8.10.3 Cysticercosis 1533

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8.10.3 Cysticercosis 1533 the prevalence of hydatidosis in sheep decreased from 26% before the vaccine was introduced to 8% 3 years after the vaccine was intro- duced. Although the results of these initial trials seem promising, further research is needed to assess the cost benefit of using these vaccines. FURTHER READING Allan JC, et al. (1992). Coproantigen detection for immunodiagnosis of echinococcosis and taeniasis in dogs and humans. Parasitology, 104, 347–55. Brunetti E, Junghanss T (2009). Update on cystic hydatid disease. Curr Opin Infect Dis, 22, 497–502. Brunetti E, et al. (2010). Expert consensus for the diagnosis and treat- ment of cystic and alveolar echinococcosis in humans. Acta Trop, 114, 1–16. Brunetti E, et al. (2011). Cystic echinococcosis: chronic, complex, and still neglected. PLoS Negl Trop Dis, 5, e1146. Budke CM, Deplazes P, Torgerson PR (2006). Global socioeconomic impact of cystic echinococcosis. Emerg Infect Dis, 12, 296–303. Craig PS, et al. (2007). Prevention and control of cystic echinococcosis. Lancet Infect Dis, 7, 385–94. Frider B, Larrieu E, Odriozola M (1999). Long-term outcome of asymptomatic liver hydatidosis. J Hepatol, 30, 228–31. Gavidia CM, et al. (2008). Diagnosis of cystic echinococcosis, central Peruvian Highlands. Emerg Infect Dis, 14, 260–6. Larrieu E, et al. (2013). Pilot field trial of the EG95 vaccine against ovine cystic echinococcosis in Rio Negro, Argentina: early impact and preliminary data. Acta Trop, 127, 143–51. McManus DP, Thompson RCA (2003). Molecular epidemiology of cystic echinococcosis. Parasitology, 127, S37–51. Macpherson CNL, et al. (1987). Portable ultrasound scanner versus serology in screening for hydatid cysts in a nomadic population. Lancet, ii, 259–91. Moro PL, et al. (1997). Epidemiology of Echinococcus granulosus
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H. Gilman ESSENTIALS Cysticercosis, infection by larvae of the pork tapeworm Taenia solium (see Chapter 8.10.1), is the most common helminthic in- fection of the human central nervous system. It accounts for up to 30% of all seizures and epilepsy in endemic countries, and travel and immigration now lead to its more frequent presentation in in- dustrialized countries. Ingestion of raw or undercooked pork can lead to infection with the T. solium cysticercus, formerly known as 'Cysticercus cellulosae', which is an encysted immature tape- worm. Once attached to the person's small intestine, the head, or scolex, evaginates from the cysticercus, anchors in the intes- tinal mucosa and develops segments (proglottids) to become an adult tapeworm. Proglottids discharged in the faeces contain tens of thousands of ova that can autoinfect the human host or pigs and, rarely, other susceptible mammals. Ingestion of T. solium ova by the faecal-oral route in those infected with adult tapeworms or their close contacts can result in development of cysticerci in various tissues, but not an adult tapeworm. The ingested ova re- lease embryos that penetrate the intestinal mucosa and migrate in the blood stream to the brain (causing neurocysticercosis), muscles, and subcutaneous tissues. Only by ingesting T. solium ova can humans develop cysticercosis. Clinical features and diagnosis—manifestations of neurocysticercosis depend on the number, location, size, and stage of the parasite cysts in the brain, as well as on the immunological response of the host. The most common syndromes are late-onset epilepsy and intra- cranial hypertension. Diagnosis is based on brain imaging studies (CT or MRI) and supported by highly specific serology. Treatment and prognosis—treatment is (1) symptomatic (e.g. anti- convulsants); shunts for intracranial hypertension in patients with hydrocephalus; and (2) antiparasitic—albendazole or praziquantel, which are generally given with steroids to control cerebral oedema; but there is no role for these drugs in inactive neurocysticercosis (i.e. calcifications with or without enhancement on CT scan). Prognosis depends mainly on whether the cysts are intraparenchymal (better prognosis) or extraparenchymal (subarachnoid or intraventricular, poorer prognosis). Introduction Known since the Hippocratic era, cysticercosis is the most common helminthic infection of the human central nervous system. It is probable that the suspicion of its origins led some religions expressly to forbid the consumption of pork. Socioeconomic improvements eradicated the infection in Europe and North America. However, endemic Taenia solium taeniasis/cysticercosis persists in most developing countries, where human cysticercosis is an important cause of epilepsy and other neurological morbidity, and porcine in- fections cause considerable economic losses to peasant farmers.

