Other Special Diseases and Treatment

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29.1 Syphilis of Otorhinolaryngopharynx

Etiology

Syphilis is a systemic infectious disease caused by Treponema pallidum, which can involve multiple or single organs. Syphilis can be divided into congenital and acquired syphilis. Congenital syphilis is transmitted through mother to child. Acquired syphilis is transmitted mainly through sexual pathways, blood transfusion and breastfeeding. Damaged skin and mucous membrane can also be infected by body fluids and saliva with pathogens.

Treponema pallidum can intrude skin or mucous membrane of the human body, which can invade multiple organs and become complex. The disease is asymptomatic for many years but later causes various various symptoms. The early invasion of the skin and mucous membrane is manifested by an ulceration. In the late stage, the heart, central nervous system, viscera, and bones can be invaded. There is no internal and external toxin in Treponema pallidum, and the pathogenesis is still not clear. The strength of the immune system determines the outcome of the infection. The organism can produce humoral immunity and delayed allergy to the pathogen, and local granulomatosis is formed. There are two basic diseases of syphilis: (1) focal occlusive arteritis and perivascular inflammation; (2) syphilis granuloma. The progression of invading pathogens can be divided into three stages: first stage: hard chancre period, second stage: syphilis rash and third stage: syphilis tumor stage.

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Clinical Manifestations

Syphilis has a certain specificity in Otolaryngology, and it is sometimes easy to diagnose with the epidemiological characteristics of the patients. However, some patients begin to appear only in general inflammation, which is difficult for clinical diagnosis.

1. Ears Syphilis: early congenital syphilis patients are mostly in the first 1~2 years of birth, and late congenital syphilis often occurs at the age of 8~10 years. The patient has deafness and vertigo. Hutchinson triad is called labyrinthine, interstitial keratitis and serrated teeth in patients with congenital syphilis. If the bone is broken into the labyrinthine fistula or annular ligament to soften the stapes loose, it appears normal and the middle ear fistula test is positive, known as Herbert syndrome. Labyrinthine and facial paralysis can also occur in patients with acquired syphilis. The symptoms of acquired inner ear syphilis and late congenital inner ear syphilis are basically the same, and facial nerve paralysis and labyrinthine can occur.
2. Nasal Syphilis is divided into congenital and postnatal nature. The syphilis may infringe on the nose at all stages, which is seen in the three stages. Primary syphilis is rare. Second stage syphilis often involves nasal septum and anterior inferior turbinate. The local mucosa is red, swollen and erosive. It can form white mucous patches. It is called syphilitic rhinitis, which is highly infectious. The characteristics of early congenital syphilis are similar to that of this period, and can be found at 1~3 months after birth. Secretion block make the children crying uneasy, breathing and suckling are difficult. Late congenital syphilis and acquired syphilis are manifested in three stages, and the nasal septum is mainly damaged which causes the nasal shape to change. At the age of 3 to puberty, the Hutchinson triple sign is often accompanied by the collapse of the nose. Patients with syphilis tumor invasion of nasal septum and the hard palate bone have a perforated nasal septum and hard palate, a saddle nose and even nasal damage.

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3. Pharynx Syphilis: the pharynx lymphoid tissue is rich, and the syphilis may occur in the pharynx at various stages. Pharynx syphilis is rare, and is usually on one side tonsil chancre, ipsilateral cervical lymph node hardens second stage syphilis of pharynx in about 2 months after the emergence of scarlet fever and rash manifested by pharyngitis, throat congestion, swollen tonsils, oral and pharyngeal mucosa often appears with gray round or oval infiltrating.

   Third stage is often accompanied by generalized lymphadenopathy and diffuse rash. The third stage of syphilis of the pharynx occurs years after the initial infection, syphilis lesions by tumor infiltration, softening, ulceration, and finally the formation of scar contracture, can appear. There is perforation of the hard palate pharyngeal tissue, adhesion, stenosis or atresia deformity.
4. Laryngeal Syphilis
   (a) A period of Laryngeal Syphilis: is extremely rare. It can appear as an epiglottis chancre.
   (b) Second Stage Laryngeal Syphilis: Is similar to catarrhal laryngitis, there is laryngeal mucosa diffuse hyperemia. In addition, the mucous plaque can occur in the vocal cords and the dipper area, which is often accompanied by systemic rash and pharynx mucous plaque.
   (c) Third stage laryngeal syphilis: first or second stage syphilis is slightly more common, the common symptoms vary, change of voice to a light, or hoarseness, cough and a mild pharyngache (this is different from the laryngeal tuberculosis), dysphagia (epiglottis, tongue and lateral pharyngeal wall involvement). There are four common types: (1) gumma, mostly located in epiglottis, is a dip-like epiglottis, dark red or purple red arytenoid cartilage, involving the vocal cord or ventricular zone (2) The ulcer formation, the gum swells after the formation of an ulcer covered with yellow rotting tissue, the surrounding tissue is hyperemic There is soft periostitis and necrosis, the ulcer develops in depth, caused by laryngeal cartilage necrosis deformity. If the thyroid cartilage or cricoid cartilage is necrotic, then laryngeal stenosis will occur. The scar and adhesion, due to ulcers and perichondritis after healing, fibrous tissue hyperplasia evolved between the epiglottis and tongue, adhesion between or on both sides of the vocal cords, can also occur in arytenoid cartilage causing a deformity.

