


Primary Hydatidosis of the Peripheral Muscles: Treatment with Albendazole

Infection due to Echinococcus species mainly affects the liver and lungs, and, to a lesser extent, the brain, bones, and other organs [1]. There are few reports of primary muscular hydatidosis [2], perhaps partly because the parasites have to cross pulmonary and hepatic barriers to reach the muscles [1] and because the presence of lactic acid in the muscles renders them an inhospitable environment. We describe a patient with primary hydatidosis of a muscle in the forearm.

A 20-year-old woman was referred to our unit on 28 March 1994 with suspected soft-tissue sarcoma. Over the previous 3 months she had noticed a swelling on the right forearm and difficulty in extending the hand and wrist. Physical examination showed a relatively immobile subcutaneous mass that was ~10 × 8 cm in diameter and adhered to the deeper layers of the forearm; the overlying skin was inflamed, and the wrist was flexed and could not be extended. Plain films of the affected area yielded no information. An MRI revealed a mass (figure 1) suggestive of soft-tissue sarcoma. Routine blood tests yielded the following results: erythrocyte sedimentation rate, 35 mm/h; leukocyte count, 5.25 × 10⁹/L; eosinophil count, 0.15 × 10⁶/L. Other test results were normal.

A biopsy was performed, and ~50 mL of pus was drained together with a number of small white membranes that resembled grape skins. Following histological and bacteriologic analysis of the specimen (during which Staphylococcus aureus was isolated), the condition was diagnosed as a hydatid cyst. A hemagglutination inhibition test revealed that the titer of antibodies to Echinococcus was 1/160; findings on plain films of the chest and abdomen were normal, as were findings on an abdominal CT scan. The patient began receiving therapy with oral albendazole at a dosage of 10 mg/(kg·d) (200 mg every 8 hours) in preparation for surgical removal of the cyst.

Examination of the patient after 1 month had elapsed revealed that the mass had decreased considerably in size and was barely palpable; the patient was able to flex her wrist and fingers almost fully. She refused the operation and requested to be allowed to continue receiving albendazole. After she had received a 4-month course of therapy, findings of the physical examination were completely normal, no tumorous mass was palpable, and the patient had almost complete mobility of her hand and wrist. Treatment with albendazole was stopped. One year later, the patient had no symptoms at all, and findings on another CT scan with contrast

Figure 1. MRI of the forearm of a patient with primary muscular hydatidosis; axial projection shows a tumorous mass within the anterior musculature and internal cyst-like lesions.
were normal. To date, the patient has had no symptoms and has normal function of her hand and wrist.

Muscular involvement in echinococcus infection is uncommon [1]; involvement of muscle alone is even rarer [2]. Our patient’s case fulfilled the criteria for muscular hydatidosis, with impairment of muscle function due to compression; involvement of the liver and/or lungs was ruled out. Although surgery remains the treatment of choice for hydatid cysts, cyclic and prolonged treatment with albendazole (10 mg/[kg • d]) is recommended. Albendazole therapy should be instituted at least 1 month before surgery is performed [3, 4], since it leads to a decrease in the volume and viability of larvae, and subsequent recurrence of the condition is less frequent [5]. However, to our knowledge, the world literature contains no reports of a complete cure of primary muscular hydatidosis solely by means of albendazole therapy.

Our patient exhibited no local or systemic signs of recurrence of the infection during 1 year of follow-up; she is thus considered cured. Medical treatment with albendazole before surgical removal of a hydatid cyst should be compulsory in cases of primary muscular

Variability of Plasma Fluconazole Levels in Patients with Hematologic Malignancy

Only scant information relating to plasma fluconazole levels is available. However, reasonably constant steady-state values have been reported to be reached by 7 days of therapy [1]. We obtained blood specimens between day 18 and 49 ("day 28") for 26 leukemia patients who were undergoing chemotherapy, precisely 2 hours (peak level) after the patients received 200 mg of daily fluconazole prophylaxis. Plasma levels of fluconazole were measured after extraction of NAOH, ethyl acetate, and HCl, and the samples were analyzed by HPLC/ultraviolet light detection [2]. Creatinine clearance (CC) was calculated within ±1 day of the plasma fluconazole levels as follows:

\[(140 - \text{age}) \times \text{ideal body weight (kg)} \times 0.85 \text{ (female)} / \text{serum creatinine (mg/dL)} \times 72\]

Student’s t test and Wilcoxon’s nonparametric tests were used to evaluate all intergroup comparisons. Conservative two tailed P values were used. Paired comparisons were made with use of Student’s t test and Pearson’s correlation coefficient. Between-group comparisons of means were performed with use of Wilcoxon’s nonparametric test. Steady-state mean values from published data were calculated assuming a linear dose-response relationship.

The mean (±SD) level of plasma fluconazole on day 28 was 9.32 ±3.74 mg/L. This level is in contrast with previously published data were calculated assuming a linear dose-response relationship.

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Student’s t test and Pearson’s correlation coefficient. Between-

corresponding CCs (mean ± SD) in these 22 patients whose CC was normal on day 28 (>77 mL/min) had a mean (±SD) peak plasma fluconazole level of 8.36 ±3.14, while four patients whose CC was low (<77 mL/min) had a significantly higher level of fluconazole (mean ± SD, 14.60 ±1.87) (P < .01). However, 8 of the 22 patients whose CC was normal had plasma fluconazole levels of at least 1 SD above (four patients) or below (four patients) the mean (table 1). There was no significant correlation between plasma levels of fluconazole and CC in the 22 patients whose CC was normal.

Blood was available on day 7 from 13 of 26 patients. The mean (±SD) plasma level of fluconazole in these 13 patients rose from 7.10 ±2.27 on day 7 to 10.69 ± 3.21 on day 28 (P < .02), despite either a persistently normal CC in 10 of 13 patients and a mildly abnormal CC in 3 patients. The mean plasma level of fluconazole also rose in patients whose renal function was entirely normal, but there were no significant correlations between levels of plasma fluconazole on day 28 and CC.

No significant association was found between plasma levels of fluconazole and the maximum alanine aminotransferase or bilirubin levels or concomitant medications. Four patients developed either oral, urinary, or groin infection (three developed infection due to Candida glabrata; one developed infection due to Candida lusitaneae). The mean (±SD) peak plasma level of fluconazole on day 28 for the patients with candidal infection was 7.57 ±4.26 compared with 9.64 ±3.65 for 22 patients without candidal infection (P = NS). The corresponding CCs (mean ± SD) in these two groups were 163.1 ±76.23 and 116.8 ±44.2, respectively (P = NS).

Our data indicate that there is considerable variation in peak plasma levels of fluconazole in our patients who were compliant and who had normal renal function. This finding contrasts sharply with previous published data on patients who have undergone bone

References