section 8 Infectious diseases 1534 Aetiology Cysticercosis is infection with the larval stage (cysticercus) of T. solium, the pork tapeworm (Chapter 8.10.1). In the life cycle of this two-host zoonotic cestode (Fig. 8.10.3.1), humans are the only definitive host and harbour the adult tapeworm by ingesting cysts in infected pork, whereas pigs are intermediate hosts by ingesting eggs in human stools. The hermaphroditic adult T. solium inhabits the human small intestine. Its head, or scolex, bears four suckers and a double crown of hooks, connected by a narrow neck to a large body (strobila) between 2 and 4 m long, composed of several hundred proglottids (Chapter 8.10.1, Fig. 8.10.1.1b, c). Gravid proglottids, each containing 50 000–60 000 fertile eggs, detach from the distal end of the worm and are excreted in the faeces. The cycle is com- pleted when pigs ingest stools contaminated with T. solium eggs. Once ingested by the pig, the invasive oncospheres in the eggs are liberated by the action of gastric acid and intestinal fluids and ac- tively penetrate the bowel wall, enter the bloodstream, and are car- ried to the muscles and other tissues where they develop into larval cysts (Chapter 8.10.1, and see Fig. 8.10.3.1). When humans ingest undercooked pork containing cysticerci, the larva evaginates in the small intestine, its scolex

attaches to the intestinal mucosa, and it begins forming proglottids. By accidentally ingesting taenia eggs, humans can also act as intermediate hosts for T. solium and develop cysticercosis. Epidemiology The availability of neuroimaging studies and the subsequent devel- opment of specific serodiagnostic tests have resulted in the identi- fication of neurocysticercosis as a frequent neurological disorder in Latin America, Africa, and Asia, where the prevalence of active epilepsy is almost twice that in Western countries. Cysticercosis was introduced from Bali to the highlands of Papua, Indonesia nearly 40 years ago. Its seroprevalence is more than 20% in many communities. Neurocysticercosis is also an emerging problem in industrialized countries, seen mainly in immigrants from endemic areas, some of whom may spread the infection as tapeworm carriers. This applies to California and other southern areas of United States of America bordering Mexico. The main sources of human cysticercosis are faecal-oral contam- ination in those carrying the tapeworm, or their contacts, and in- gestion of food contaminated with T. solium eggs. Epidemiological studies suggest that almost every newly diagnosed patient with cysticercosis has been infected by someone in their close environ- ment who is harbouring a T. solium and the tendency is to dismiss the role of environment or water in transmission. Airborne trans- mission of T. solium eggs and internal autoinfection by regurgitation of proglottids into the stomach have been suggested but not proved. Pathogenesis Any organ can be infected, but parasites survive more frequently in the nervous system, possibly because the reduced immune response. Signs and symptoms are caused by perilesional inflammation and oe- dema, mass effect, or obstruction of cerebrospinal fluid circulation. Although complete development of cysts takes 2 to 3 months, symp- toms usually develop years after the initial infection. This clinically silent period, and finding inflammation around cysts in symptomatic cases, suggests that in many cases symptoms are due to inflammatory processes associated with the recognition of the parasite by the im- mune system of the host (presumably progressing towards the death of the parasite) rather than to the presence of the parasite itself. Subarachnoid cysticerci elicit an intense inflammatory reaction causing thickening of basal leptomeninges. The optic chiasma and other cranial nerves are usually entrapped within this dense exudate, resulting in visual field defects and other cranial nerve abnormal- ities. The foramina of Luschka and Magendie can be occluded by the thickened leptomeninges, leading to hydrocephalus. Blood vessels can also be affected by the inflammatory reaction. The walls of small penetrating arteries are invaded by inflammatory cells, leading to a proliferative endarteritis with occlusion of the lumen, and this can result in cerebral infarction. Clinical features Neurocysticercosis is a pleomorphic disease, whose manifestations vary with the number, size, and topography of the lesions and the intensity of the host’s immune response to the parasites. Patients can be classified by the number, stage, and location of the cysticerci, and the presence or absence of associated inflammation or calcifications. Epilepsy, the most common presentation of neurocysticercosis, is usually the primary or sole manifestation of the disease. Seizures occur in 50–80% of patients with parenchymal brain cysts or calci- fications but are less common in other forms of the disease. Other focal signs are less frequent and include pyramidal tract signs, sen- sory deficits, signs of brainstem dysfunction, and involuntary move- ments. These manifestations usually follow a subacute or chronic course, making neurocysticercosis difficult to differentiate clinically from neoplasms or other infections of the central nervous system. Focal signs can occur abruptly in patients who develop a cere- bral infarct as a complication of subarachnoid neurocysticercosis. Human (definitive host) Pig (intermediate host) Ingestion of infected pork, poorly cooked: taeniasis Ingestion of T. solium eggs by faecal contamination: human cysticercosis Ingestion of T. solium eggs or proglottids: porcine cysticercosis Fig. 8.10.3.1 Life cycle of T. solium.