Diagnosis

1. There is a history of an unprotected sexual contact with a syphilis partner.
2. The clinical symptoms and signs conform to the characteristics of mucous syphilis.
3. Histopathological examination reveals histologic evidence of mucous syphilis.
4. Serological screening tests for syphilis and positive test for syphilis specific diagnosis are positive.

Treatment

1. Treatment: penicillin is the first choice for syphilis. Erythromycin can be used in patients allergic to penicillin.
2. Symptomatic treatment with saline, boric acid, hydrogen peroxide solution, Furacilin Solution to clean the wound, and keep it clean; for the repair of the scar deformity, a plastic Operation is feasible.

Otolaryngology syphilis has its specific manifestations. However, it is difficult to diagnose patients with general inflammation and deny the history of epidemiology. Therefore, serious medical examination and medical history collection are very important for the diagnosis.

29.2 AIDS Manifestation at Otorhinolaryngopharyngology Head and Neck

40~70% of AIDS patients have otolaryngology head and neck lesions. The neck of AIDS patients is mainly manifested as cervical lymph node enlargement, which is one of the early symptoms. Due to HIV infection, follicular hyperplasia is often seen, and cervical lymph node enlargement is common, especially in the posterior cervical trigone area. Kaposi's sarcoma can also occur in the skin of the head and neck. When it invades the lymph nodes of the neck, they increase rapidly, a on Hodgkin's lymphoma and mycobacterial infection should also be considered when there are neck masses. A fine needle aspiration is helpful for the diagnosis and differential diagnosis. Squamous cell carcinoma in the head and neck is also more common in AIDS patients. The virus infection can cause parotid enlargement.

Etiology

HIV is a virus in the family lentivirus. It is a single strand RNA virus, which has a lifetime in the host. When HIV invades the human body, it can adsorb on the surface of CD4+T cells, enter the cells through the cell membrane and integrate into the DNA of CD4+T cells, resulting in the decrease of CD4+T cell number, the CD4+T lymphocyte dysfunction and cause an abnormal immune activation. The inhibitory/cytotoxic lymphocytes (CD8+T lymphocytes) in HIV infected patients show normal or increasing number of functions, which may contribute to further immunodeficiency and lead to CD4+T/CD8+T < 1. HIV can also infect

non lymphocyte, such as macrophages, small neuroglia cells, various endothelial and epithelial cells. HIV can adhere to the surface of dendritic cells in the lymph nodes but does not invade the cells. HIV infection results in the number of functional of T cells, B cells, natural killer cells, monocytes and macrophages, which are characterized by opportunistic infections, malignancies, neurological dysfunction and other syndromes.

Clinical Manifestations

There are a variety of general manifestations of HIV infection, but it is often non-specific. It is easy to be misdiagnosed. Some patients have HIV infection before physical examination or preoperative examination. In general, infected people usually have infection and new organisms in the otolaryngology. These symptoms and signs can occur in one site or in multiple sites. In addition, the degree of immunosuppression in patients can affect the severity of the infection, the probability of occurrence, and the response of the infection to a new biological treatment.

