are part of a limited mixed inflammatory response in the background of hemorrhagic and necrotic mucosa. (Fig. 3).

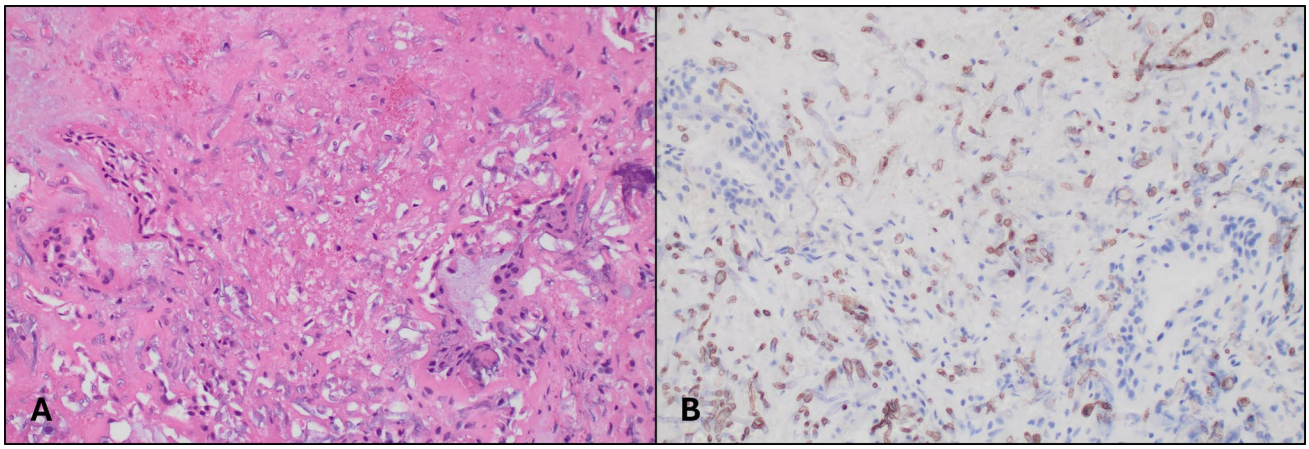

Fig. 3 Invasive fungal rhinosinusitis. **A** Fungal hyphae invading tissue, H&E 20x. **B** In situ hybridization probe targeting Fusarium species, ISH 20x

The morphology of the fungi plays a significant role in diagnosis: Zygomycetes are characterized by broad, "ribbon-like" non-septate hyphae with 90-degree branching, whereas Aspergillus species display thin, septate hyphae with 45-degree branching. Special stains, such as PAS and GMS, can help visualize the fungal forms. Additionally, tissue samples should be sent for definitive microbiologic speciation, such as next generation sequencing [9]. In cases where the microbiological workup is negative, in situ hybridization probes targeting Rhizopus, Aspergillus, or Fusarium species can provide further diagnostic assistance.

Histoplasmosis

Histoplasmosis is a fungal infection caused by the dimorphic fungus Histoplasma capsulatum, which thrives in soil enriched with bird or bat droppings. This infection is primarily endemic in the central and eastern United States, particularly in regions surrounding the Ohio and Mississippi River valleys. Humans typically acquire histoplasmosis by inhaling airborne spores, leading to pulmonary infections. However, the fungus can also disseminate to other parts of the body [10].

In immunocompetent individuals, histoplasmosis often presents as a mild respiratory illness, resembling influenza, with symptoms such as cough, fever, and fatigue. In contrast, immunocompromised patients may experience more severe forms of the disease, including disseminated histoplasmosis, which can affect various organs, including the oral cavity, nasal cavity, and larynx [11, 12].

While peripheral eosinophilia is not a classic feature of histoplasmosis, some studies have reported elevated eosinophil counts in certain patients, particularly those with chronic or disseminated forms of the disease [13, 14]. This eosinophilia may indicate an atypical immune response to the fungal infection and could be associated with the inflammatory processes occurring in affected tissues.

In the head and neck region, histoplasmosis can lead to significant complications, including cervical lymphadenopathy and oropharyngeal lesions [11, 12]. Patients may present with swollen and tender lymph nodes, as well as ulcerative lesions in the mouth or throat, which can cause difficulty swallowing and contribute to systemic symptoms like fever and malaise.

Diagnosing histoplasmosis involves a combination of clinical evaluation, serological tests, and tissue biopsies. Although peripheral eosinophilia is not a definitive diagnostic criterion, its presence alongside clinical symptoms can help in assessing the severity of the infection and the immune response. Histopathological examination of affected tissues often show granulomas and lymphohistiocytic aggregates with a mixed inflammatory infiltrate, including eosinophils. Special stains highlight the fungus as small (2–4 μm), uniform, narrow-based budding yeasts clustered within host cells. (Fig. 4).