8.10.3 Cysticercosis 1535 Subarachnoid cysticerci can reach 10 cm or more in diameter (‘giant’ cysticercosis, Fig. 8.10.3.2), and exert a mass effect. Neurocysticercosis may present with increased intracranial pressure, usually from hydrocephalus secondary to basal sub- arachnoid cysticercosis or intraventricular cysts, cysticercotic arachnoiditis, or granular ependymitis. In these cases, intracra- nial hypertension develops subacutely and progresses slowly. An encephalitic picture can result from overwhelming inflammation around many parasitic cysts, a syndrome that occurs more fre- quently in younger people, especially women. In contrast, some patients tolerate hundreds of intraparenchymal cysticerci with only minor symptoms. Ocular cysticercosis can involve the posterior segment, retina, vit- reous, subconjunctiva, orbit or eyelid (Fig. 8.10.3.3). Muscular pseudohypertrophy, a rare presentation, is caused by heavy cysticercal infection of skeletal muscles (Fig. 8.10.3.4) giving a ‘Herculean’ appearance. The few cases reported are come most frequently from India. Other apparent differences in clinical manifestations between Asia and Latin America include a high frequency of subcutaneous cysts and single degenerating brain lesions in Asia. Pathology The cysticerci are liquid-filled vesicles consisting of vesicular wall and scolex (Fig. 8.10.3.5). The vesicular wall is composed of an outer, or cuticular, layer, a middle, or cellular, layer with pseudoepithelial structure, and an inner, or reticular, layer. The invaginated scolex has a head, or rostellum, armed with suckers and hooks, and a rudimentary body, or strobila, that includes the spiral canal. The macroscopic appearance of cysticerci varies in different lo- cations within the central nervous system. Cysticerci within the brain parenchyma are usually small and tend to lodge in the cere- bral cortex or basal ganglia (Fig. 8.10.3.6). Subarachnoid cysts might be small if located in the depths of cortical sulci, or grow to 5 cm or more in the basal cisterns or Sylvian fissures. Ventricular cysticerci are usually single, might or might not have a visible scolex, and might be attached to the choroid plexus or float freely in the ventricle. Spinal cysticerci are usually located in the sub- arachnoid space (rarely intramedullary). Here they can develop areas of arachnoiditis. Basal subarachnoid cysticerci can undergo a disproportionate growth of their membrane, with extension processes, resembling Fig. 8.10.3.2 Giant cysticercotic cyst (brain CT). Fig. 8.10.3.3 Intraocular cysticercosis: cysticercus in the anterior chamber of a Thai patient. Courtesy of the late Professor Sornchai Looareesuwan. Fig. 8.10.3.4 Heavy cysticercal infection of skeletal muscles. Courtesy of the late Professor Sornchai Looareesuwan.

section 8 Infectious diseases 1536 5 mm (a) (c) (e) (b) (d) Gd-enh Fig. 8.10.3.5 (a) Histopathology of a complete cysticercus removed by brain biopsy in a patient with recent onset of focal epilepsy (×4). (b) Structure of the cyst wall (×40). (c) Cerebral imaging CT enhanced. (d) MRI T2-weighted. (e) MRI T1-weighted with and without gadolinium enhancement. Copyright D. A. Warrell.