1. Ear Manifestations: the ear manifestations of AIDS patients show multiple hemorrhagic card Posey's sarcoma, Pneumocystis carinii infection, otitis media, hearing impairment etc. Multiple hemorrhagic Posey's sarcoma can occur in the auricle and external auditory canal, showing red purple patches or nodules slightly higher to skin surface, with different sizes, unequally from several millimeters to several centimeters. The outer otic pneumocystis carinii infection manifestates the multilocular cyst, and the protozoon could be found on the biopsy. The pneumocystis serous otitis media often occurs in adults, and HIV can be detected from the tympanic effusion. Among the infants, acute otitis media caused by the fungi, protozoan, virus or myobacteria can be observed from the pus culture. The HIV easily invades the central nervous system or auditory nerve, commonly leading to a sensory neural hearing loss at an early stage.
2. Rhinal and Rhinosinuses' Manifestation

   The AIDS patient's nose and rhinosinus mainly manifest all kinds of symptoms and signs evoked by the amebic protozoa, cytomegalovirus, herpes virus, cryptococcal infection etc. The amebic protozoa infection could cause the rhinal and rhinosinus mucosa to swell and cause a pus snot or the epistaxis to the rhinobyon and other symptoms. The cytomegalovirus infection can cause suppurated rhinitis, the granules and the erythema existing at the rhinomucosa, and the cytomegalovirus inclusion body and the squamous metaplasia at the intravascular endothelial cells can be seen at rhinomucosal biopsy. The herpes virus infection can cause a herpetic ulcer, expanding from the rhinovestibule to the rhinoseptum and the adjacent rhinoala and facial segment. The cryptococcal infection can cause the whole group rhinosinusitis. Besides, the lymphoma and Kaposis sarcoma can also occur.
3. Lingual and Laryngeal Manifestation

   The AIDS patients' oral and pharyngeal manifestations are mainly monilial infection, villous mucosal leukoderma, single herpes, amygdalitis, and Kaposi's sarcoma, etc.
4. Laryngeal Manifestation

   The AIDS patients' laryngeal manifestations are mainly the Kaposi's sarcoma and the monilial infection, that finally leads to hoarseness, laryngostridor and laryngostasis.
5. Cervicocranial Manifestation

   The AIDS patients cervical manifestation is based on early symptoms. It mainly manifests as cervicolymphatic swelling, Kaposis sarcoma, non-hodgkin lymphoma, mycobacteria infection and other infections, squamous cell carcinoma, parotid swellings etc. The cervical lymphatic swelling is commonly seen and is HIV evoked. Follicular hyperplasia is usually seen at the supracervical triangle region. The Kaposis sarcoma can occur on the cervicocranial skin, and when it invades the cervical lymph nodes, they will rapidly enlarge. The cervicocranial squamous-cell carcinoma is also oftenly seen. The virus infection and other infection can cause the parotid to swell, and it is also tagged as the AIDS omen.

Diagnosis

Based on the disease history, clinic manifestation and lab examination results, diagnosis could be made.

1. History Detail Enquiry

   Especially in the homosexuality, promiscious sexual behavior, intravenous drug-taking, blood transfusion and other histories should be taken.
2. Infection-Possibility Manifestation

   Such as to the pneumocystic carnii pneumonia and the Kaposi's sarcoma patient, this is an important diagnosis. Long-term and low-grade fever, diarrhea, emaciation and systemic lymphatic swelling combined with oral, pharyngeal and other regions' monilial infection, this is all similar to the AIDS premonitory symptoms and they should be paid attention to cautiously.
3. Immunity-Deficiency-Metrics CD4+T Cells Reduction

   The Center for Disease Control and Prevention (CDC) revised-diagnosis-gist noticed in 1991 that the CD4+T < 200/mm^3 could be diagnosed as the AIDS. Besides; there should be CD4+T/CD8+T < 1.
4. HIV Lab Diagnosis

   It include virus isolation culture, antigen detection, antibody detection, virus nucleic acid detection, etc.

When the primary screening test result is positive, recheck to avoid false positive results and if latter result is positive, ascertain an HIV infection existence. HIV antibody can be commonly detected 2 months after the time of infection.

Treatment

1. Anti-HIV-virus drug
2. Immunoregulation drug
3. Prevention and treatment of opportunistic infections.
4. Traditional Chinese Medicine treatment

AIDS otorhinopharyngocervicocranial manifestations vary, but they lack specificity, so it's not hard to misdiagnose. The AIDS incidence is higher, and the medical personnel should enhance the vigilance to the AIDS and improve its cognition.

29.3 Leprosy of Otorhinolaryngopharyngology

Etiology

Leprosy is a chronic inflammatory disease evoked by the mycobacterium leprae. The pathogenic bacteria's detection rate is related to the leprosy types. Relatively more mycobacterium leprae can be found in the lepra lepromatosa patients' mucosa, skin and lymph nodes; but the tuberculoid leprosy's pathogenic bacteria isn't easy to detect and it mainly damages the skin, mucosa and the peripheral nerves. The rhinoleprosy is the most favored type, and the nose is also the earliest invaded region. Leprosy is mainly acquired through touching and after being infected, the incubation period is very long and the lesion develops slowly.

