Ancylostoma caninum is the principal cause of canine hookworm disease in most tropical and subtropical areas of the world. The larvae cause damage to the host at the point of entry through the skin leaving a wound vulnerable to secondary infections. As the larvae migrate through the skin an inflammatory response, dermatitis, is often stimulated which can be exacerbated in hosts which give hypersensitive responses. Further damage is caused when the larvae leave the circulation and enter the lung with the amount of damage dependent on the extent of the infection; pneumonia and coughing are common consequences [1,2]. Dermatitis is an inflammation of the skin. Contact dermatitis is a localized rash or irritation of the skin caused by contact with a substance that is foreign to the body. It caused by an irritant may also cause burning or pain as well as itching. Irritant dermatitis often shows as dry, red, and rough skin. Allergic contact dermatitis occurs when the skin develops an allergic reaction after being exposed to a foreign substance. This causes the body to release inflammatory chemicals that can make the skin feel itchy and irritated [3,4]. We evaluated a case of contact dermatitis attributed to nickel allergy but caused by Ancylostoma caninum infection.

Case presentation

The patient was a 44-year-old man with a history of hypertension presented to the outpatient clinic with complaints of dry, scaly, flaky skin, skin redness and extreme itching affecting his hands. The first consultation took place in 2008, during which patch tests initially performed gave a positive result only to nickel. The patient avoided any possible contact with nickel, but dermatitis recurred regularly at intervals of six months. There were no skin lesions, or noteworthy comorbidities. His family history was positive for asthma, but there was no personal or familiar history of contact dermatitis. The basic investigations such as complete blood count, blood sugar, renal function tests, liver function tests, and urine routine were all within normal limits. Thyroid function was also normal. Three years later the disease affected his right foot and was treated with topical steroids, but in the following years also edema of the foot with impaired walking occurred. Patch testing confirmed the positive result for nickel sulfate. The patients also complained about recurrent headache and asthenia especially in the morning. By routine blood tests, only peripheral eosinophilia and total IgE levels were abnormal. The faeces were sampled and the ovular, thin-shelled eggs of A. caninum were found.
demonstrated. The patient was evaluated for immunological tests including ANA, ENA, and anti-parasitic antibodies, yielding a positive result by Western blotting and ELISA for *Ancylostoma caninum*. Mebendazole was administered in the dose of 200 mg per day for 3 days. Immediate efficacy on feet dermatitis and edema was achieved. Other 3 courses of mebendazole treatment were performed, with complete regression of dermatitis. The headache and asthenia were disappeared and the peripheral eosinophilia turned to normal value. ELISA test and Western blotting turned negative for *Ancylostoma* antibodies. Periodic examination at the end of 2nd, 4th, and 6th month showed complete treatment of the disease.

**Discussion**

*Ancylostoma caninum* is a species of nematode which principally infects the small intestine of dogs. The result of *A. caninum* infection ranges from asymptomatic cases to death of the dog; better nourishment, increasing age, prior *A. caninum* exposure or vaccination are all linked to improved survival. Other hosts include carnivores such as wolves, foxes and cats with a small number of cases having been reported in humans. The eggs of these parasites are shed in the feces of infected animals and can end up in the environment, contaminating the ground where the animal defecated. People become infected when the zoonotic hookworm larvae penetrate unprotected skin, especially when walking barefoot or sitting on contaminated soil or sand. This can result in a disease called cutaneous larva migrans (CLM), when the larvae migrate through the skin and cause inflammation. An acute normocytic, normochromic anemia followed by hypochromic, microcytic anemia in young puppies is the characteristic, and often fatal, clinical manifestation of *A. caninum* infection. Surviving puppies develop some immunity and show less severe clinical signs. Diarrhea with dark, tarry feces accompanies severe infections. Anorexia, emaciation, and weakness develop in chronic disease [5,6]. Contact dermatitis is a type of eczema that causes the skin to become red, blistered, dry, scaly and cracked. There are two types of contact dermatitis: allergic and irritant. They often can appear to be visually identical: allergic contact dermatitis often results from an immune response to a small, structurally simple, nonprotein molecule and irritant contact dermatitis results from coming in contact with a substance that directly damaging and irritating to the skin. No allergy is required, and it will occur on the first exposure. Symptoms vary depending on the cause and whether the dermatitis is due to an allergic reaction or an irritant. The same person may also have different symptoms over time. Allergy testing with skin patches (called patch testing) may determine what is causing the reaction [7,8]. Patch testing is used for certain patients who have long-term or repeated contact dermatitis. It requires three office visits and must be done by a health care provider with the skill to interpret the results correctly. Substances that commonly cause contact dermatitis include plant sap, metals, cleaning solutions, cosmetic additives, perfumes, industrial chemicals, and latex rubber additives [9,10]. Our study presented the *Ancylostoma* infection as apparent contact dermatitis caused by nickel and suggests bearing in mind, in cases of contact dermatitis not responding to avoidance of the responsible hapten and to medical treatment, the possible causative role of *Ancylostoma*.

**References**


Received 6 April 2015
Accepted 15 May 2015