8.10.3 Cysticercosis 1537 a brunch of grapes (racemose cysticercosis, Fig. 8.10.3.7). In these cases, the scolex is frequently unidentifiable even by microscopy. Viable vesicular cysticerci elicit little inflammatory change in sur- rounding tissues because of active immune evasion mechanisms. The appearance of symptoms is interpreted as the result of immuno- logical attack from the host, in a process of degeneration that ends with the death of the parasite. Inflammatory changes in the parasite membrane and increased density of cyst fluid mark the transition between four defined stages: viable, colloidal, granular nodular, and calcified cyst. Viable cysts may coexist with degenerating cysts or calcifications. Laboratory/imaging diagnosis The pleomorphism of neurocysticercosis makes it impossible to diagnose on clinical grounds alone. In endemic regions, late-onset seizures in otherwise healthy individuals are highly suggestive of neurocysticercosis. Most of these patients are normal on neuro- logical examination. Routine neuroimaging and

serological studies are, therefore, mandatory. Finding cysticerci outside the central ner- vous system (eye, subcutaneous tissue, muscle) assists the diagnosis of neurocysticercosis. Muscular and subcutaneous cysticerci are far less common in American than in African or Asian patients with neurocysticercosis. Neuroimaging CT and MRI have markedly improved diagnostic accuracy by providing objective evidence about the topography of the lesions and the degree of the host inflammatory response to the parasite. Imaging findings in parenchymal neurocysticercosis depend on the stage of involution of cysticerci. Viable cysticerci appear as rounded cystic lesions on CT (Fig. 8.10.3.2), hypointense on T1 and FLAIR sequences on MRI (Fig. 8.10.3.6), without associated enhance- ment, whereas degenerating parasites are seen as focal enhancing lesions surrounded by oedema (Fig. 8.10.3.4c–e), and calcifications as hyperdense dots or nodules (Fig. 8.10.3.8). Disappearance of cyst fluid signals the degenerative phase and calcified nodules the re- sidual phase. Single or multiple ring-like or nodular enhancing le- sions are nonspecific and present a diagnostic challenge. Pyogenic brain abscesses, fungal abscesses, tuberculomas, toxoplasma ab- scesses, and primary or metastatic brain tumours may produce similar findings on CT or MRI. CT and MRI findings in subarachnoid neurocysticercosis are less specific. They include hydrocephalus, abnormal meningeal enhancement, and subarachnoid cysts. Cerebral angiography can show segmental narrowing or occlusion of major intracranial Fig. 8.10.3.6 Uncontrasted T1 MR image showing two intraparenchymal cysticerci with visible scolices. Fig. 8.10.3.7 Basal ‘racemose’ cysticercosis. Fig. 8.10.3.8 Calcified neurocysticercosis.

section 8 Infectious diseases 1538 arteries in patients with cerebral infarcts secondary to parasitic vasculitis. In neurocysticercosis there is rarely fever or signs of meningeal irritation; glucose levels in cerebrospinal fluid are usu- ally normal. MRI is generally better than CT for the diagnosis of neurocysticercosis, particularly in patients with basal lesions, brain- stem or intraventricular cysts, and spinal lesions. MRI is, however, less sensitive than CT for the detection of calcifications. Immunological tests Immunoblot (Western blot) using lentil-lectin purified parasite glycoprotein antigens is the best available serological test for T. solium antibodies. It performs well with serum samples and is 98% sensitive in cases with more than one active lesion, and 100% specific. Its sensitivity may drop in patients with a single cyst. Other assays using unfractionated antigens (e.g. enzyme immunoassay, ELISA) suffer from poor specificity but are more reliable when performed with cerebrospinal fluid than serum. Antigen-detection tests can provide a tool for serological moni- toring of antiparasitic therapy. Although results of serology and imaging studies might be similar, they evaluate different aspects of the disease and can be discordant in some patients. Intestinal tapeworm carriers, naturally cured patients, or nonneurological infections can have normal brain images but be positive serologic- ally. Those with only inactive lesions or a single cerebral lesion might be seronegative. Parasitological diagnosis A proportion (c.10–15%) of patients with neurocysticercosis are tapeworm carriers at the time of diagnosis, and in another 10% or so a carrier can be detected in the household. Parasitological diagnosis is difficult: eggs and proglottids are shed only intermittently in stool and are frequently missed by routine stool examination. Stool as- says to detect parasite antigens are more sensitive than microscopy, but are not widely available. A recently described serological test for tapeworm carriers might improve detection. Diagnostic criteria A set of diagnostic criteria based on neuroimaging studies, sero- logical tests, clinical presentation, and exposure history has been proposed by Del Brutto and colleagues. Besides absolute demon- stration of the presence of the parasite, ‘major’ criteria (including typical findings on neuroimaging, demonstration of specific anticysticercal antibodies, or the presence of typical cigar-shaped calcifications in muscle) are combined with ‘minor’ criteria and