Clinical Manifestation

Except the systemic manifestations, its otorhinopharyngolaryngeal manifestations are as follows.

1. Otic Leprosy

   It mainly occurs at the auricle, especially the ear lobule. Its main manifestations are the local infiltration, nodular formation, ulceration, cicatrix, and the skin folds and tissue defects etc. The great auricular nerve bulges and there is pain on palpation. These are the diagnosis valuable symptoms. The facial nerve spasm and (or) paralysis is caused by the stimulation from the lesion invasion.
2. Rhinal Leprosy

   Rhinal leprosy is the most common one in the otorhinopharyngolaryngeal leprosy, and nose is one of the leprous earliest invaded regions. It is nearly all lepra lepromatosa. The early lesion invades the rhinovestibular hair follicle, which causes loss of the rhinothrix, there is an ulcer formation and the rhinocavum submucosal nobular infiltration, then the nubular diabrosis could cause the refractory ulcer or cicatrical synechia; at the late stage, the mucosal gland bodies shrink, the dry rhinocavum incrustates and presents a change similar to the atrophic rhinitis. In severe patients, their rhinoseptum cartilage perforates, the rhinocolumella damages, the rhinal apex collapses and abutts to the upper lip, so it is easy to differentiate it from the saddle nose caused by the atrophic rhinitis and syphilis. In rhinoleprosy secretion, abundant of the mycobacterium leprae often exist, so this disease has high infectivity.
3. Pharyngeal Leprosy

   It is mostly caused by the desposingly extending rhinal lepra lepromatosa. Except presenting the acute edema at the early stage, the pharyngomucosa commonly manifest as dryness, escharosis, infiltrated nubule and ulceration; if necrosis exists, the open rhinolalia symptom and eating reflux symptom may occurs.
4. Laryngeal Leprosy

   It is mainly secondary to the rhinal and the pharyngeal leprosy, favorably occurring at the epiglottis root and its anterior commissure, secondarily at the arytenoid epiglottis folds and the ventricular cords. It manifests the nobular infiltration and ulceration, and finally cicatrizates. On examination, there is epiglottis congestion or paleness, incrassation, crinked deformity, and even damage can occur. Hoarseness, stridor and slight dyspnea can occur.

   Leprosy commonly incubates for a long time and develops slowly. But under some conditions, like climate change, infection, emotional change and others, the acute or subacute symptoms suddenly occur, which is also called the leprosy reaction. The reaction contains two types: the type I and the type II. The type I is the cellular immunity allergic reaction, manifesting as skin redness and swelling, local pyrexia, nerve trunk lesion sudden augmentation and obvious pain, but no systemic symptoms. The type II is the immunity complex allergic reaction, consisting the systemic symptoms, such as fever, headache and systemic lymphatic swelling, joint gall, existance of a skin erythema, pain and acute iris conjunctivitis, acute orchitis, etc.

Diagnosis

Primary diagnosis can be made based on the leprosy contraction history and the typical damage manifestations at the skin, the mucosal and the peripheral nerves, and the diagnosis could be ascertained if the mycobacterium leprae is found on examination from the secretions got from the lesion or the biopsy. The upper respiratory tract leprosy lesion should be differentiated from tuberculosis and the syphilis.

Treatment

Systemic anti-leprosy treatment is the main method, accompanied with each otorhinopharyngolaryngeal regional symptomatic therapy.

1. Systemic Treatment

   Main leprosy bacillus's therapeutic drugs include the dapsone, the rifampicin, protionamide, clofazimine etc., and the recent proposition is the alternative-three drugs combination.
2. Symptomatic Treatment

   It is mainly used to dispose the leprosous reaction, from preventing and avoiding the aggravated abnormality. Its main therapeutic drugs include glucocorticoid, thalidomide (thalidomide), etc. If there is relatively more severe neurodynia, procaine could help the local sealing.
3. Local Treatment

   Clearing the rhinocavum crusta prevents the secondary infection. Lubricate the rhinocavum with the liquid paraffin, peppermint oil etc., or relieve the symptoms by daubing the rhinocavum with the Aureomycin or the erythromycin ointment. The local ulcerations can be cauterized with the 30% trichoroacetic acid (TCS).

