A 56-year-old man presented with a recurrent cutaneous eruption of the trunk, associated with itching, which had lasted for several months. Antihistamines, such as cetirizine, loratadine, and fexofenadine, and topical steroids had been used with only temporary relief. Recently, the cutaneous manifestations had become continuous and very tedious and had not responded to local or systemic treatments. The patient was a doctor of medicine, was married, and had not been taking any medications. He had travelled for scientific purposes to different areas of Italy and The Netherlands in the last 12 months. He had no animals at home and no history of fever, sweats, arthralgia, cough, dyspnea, or allergic diseases. A history of gastritis and mild hyperglycemia was present.

Skin examination showed large, erythematous, slightly edematous macules and pruritic, erythematous papules densely aggregated over scattered areas of the trunk, especially on both flanks, the abdomen, and the back (Fig. 1). The patient appeared well and no lymphadenopathy was found.

Laboratory investigations showed a hematocrit of 40.2% and a white cell count of 9000/mm$^3$, with 54% neutrophils, 28% lymphocytes, 4% monocytes, and 14% eosinophils. The platelet count was 233,000/mm$^3$. The levels of urea nitrogen, creatinine, conjugated and total bilirubin, aspartate aminotransferase, alanine aminotransferase, and aldolase were normal.

Serologic tests for *Toxoplasma*- and *Toxocara*-specific immunoglobulin G (IgG) were negative. The IgE level was 226 IU/mL (normal range, 0–120 IU/mL). Prick tests for house dust mite, pollen, animal dander, and food proved to be negative. Patch tests with the European Contact Dermatitis Group (ECDRG) standard series and textile color and finish series were negative.

Histologic examination of the dermis showed a slight perivascular superficial inflammatory infiltrate composed of lymphocytes, neutrophils, and eosinophils, in association with dermal fibrosis. Immunohistochemistry (anti-CD4 Dako, anti-CD8 Dako, anti-CD79 Dako, anti-CD3 Dako) showed lymphocytes with a CD4+/CD8+ T phenotype. Direct immunofluorescence gave a negative result.

Microscopic examination of the peripheral blood showed no microfilariae, and no ova or parasites were detected in the first stool specimen. The second stool specimen revealed the presence of ova of *Hymenolepis nana*.

The patient was treated with 2 × 100 mg mebendazole (Vermox®) given orally for 3 days. This treatment aggravated the skin eruption and the pruritus became more intense (Fig. 2).

Two weeks later, the patient presented with skin manifestations with similar characteristics to the first eruption, but less severe. Pruritus was still present but was only mild. New parasitologic investigations still showed a few ova of *Hymenolepis nana* in one of two stool samples. The patient was treated with praziquantel in a single oral dose of 40 mg/kg which was repeated after 7 days. The cutaneous eruption gradually disappeared and the itching decreased and cleared more slowly. The eosinophils in the blood and the total serum IgE level dropped to normal limits, and parasitologic investigations were repeatedly negative. The patient has been symptom free for 8 months.

**Discussion**

Cutaneous symptoms consisting of urticaria, facial edema, flush, and generalized pruritus are well-known dermatologic signs of helminth infestations, but are not commonly observed with cestode parasitoses. We report the case of a patient who developed a generalized macular and papular eruption during a dwarf tapeworm infection (*Hymenolepis nana*).
Hymenolepis nana is a cosmopolitan dwarf tapeworm which is endemic in tropical and subtropical countries. Its eggs are usually spherical, 30–47 µm in diameter, with a thin outer membranous shell and a thicker internal embryophore containing the hexacanth embryo.

The life cycle of Hymenolepis nana involves humans or rodents as the definitive host and arthropods (beetles, fleas) as the intermediate host. Humans and rodents are infected when they ingest cysticercoid-infected arthropods or embryonated eggs from contaminated food, water, or hands. When the eggs are ingested, the oncospheres contained in the eggs are released. They penetrate the intestinal villus developing into cysticercoid larvae.

Unlike virtually all other species of tapeworm, however, no intermediate host is required in the life cycle of Hymenolepis nana. Autoinfection can occur if the eggs remain in the intestine of the definitive host. In this case, the eggs release the exacanth embryo, which penetrates the intestinal villus, continuing the infective cycle without passage through the external environment. The life span of adult worms is 4–6 weeks, but internal autoinfection allows the infection to persist for years. Thus, this species can be a particular problem in humans in areas of high population density, close contact, and where sanitary conditions are poor. It is probably the most abundant tapeworm in the world commonly found in young children.

This parasitosis can be asymptomatic or characterized by enteritis, abdominal pain, diarrhea, anorexia, headache, dizziness, weight loss, meteorism, and flatulence. Skin manifestations are usually absent. The diagnosis is based on the identification of eggs or worms in the feces.

Our patient presented with clinical manifestations possibly arising from a helminth infestation, with urticarial reaction and generalized itch; in addition, papular lesions were observed. The clinical manifestations, which had been present for several months, worsened after the first unsuccessful treatment with mebendazole, suggesting a Jarisch–Herxheimer phenomenon. The lesions recurred simultaneously with the persistence of the ova of Hymenolepis nana in the stools, and disappeared with the cure of the helminth infestation with praziquantel. Laboratory investigations showed an increase in serum immunoglobulin E (IgE) and eosinophils in the peripheral blood in the absence of a personal or family history of atopy or allergy. These returned to within normal limits after skin and microbiologic cure.
In the differential diagnosis, we considered other allergic dermatoses, including contact dermatitis by clothes and other skin disorders commonly associated with eosinophilia, such as urticaria, bullous pemphigoid, and eosinophilic cellulitis. Abnormalities of serum IgE and eosinophils can occur in a wide variety of conditions, but they are important hallmarks of intestinal parasitic infestations by nematodes (roundworms), cestodes (tapeworms), and trematodes, due to the stimulation of IgE synthesis by intestinal parasite proteinases. Although most infected residents of endemic areas are asymptomatic, a more pronounced eosinophilia and the occurrence of symptoms, such as pruritus and urticaria, are quite common in individuals who do not live in regions of the world in which parasites are endemic. In particular, although adult worms are probably only weakly immunogenic, the eggs of *Hymenolepis nana* may elicit a strong immune response.

Papular eruptions have rarely been observed during dwarf tapeworm infestations. Due to the particular life cycle of *Hymenolepis nana*, which can generate an autoinfection, the resulting persistent or permanent antigen stimulation could be the cause of the papular eruption, although we were unable to detect helminth antigen in the skin lesions.

**References**