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Medical Therapy for Cysticercosis: Indications, Risk, and Benefits.

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Neurocysticercosis is a pleomorphic disease that causes serveral neurological syndromes and pathological lesions. Therefore, a unique therapeutic shceme can not be useful in every patient. A proper characterizcion of the disease in terms of viability of cysts, degree of the host's immune response to the parasites, and location of the lesions is of major importance for a rational therapy. Therapy include a combination of symptomatic drugs, cysticidal drugs, surgical resection of lesions, ana placement of ventricular shunts.

The firts cysticidal drug used in humans was metrifonate. However, the presence of severe cholinergic side efects limited its use. Thereafter, praziquantel, a drug that was originally used in veterinary medicine, proved to be effective in human cysticercoiss. Preliminary suties only included anecdotal case reports with differn forms of the disease and the efficacy of praziquantel was difficult to evaluate. A new age of knowledge on medical treatment for NCC began when the first controlled trials of praziquantel therapy for patients with parenchymal brain cysts were published. In those trials, it was demostrated that viable systicerci may live unchanged for several years within the brain parenchyma, and that a course with praziquantel at daily doses of 50 mg per Kg of body weight for 15 days destroys more than 60% of these cysts in less than three months. This regimen of praziquantel was arbitrarily chosen. Since then, recomeended dosages of praziquantel have ranged from 10 to 100 mg/kg for periods of 3 to 21 days. In almost all studies, praziquantel has been administered every eight hours. Since plasma levels of praziquantel decline one to three hours after administration, it seems that the cysticidal effect has been reached by exposing the parasites to several intermittent peaks of the drug. On this basis, it was recently hypothesized that if parenchymal brain cysticerci were exposed to a high concentrationo fo praziquantel maintained for up to six hours by giving three individual doses of 25 to 30 mg/kg at two-hours intervals this might be sufficient to destroy the parasites. Preliminary results were encouraging since the percentage of cyst disappearance ofn neuroimaging studies was similar to that observed in patients reveiving larger courses of the drug at conventional doses. Currently, more than 30 patients with parenchymal brain cysts have been treated with such regimen of praziuantel and the results are highly promising, not only in terms of effectiveness but also with regard to improved compliance and significant reduction of the cost of treatment.

Some imidazole have also been used to treat parenchymal brain cysticerci. Flubendazole was administered to 13 patients with neurocysticercosis at daily doses of 40 mg/kg for 10 days, and showed promising results. However, problems related to poor intestinal absorption of the drug have limited its use. Thereafter, albendazole appeared as an excellent alternative to praziquantel. Albendazole was first tested in seven patients with parenchymal brain cysts in whom an 86% reduction in the number of lesion was documented after a course with this drug. Albendazole was initially administered at daily doses of 15 mg per kg of body weight for 30 days; nevertheless, further studies showed that at similar doses, the length of therapy could be shortened from 30 to 8 days without lessening the efficacy of the drug. Several controlled trials comparing the efficacy of albendazole vs praziquantel hace been published. Overall, these trial have that praziquantel destroys 60% to 70% of parenchymal brain cysts while albendazole destroys 75% to 90% of such lesions. The advantage of albendazole over praziquantel is no limited to its better percentage of parenchymal cysts' destruction, but to its better penetrance in the subarachnoid space which also allows destruction of meningeal cysticerci.

The efficacy of cysticidal drugs was initally evaluated by counting the percentage of cysts ' destruction on CT and little emphasis was placed on the clinical status of patients before and after the trial. More recent studies have also focused on the clinical outcome of the patients before and after the trial with such drugs. Such studies have noted that the control of seizures in patients with parenchymal NCC is better after a course with cysticidal drugs than when the disease is left untreated; in one of these series, 83% of patients who received either albendazole or praziquantel

had adequate control of seizures, as compared with 27% of those who did not receive such drugs.Moreove, in about 60% to 80% of parenchymal brain cyst destroyed by cysticidal drugs, no residual calcificacion is left. It has been argued that cysticidal drug therapy leads to more produnt cerebral cicatrix within the brain parenchyma than might occur if the cysts are left untreaded; however, this assumption has not been confiermed in pathological studies and should not create injustified concern.

The optimal lengt of antiepileptic drug therapy in patients with epilepsy due to parenchymal brain calcifications remains undefined. Two recent studies have shown that the risk of seizuere recurrence after antiepileptic drug withdrawal is high, even in patients who had been seizure-free for two years on therapy. This high risk of seizure recurrence was found to be independent of the patien's age, seizures type (generalized, partial complex partial) or the number of seizures before diagnosis. CT studies performed immediately after seizure relapse have shown in some patients, focal edema and abnormal contrast uptake around previously inert calcifications. These changes are related to a breadowsn of the blood-brain barrier surrounding a epileptogenic focus, suggestiong that parenchymal brain calcifications may actually represent permanent epileptogenci foci susceptible to reactivation when the inhibitory influence of antiepileptic drugs is withdrawn. While epilepsy duer to parenchymal brain calcifications is easily controlled with antiepileptic drugs, a seizuere-free sate without medication appears to be difficult to archive in most of these patients.

Another benefical effect of cysticidal drugs is the clinical improvement in focal neurological deficits that most patients experience after therapy. Patiens with profund hemiplegia or marked diminution of visual acuity have succesfully recoverd after albendazole treatment because the pressure effects exerted byte cuysts against eloquent cerebral areas has been resolved as the result of therapy.

Cysticidal drugs are also of value as a diagnostic tool in patients in whom the diagnosis of neurocysticercosis in doubt even after reviewing the medical record and the CT findings. Such cases usually correspond to patients with partial seizures due to a singel enhacing CT lesion within the brain parenchyma in whom several entities, including intracranial tuberculomas, brain tumores, and mycotic granulomas, may be included in the diferential diagnosis. In the past, these patients were only treated with symptomatic drugs and were re-evaulated with CT after 8 to 12 weeks, and aggressive investigation was indicated only in the lesion persisted after that time. It has recently been demonstrated that routine administration of albendaole in this setting permits early detection of those patients who need an aggressive diagnostic approach (obviating the hazards of prolonged delays) by hastening the resolution of cysticerci-related sngle enhancing CT lesions.

Some forms of neurocysticercosis should not be treated with cysticidal drugs. Both albendazole and praziquantel may exacerbate the syndrome of intracranial hypertension observed in patients with cysticercotic encephalistis, and are contraindicated during the acute phase of the disease. In patients with mixed forms of neurocysticercosis including hydrocephalus and parenchymal brain cysts, cysticidal drugs can be used only after a ventricular shunt has been placed to avoiid further increases in intracranial pressure as the result of drug therapy. Finally, patients wiht