29.4 Diphtheria of Otorhinolaryngopharyngology

Etiology

Diphtheria is a kind of acute respiratory tract inflammation caused by the corynebacterium diphteriae. Its main lesion includes the pharyngeal and the laryngeal mucosal congestional swelling, necrosis and exudation, which develops its specific hard-deciduous incanus pseudo-membrane and the systemic poisoning symptoms evoked by the diptheria exotoxin. The air droplets or the bacterial-contaminated towels, table ware, toys, books and newspapers and other methods, mainly transmit it. Diphtheritis commonly occurs in autumn, winter and spring, and is mainly in infants younger than 10-year-old, of which the 2~5-year-old incidences are the highest. Because of improved living conditions and widely arranged prophylactic vaccination, the diphtheric incidence has evidently decreased, and is rarely seen at recent time.

Clinical Manifestation

1. Pharyngeal Diphtheritis

   It is the most common type of the diphtheritis, with 80% incidence. In clinic, it is divided into three types: the limited type, the dispersal type and the poisoning type.

   (a) The Limited Type

   This type onset is slow, its systemic manifestations are fever, weakness, inappetence etc. Its local symptoms are mild, with slight pharyngeal pain. On the tonsil, the incanus pseudomembrane can be seen, and the pseudomembrane surpasses the palatoglossal arch, covering the soft palatine and the uvula or the postpharyngeal wall. The pseudomembrane adheres closely to the tissues, indelible, and if forcibly peeled, bleeding will occur. With the pseudomembranous smear or culture, the corynebacterium diphtheriae will be seen.

   (b) The Dispersal Type

   Its lesion often surpasses the tonsil, involves the palatina arch, soft palatine, uvula or the postpharyngeal wall, rhinopharynx or the larynx. The pseudomembrane is lamellar. The systemic symptoms are relatively evident, including slight or moderate fever, accompanied by weakness, the inappetence, nausea, emesis, headache and cervical lymphadenectasis.

   (c) The Poisoning Type

   This type onsets acutely, the pseudomembrane expands rapidly, and the systemic poisoning symptoms soon occur, such as high fever, fidget, polypnea, pallor, lips' cyanosis, cold limbs, fine-speedy pulse, low blood pressure, arrhythmia, etc. The pharyngomucosa, tonsil, uvula and palatina arch obviously swell. There is cervical lymph nodes tumefication, soft tissues edema, even neck augments like a 'bovine neck', and severe complications can occur, such as the myocarditis, leading to heart failure, cardiogenc shock (CGS), etc.
2. Laryngeal Diphtheritis

   It occurs in 20% diphtheritis cases, it is also caused by the pharyngo-diphtheritis despondingly extending to the larynx, and occasionally it originates from the larynx. It onsets is slow, its tussiculation sounds like barking and there is hoarseness. When the laryngeal tumidness or the pseudomembrane obstructs the glottis, it causes inhaled dyspnea and laryngeal stridor, the three-concave sign and cyanosis can occur if the obstruction becomes more severe, even the patients will die from suffocation if the obstructions aren't relieved. The laryngeal lesion extends downwards to the trachea and the bronchus to evoke lower respiratory tract obstruction.
3. Rhinodiphtheritis

   It is rarely seen, and it includes the primary type and the secondary type. Its rhinal symptoms are similar to the common rhinitis, manifestating as the rhinobyon and a running nose (often stained with blood). At examinations, the rhinovestibular and the upper lip skin versenkbar and erosion can be seen, and it is found that the rhinocavum mucosal surface is covered with the incanus pseudomembrane, especially seen at the rhinoseptum, and if the pseudomembrane is removed, there will be bleeding at the ulceration.

4. Ear Diphtheritis

   It is rarely seen. It is often secondary to the rhinal and the pharyngeal diphtheritis. Nearly none of this type is primary. This type often occurs in 1~6-year-old infant. Its symptoms are similar to the common suppurative otiti media, with severe otalgia, and smelly hemorrhagic pus or the polluted pseudomembrane-like discharge after the tympanic tresis occurs.

Diagnosis

According to the disease history, the symptoms and the physical signs, combined with the bacterial examinations, the diagnosis is often not hard. But one time of the negative bacterial result can't exclude the diagnosis, so the bacterial examination should be repeated several times to ascertain the diagnosis at early stage. The bacterial examination methods include; the secretion smear scope, the immunity fluoroscopy and the bacterium culture, and if necessary, the Schick test and the immunochromatographic method should assist diagnosis.