As a dimorphic fungus, Histoplasma capsulatum appears as slow-growing yeasts that transition to fluffy white mold colonies within 1 to 6 weeks. The diagnosis is often achieved through serological tests, such as the Histoplasma antigen test using enzyme immunoassay (EIA). The urine antigen test is typically the initial screening method due to its high sensitivity and rapid turnaround time. This antigen test is especially useful for acute and disseminated disease, often seen in immunocompromised patients who may not produce antibodies for serological tests. However, it's important to note that the antigen test can cross-react with other fungi [15].

The EIA urine antigen test is commonly used alongside the immunodiffusion test and complement fixation (CF)

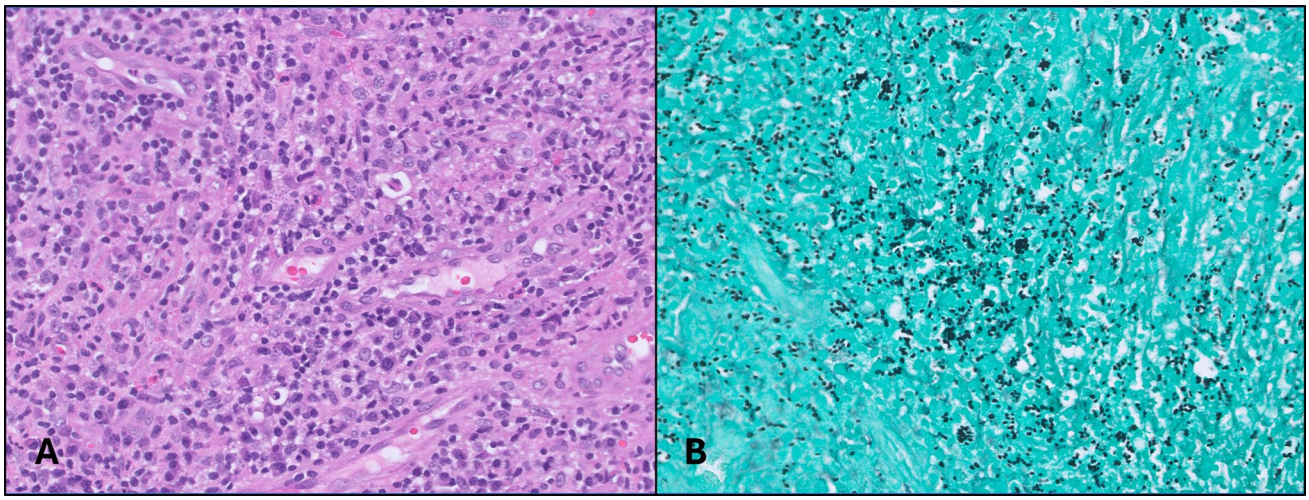

Fig. 4 Histoplasmosis involving base of tongue. **A** Mixed inflammation with yeast forms, H&E, 40x. **B** GMS highlighting yeast forms, GMS, 40x

serum antibody testing [16]. The immunodiffusion test, also known as the double immunodiffusion or Ouchterlony test, detects antigen–antibody precipitation by identifying precipitin bands: H, which indicates chronic or severe acute infection, and M, which appears within weeks of acute infection. These bands can persist for months or even years after the infection has resolved. The CF test detects antibodies against Histoplasma capsulatum by incubating the patient's serum with antigens and complement, allowing assessment of complement cascade activation. Additionally, molecular techniques, including PCR-based and LAMP-based assays, have significantly improved the detection and management of histoplasmosis.

Rhinosporidiosis

Rhinosporidiosis is a chronic infection caused by the sporulating fungus-like parasite Rhinosporidium seeberi. It primarily affects the nasal cavity and nasopharynx, but it can also involve other areas of the head and neck, including the larynx, trachea, and oropharynx. The condition is endemic in regions such as India, Sri Lanka, and Brazil, and it is more commonly observed in men than in women, particularly in their third to fourth decade of life [17].

The mode of transmission is believed to occur through water, dust, or digital trauma, allowing the endospore to penetrate the mucosa of the nasal cavity. Once inside, it matures into a sporangium within the submucosal layer. After maturation, the sporangia burst, releasing endospores into the surrounding tissue.

Common symptoms of rhinosporidiosis include nasal obstruction, epistaxis (nosebleeds), and occasional facial swelling. The disease can affect various sites in the body, presenting in four clinical forms: nasal, ocular, cutaneous, and disseminated. Peripheral eosinophilia is not often seen. Radiologically, rhinosporidiosis appears as a moderately enhancing lobulated or irregular soft tissue mass on contrast-enhanced CT scans [18].