epidemiological data to suggest a probable or possible diagnosis. Application of these criteria should improve the consistency of diagnosis. Treatment Because of the clinical and pathological pleomorphism of neurocysticercosis, precise assessment of the viability and size of cysts, the location of parasites, and the severity of the host's immune response is important before planning treatment. Symptomatic treatment is very important. Seizures secondary to parenchymal neurocysticercosis can usually be controlled with anticonvulsants. However, the optimal duration of anticonvulsant therapy in patients with neurocysticercosis has not been deter- mined, and it is difficult to withdraw this treatment. Prognostic factors associated with recurrence of seizures include the develop- ment of parenchymal brain calcifications, and occurrence of recur- rent seizures or multiple brain cysts before starting antiparasitic therapy. Antiparasitic agents destroy viable cysts and are associated with fewer seizures in the long-term follow-up. Antiparasitic treat- ment in patients with a single enhancing lesion seem to improve radiological resolution and decrease the chance of seizure relapses, albeit the magnitude of this effect is small. Albendazole is the drug of choice for antiparasitic treatment of cerebral cysticercosis (15 mg/kg per day for 7 to 15 days, with steroids), although a re- cently described single-day praziquantel regimen (75–100 mg/ kg, in three doses at 2-h intervals, followed by steroids 6 h later) demonstrated similar cestocidal activity in patients with few cysts. The combination of albendazole with praziquantel is more ef- fective in patients with multiple parenchymal cysts. Longer courses may be required in patients with many lesions or subarachnoid cysticercosis. Transient worsening of neurological symptoms can be expected during antiparasitc therapy, secondary to the perilesional inflammatory reaction. There is no role for antiparasitic drugs in inactive neurocysticercosis (i.e. calcifications with or without en- hancement on CT scan) since the parasites are dead. Between the second and fifth day of antiparasitic therapy there is usually an exacerbation of neurological symptoms, attributed to local inflammation caused by the death of the larvae. For this reason, albendazole or praziquantel are generally given simultan- eously with steroids in order to control the oedema and intracranial hypertension. Serum levels of praziquantel decrease when steroids are administered simultaneously, an effect that does not occur with albendazole. However, there is no evidence that cysticidal efficacy is decreased. Serum levels of praziquantel or albendazole might be lowered by simultaneous antiepileptic drug (phenytoin or carba- mazepine) administration. Some forms of neurocysticercosis should not be treated with antiparasitic agents. In patients with severe cysticercotic encephal- itis, these drugs may result in worsening cerebral oedema and fatal herniation. In this case, the mainstay of therapy is high doses of corticosteroids or mannitol to decrease the inflammatory response. In patients with both hydrocephalus and parenchymal brain cysts, antiparasitic drugs should be started only after placement of a ven- tricular shunt in case the intracranial pressure increases as a result of drug therapy. Antiparasitic drugs must be used with caution in patients with giant subarachnoid cysticerci. In such patients, con- comitant steroid administration is mandatory to avoid cerebral in- farction. Albendazole can successfully destroy ventricular cysts, but the surrounding inflammatory reaction can cause acute hydroceph- alus if the cysts are located within the fourth ventricle or near the foramina of Monro and Luschka. Surgery is limited to ventriculoperitoneal shunts to relieve ob- structive hydrocephalus, and excision of single cysts (in the fourth ventricle or giant intraparenchymal cysts). However, shunts fre- quently malfunction. The protracted course in these patients and their high mortality rates (up to 50% in 2 years) is directly related to the number of surgical interventions required to change the shunts. Recently, neuroventriculoscopy has been employed as a less invasive option for resection of ventricular cysticerci.

