

(1) In their discussion they stated, 'although it has been reported (by Casteels-Van Daele *et al.*) that children can have these side effects with the dose recommended by the manufacturers their patients received a dose varying between 0.56 and 1.15 mg/kg per day'. This statement was made in 1970 when we first reported on dystonic reactions caused by metoclopramide and I want to stress that at that time that was right.² Indeed, then manufacturers recommended a dose of 0.5 to 1 mg/kg per day, but later they lowered it to 0.5 mg/kg per day. However, even on this dose dystonic reactions can occur as shown by Cases 7 and 9 in our later series of patients who received a dose of 0.52 and 0.48 mg/kg per day.³ We therefore have to accept that a normal dose of metoclopramide can induce acute dystonic reactions. The patient should be warned in order to avoid unexplained and distressing situations later.

(2) It is right that 'such reactions can be effectively stopped by administration of benztropine 1 mg intravenously'. We too have used benztropine with spectacular results, all symptoms stopping almost immediately, but one of our patients developed an atropine-like intoxication characterised by a psychotic state with extreme agitation, loquaciousness, hallucinations, motor restlessness, and bradycardia, starting 3 hours after intravenous injection and lasting for 17 hours. Children seem to be sensitive to atropine-derivatives and we suggest that 1 mg intravenously (irrespective of age) may be too high a dose. In the younger ones (1 to 5 years old) a dose of 0.5 mg seems to be safer. In our experience a small dose can be given by slowly injecting intravenously and stopping as soon as the dystonic reactions disappear. If the dystonic reactions are not too severe, by far the safest therapy is to stop the administration of metoclopramide immediately.

References

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Visceral larva migrans and *Trichuris vulpis*

Sir,

Sakano *et al.*¹ reported 2 cases of visceral larva migrans (VLM) which they concluded were due to infection with *Trichuris vulpis*. They based this conclusion on the results of immunoelectrophoretic studies in which precipitation lines were found between the patients' serum and *T. vulpis* antigen. Precipitation was also found against

Toxocara canis but it was concluded that this was non-specific according to results of immunoelectrophoresis.² Treatment with thiabendazole was considered effective on the basis of a decrease in the number of arcs of precipitate and in the eosinophil count.

We are reluctant to accept *T. vulpis* as the cause of the VLM in these cases for the following reasons:

- (1) There is no evidence that *T. vulpis* (a common parasite of the caecum and large-intestine of dogs) follows more than a transient migration, as an immature worm, into the glands of the gut wall of its usual host before emerging into the gut lumen as an adult.
- (2) Immunological methods of diagnosis for helminth infections are not generally precise, even with so-called 'purified' specific antigens, and can seldom give more than an indication of relationship to a nematode and not true identity.
- (3) Even if the results were to prove that the patients had an antibody response specific for *T. vulpis*, this would not be evidence of visceral migration. Dogs infected by the parasite show a rise and fall in antibody titres in response to local invasion of the bowel and there is no reason to believe this would not happen in man. The immunology of *T. vulpis* has been reviewed by Beer.³
- (4) Thiabendazole is not a particularly effective anti-helminthic for treatment of adult *T. vulpis* in dogs. In VLM due to *T. canis* symptoms subside spontaneously, including the eosinophilia (though generally after the clinical symptoms), irrespective of treatment. We feel that in these cases the response ascribed to thiabendazole was likely to be coincidental.
- (5) We think that the positive precipitation reaction to *T. canis* antigen (admittedly non-specific) is a more likely cause of the symptoms described, particularly since *Toxocara* sp. is a known cause of VLM.

We agree that the diagnosis of VLM can be difficult and final proof can be established only by demonstrating the larvae in the tissues. Nevertheless, we are unhappy to add yet another parasite as a cause of this syndrome without clearer evidence.

References

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