Grossly, the infection manifests as a polypoid or pedunculated mass that resembles sinonasal inflammatory polyps. The cut surface typically exhibits a pink or purple hue with mucoid yellow pinpoint spots. Histopathologic assessment is the gold standard for diagnosis. Histologic examination reveals a polypoid lesion containing numerous large (30–100 µm in diameter), round, thick-walled sporangia filled with countless sporangiospores. The large spores (5–10 µm in diameter) are more mature and tend to cluster towards the center, while the smaller spores (1–2 µm in diameter) are more dispersed.

Systemic eosinophilia is not common. Mixed chronic inflammation is often present, featuring lymphocytes, plasma cells, and eosinophils. The rupture of sporangia can trigger an acute inflammatory response. The overlying epithelium may exhibit hyperplasia or squamous metaplasia. Notably, Rhinosporidium seeberi is positive for special stains such as PAS and mucicarmine, aiding in its identification [3].

Baylisascariasis

Baylisascariasis is a rare, serious zoonotic infection caused by the larval stage of the raccoon roundworm, Baylisascaris procyonis. This parasite is predominantly found in North America, particularly in the Northeast, Midwest, and West

Coast, regions where the distribution aligns with the raccoon population [19]. Individuals living in these areas or those who handle raccoons are at greater risk of infection, as are children and individuals with compromised immune systems.

Humans become accidental hosts by ingesting the eggs, typically through contaminated soil or food. Once the larvae hatch in the gut, they can migrate throughout the body, leading to neural larva migrans, ocular larva migrans, and visceral larva migrans [20]. Symptoms can range from subclinical or asymptomatic cases to severe manifestations involving the central nervous system, which may present as altered mental status, seizures, or ataxia. Ocular involvement can result in retinal damage or vision loss.

Diagnosing baylisascariasis can be challenging due to the nonspecific nature of symptoms. A detailed clinical history is essential to rule out other potential causes, and a combination of tests is often necessary. Patients typically exhibit peripheral eosinophilia, which can be detected in complete blood count or cerebral spinal fluid analyses [21]. While histologic assessment is not always practical, early diagnosis can be facilitated through enzyme-linked immunosorbent assay (ELISA), immunoblotting, or immunofluorescence staining of frozen tissue sections to identify the presence of IgG antibodies [22]. In tissue the eggs are oval shaped, thick shelled and up to 85 µm in size. The surrounding mixed inflammatory response with have eosinophils.

Toxocariasis

Toxocariasis is a zoonotic infection caused by the larval stages of the roundworms Toxocara canis and Toxocara cati, which are commonly found in dogs and cats, respectively. This infection primarily affects humans who accidentally ingest the eggs, often through contaminated soil, food, or hands, particularly in areas where pets are prevalent [23]. Children are especially at risk due to their frequent outdoor play and hand-to-mouth behaviors.

Once ingested, the eggs hatch in the intestines, and the larvae can migrate through various tissues, leading to two primary forms of the disease: visceral larva migrans (VLM) and ocular larva migrans (OLM). Symptoms of VLM may include fever, cough, abdominal pain, and hepatomegaly, while OLM can lead to vision problems, including retinal damage and potential blindness.

Diagnosing toxocariasis can be challenging due to its nonspecific symptoms and the need for a comprehensive clinical history. While imaging studies can help visualize affected organs, a definitive diagnosis typically requires a combination of clinical evaluation and laboratory tests. Peripheral eosinophilia is commonly observed in infected individuals and can be detected through a complete blood count. Early detection is often facilitated by serological tests that identify specific antibodies against Toxocara. The ELISA using Toxocara excretory-secretory antigens is the preferred and most widely used serologic test for diagnosis. Additionally, newer technologies using chimeric proteins derived from T. canis are being investigated for their diagnostic potential [24]. The eggs are spherical, thick-shelled with a pitted surface, up to 85 µm in size. The adults can be up to 18 cm long. Tissue biopsies do not commonly have eosinophils.

Onchocerciasis

Onchocerciasis, commonly known as river blindness, is a parasitic infection caused by the filarial worm Onchocerca volvulus. The disease is primarily transmitted to humans through the bite of infected blackflies, particularly in regions near fast-flowing rivers in Africa, South America, and Yemen. Once the larvae enter the human host, they mature into adult worms, which then can produce microfilariae that can migrate throughout the body.

The presence of microfilariae leads to a range of symptoms that include both ocular and dermatological manifestations, with the potential for systemic involvement. One of the most serious complications of onchocerciasis is its impact on vision. Microfilariae can migrate to the eyes causing inflammation and a spectrum of eye diseases such as conjunctivitis and sclerosing keratitis. If left untreated, these conditions can progress to vision impairment or irreversible complete blindness.