Treatment

1. Common Treatment

   Rigorous isolation, 2~4 weeks bed rest (the severe patients should take 4~6 weeks). Pay attention to the oral and the rhinal clearance. Supply enough nutrition. Tracheotomy should be done as early as possible if laryngeal obstruction exists.
2. Etiology Treatment

   The diphtheria antitoxin should be used at the early stage, combined with intravenous sensitive antibiotics drips, whose first choice is penicillin.
3. Complications Treatment

   Pay a close attention to the cardial conditions, if heart damage exists, ask the cardiovascular physicians to assist in the treatment.

29.5 Rhinoscleroma

It is a chronic process, inflammatory granulomatous lesion; was first reported in 1870 by Habra. At the International Otorhinopharyngolaryngeal Conference convoked in 1932, it is named as the 'rhino scleroma'. It is a sporadic disease. It has been reported around the world, in China it is mostly in Shandong province, occupying 46% of the total rhinoscleroma cases.

Etiology

It usually originates from the nose, and extends to the nasal sinus, soft palatine, hard palatine, pharynx, larynx, trachea, bronchus, rhino lacrimal canals and the middle ear. Besides, this disease can sporadically supervene or secondarily occur at several respiratory regions, so it is also called the respiratory track scleroma.

Clinical Manifestation

1. Catarrhal Period

   At this stage, it manifests as the local mucosal desiccation, atrophy, escharosis and hemorrhage. Its primary symptom is the rhinobyon, and if it invades other regions, the manifestation will be the corresponding regional catarrheal symptoms. The patients can restore to normal after treatment. This period could last for several months even several years.
2. Scleroma Stage

   This stage mainly manifestates as rhinal obstruction, external rhinal deformation and nobular lumps exist in the rhinocavum, with a cartilage-like texture, often lying at the rhinovestibule, prenaris, anterior rhino-septum segment, upper lip and other regions, whose surface lightens and aubergine. and if there is secondary infection purulent scab festers could presents at the lump surface and can be smelly or not. This period could last several years or longer a time.
3. Scar Stage

   Because the cicatrix contracts, symptoms, such as closed rhinolalia, hoarseness or dyspnea and others, and physical signs such as the anterior rhinal stenosis, atresia, the nasal alae ingression, uvula disappearance, laryngopharyngostenosis, etc., can occur.

Diagnosis

1. Long term, progressive development
2. It always locates at the anterior rhinocavum with hard texture and mostly without ulceration. Outer nose transformation may occur.
3. Without partial pain.
4. Plenty of plasmocyte infiltration with lymph cells and froth cells may manifest on the biopsy under the microscope. Ascertain diagnosis with the Mikulicz cells and the Russel globules appearance and sometimes repeated biopsies are also necessary (Fig. 29.1).
5. Germiculture

   Rhinoscleroma bacillus is positive.
6. Serology Examination

   Complementary fixation butter has high reliability. Apply the early cases more than other stages'.

Treatment

1. Antibiotics Treatment

   Commonly, intramuscular 1 g/day streptomycin injection totals 60~120 g, or intramuscular kanamycin or cefazobenzidazole injection.

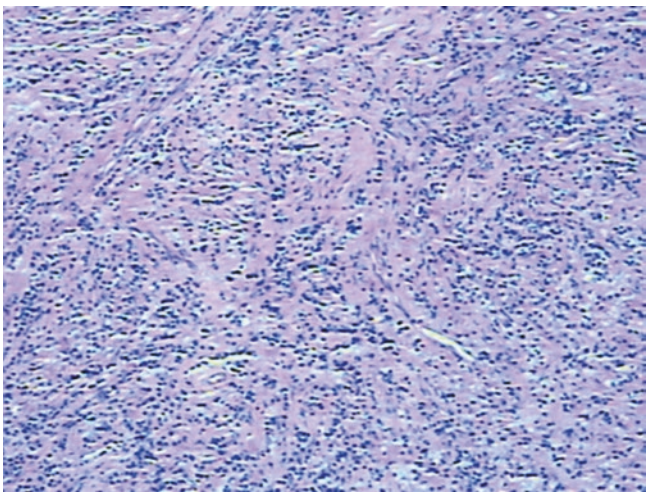

Fig. 29.1 Rhinoscleroma pathogenesis slice (HE × 10)

2. Radiotherapy

   It could remit the disease progression; total radial quantity is 40~70 Gy.

3. Operation

   Based on the conditions, scar malformation excision Operation can be applied. The scleroma excision Operation can not be applied, or it would evoke severe scar contraction. Do a tracheotomy in case of dyspnea.