8.10.3 Cysticercosis 1539 Prognosis Parenchymal cysticercosis has a good prognosis. Appropriately managed, seizures usually subside in time without sequelae. In contrast, extraparenchymal cysticercosis, and especially ra- cemose cysticercosis, has a poor prognosis, responding poorly to antiparasitic therapy, and leading to progressively deteriorating disease and death. Multiple courses of antiparasitic treatment and careful, prolonged follow-up are crucial in this type of patients. Prevention and control Cysticercosis would not exist if pigs had no access to human faeces. However, this approach is hampered in endemic zones by the lack of sanitary facilities and veterinary inspection, and more importantly, because farmers tend to raise pigs under free-range conditions in order to reduce the cost of feeding them. Intervention programmes have concentrated on mass chemotherapy to eliminate human taeniasis, but their results have not been sustained. New tools for control are oxfendazole, an effective and cheap single-dose therapy for porcine cysticercosis, and the candidate porcine vaccines under trial by several groups. TSOL18, an oncosphere-based vaccine de- veloped in Australia, may provide over 99% protection. A recent wide-scale elimination program in Peru has provided initial evi- dence on the feasibility of focal elimination. Monitoring the effect of an intervention requires suitable indica- tors. Human seroprevalence does not reflect changes in infection patterns because antibodies persist for years, even after successful treatment. Similarly, symptoms can appear years after infection. Since the prevalences of human and porcine infection are strongly correlated, pigs are likely a better indicator for recent transmission. Possible future developments Although most cysts disappear after antiparasitic treatment, the antiparasitic efficacy of currently available regimes is incomplete. Data are missing on whether new drugs, combination therapy, or different schemes of albendazole or praziquantel can improve this efficacy. Schemes and doses of antiparasitic and steroid therapy need to be assessed in controlled trials targeted to specific types of neurocysticercosis. Systematic long-term evaluation is needed to determine the impact of parasite destruction in seizure relapses in the short and long term, particularly considering the association between neurocysticercosis and mesial temporal sclerosis. The ef- ficacy and costs of comprehensive human–porcine eradication pro- grammes must be assessed. FURTHER READING Del Brutto OH, et al. (2001). Proposed diagnostic criteria for neurocysticercosis. Neurology, 57, 177–83. Del Brutto OH, et al. (2006). Albendazole and praziquantel therapy for neurocysticercosis: a meta-analysis of randomized trials. Ann Intern Med, 145, 43–51. Evans C, et al. (1997). Controversies in the management of cysticercosis. Emerg Infect Dis, 3, 403–5. Garcia HH, et al. (2003). Taenia solium cysticercosis. Lancet, 362, 547–56. Garcia HH, et al. (2004). A trial of anti-parasitic treatment to reduce the rate of seizures due to cerebral cysticercosis. N Engl J Med, 350, 249–58. Garcia HH, et al. (2014). Clinical symptoms, diagnosis, and treatment of neurocysticercosis. Lancet Neurol, 13, 1202–15. Garcia HH, et al. (2014). Efficacy of combined antiparasitic therapy with praziquantel and albendazole for neurocysticercosis: a double-blind, randomised controlled trial. Lancet Infect Dis, 14, 687–95. Gonzalez AE, et al. (1997). Treatment of porcine cysticercosis with oxfendazole: a dose–response trial. Vet Record, 141, 420–2. Gonzalez AE, et al. (2005). Vaccination of pigs to control human neurocysticercosis. Am J Trop Med Hyg, 72, 837–9. Montano SM, et al. (2005). Neurocysticercosis: association between seizures, serology and brain CT in rural Peru. Neurology, 65, 229–33. Nash TE, et al. (2006). Treatment of neurocysticercosis—current status and future research needs. Neurology, 67, 1120–7. Otte WM, et al. (2013). Drug therapy for solitary cysticercus granu- loma: a systematic review and meta-analysis. Neurology, 80, 152–62. Salim L, et al. (2009). Seroepidemiologic survey of cysticercosis- taeniasis in four central highland districts of Papua, Indonesia. Am J Trop Med Hyg, 80, 384–8. Wender JD, et al. (2011). Intraocular cysticercosis: case series and comprehensive review of the literature. Ocul Immunol Inflamm, 19, 240–5.

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