In addition to ocular symptoms, onchocerciasis manifests through various skin conditions, including intense itching, rashes, and the formation of nodules known as onchocercomas, typically located in the head and neck region. These nodules mark areas where adult worms reside and are often palpable beneath the skin. Over time, progressive keratitis can result in skin pigmentation changes and atrophy. This leads to distinct patterns: “leopard skin” describes spotty depigmentation, “lizard skin” refers to atrophy accompanied by scales, and “elephant skin” indicates hyperkeratosis. Furthermore, the chronic nature of the infection can contribute to systemic symptoms such as fatigue and malaise [25].

The diagnosis of onchocerciasis typically involves a combination of physical examination, laboratory findings, and histological assessment. Peripheral eosinophilia and lymphadenopathy are notable features of the disease, reflecting the immune response to microfilariae. Elevated eosinophil levels can be identified through a complete blood count, serving as an important diagnostic marker.

Since microfilariae of Onchocerca do not exhibit periodicity, they can be detected microscopically at any time via skin snips from affected areas. The biopsies will show a

mixed inflammatory infiltrate including eosinophils. The microfilaria measure 250–300 µm in length and are not sheathed with nuclei which do not extend to the tip of the tail. Serological testing is also useful for confirming the diagnosis, particularly in cases where microfilariae are difficult to find. In endemic regions, rapid diagnostic tests or ELISA assays detecting antibodies specific to a 16 kDa antigen of O. volvulus (Ov16) have served as a gold standard for diagnosis since its discovery in 1991 [26].

Loiasis

Loiasis, often referred to as African eye worm disease, is a parasitic infection caused by the filarial worm Loa loa. This disease is transmitted to humans through the bite of infected deer flies, predominantly found in the rainforest regions of West and Central Africa. Once inside the body, the adult worms migrate through the bloodstream and tissues, producing microfilariae that exhibit diurnal periodicity, leading to a variety of clinical manifestations [27].

The head and neck areas are frequently affected by the presence of adult worms, along with the associated inflammatory responses. A defining characteristic of loiasis is the migration of these adult worms, which can often be observed moving across the conjunctiva of the eye, causing significant discomfort and irritation. This ocular involvement is not only alarming but can also result in more severe complications, including vision impairment.

Other clinical manifestations include Calabar swelling, which occurs due to the migration of microfilariae in the lymphatic system, leading to painless, localized angioedema around the joints and in the head and neck region. Additional symptoms may involve lymphadenopathy, fever, and malaise, all stemming from the inflammatory response triggered by the migrating adult worms.

Peripheral eosinophilia is a common feature of loiasis, reflecting the immune response to the filarial infection. Elevated eosinophil levels can be detected through a complete blood count, serving as a significant diagnostic marker. This increase in eosinophils often accompanies other symptoms such as pruritus and localized swelling, particularly in the head and neck area.

Diagnosing loiasis requires a combination of clinical evaluation and laboratory tests, tissue for histopathologic examination is rarely received. The adults can measure up to 70 mm in length, the microfilaria measure up to 300 µm which are sheathed. The presence of the adult worm in the subconjunctiva is a striking clinical sign, while peripheral blood examinations can reveal microfilariae, confirming the diagnosis. Eosinophilia, along with clinical symptoms, plays a crucial role in supporting the diagnosis. Given the diurnal periodicity of microfilariae and the long pre-patent period, several molecular assays, including polymerase chain reaction (PCR)-based, mainly real-time quantitative PCR, and loop-mediated isothermal amplification (LAMP)-based methods, have been proposed to enhance diagnostic accuracy [28].

Conclusion

Eosinophilia, defined as an elevated eosinophil count in the body can be a valuable diagnostic clue in identifying various infectious processes in the head and neck (Table 1). In this paper we reviewed the clinical and pathologic findings in allergic fungal rhinosinusitis, mycetoma, invasive fungal rhinosinusitis, histoplasmosis, rhinosporidiosis, baylisascariasis, toxocariasis, onchocerciasis, and loiasis. Each condition presents unique challenges in diagnosis and treatment. By understanding each disease process and the association between eosinophilia is crucial for enhancing diagnostic accuracy, differentiating between similarly presenting conditions, and guiding effective treatment strategies for optimal patient outcomes.

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Code Availability No software or code was used in this review paper.

Declarations

Conflict of interest The authors declare no competing interests.

Ethical Approval This article does not contain any studies with human participants or animals performed by any of the authors. As this is a review paper, IRB approval was not required.

Consent to Participate For this type of study informed consent is not required.

Consent for Publication For this type of study consent for publication is not required.

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