physical exercise. The exact pathogenesis of the association of UV with malignancy is unclear. It is thought that tumour-associated immune complexes may give rise to complement fixation in the vessel wall and subsequent development of an inflammatory process [6]. In exercise-induced UV it has been shown that mast cells are the first to be involved, with the activation and subsequent release of proinflammatory mediators, which precede the influx of eosinophils with their release of granule proteins and the influx of neutrophils with their release of proteolytic enzymes [8]. We hypothesize that deposits of immune complexes and complement fixation in the vessel wall sometimes need an additional event in order to elicit the initial influx of neutrophils, which are the final effector cells responsible for the vascular damage.

The patient gave informed consent to the investigations that led to the diagnosis reported here, and written permission was given for this case to be reported.

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Early-onset polyarthritis as presenting feature of intestinal infection with Strongyloides stercoralis

SIR, In a patient with symmetrical polyarthritis the diagnosis of rheumatoid arthritis (RA) is advocated, especially if no signs of (recent) infection are present. It is considered good clinical practice nowadays to start early treatment with anti-rheumatic drugs, including corticosteroids or tumour necrosis factor-α (TNF-α)- blocking agents. To prevent a life-threatening outcome, however, some rare infections have to be excluded. We describe a patient who presented with recent-onset symmetrical polyarthritis and infection with Strongyloides stercoralis.

A 35-yr-old male was referred to our hospital because of progressive arthralgia over the last 4 months despite the use of non-steroidal anti-inflammatory drugs. His medical history was unremarkable. The complaints started after returning from a journey to Surinam, South America. He denied having any complaints during or after his travel. At admission, polyarthritis of the shoulders, wrists, hand joints, knees, ankles and feet was observed. Further physical examination was unremarkable.

Laboratory investigations revealed an erythrocyte sedimentation rate of 80 mm/h, C-reactive protein level of 121 mg/l and a leucocyte count of 5.8 x 10⁹/l with 20% eosinophilia. Routine urine examination was normal. Rheumatoid factor, antinuclear antibodies and HLA-B27 were absent. Serological tests for human immunodeficiency virus, human parvovirus B19 and Borrelia burgdorferi were negative. X-rays of the chest, hands, feet and sacroiliac joints were normal. Only after repeated examination was the presence of larvae of S. stercoralis detected in concentrated fresh stool. No micro-organisms or larvae could be found in synovial fluid aspirated from the left ankle.

The patient was treated with albendazole 400 mg once daily for 7 days. Because of persistent eosinophilia, a second course was given for another 10 days. Repeated direct stool examination remained free of Strongyloides larvae. In the following months the polyarthritis slowly disappeared and laboratory values normalized. More than 1 yr later the patient is still free of symptoms.

FIG. 1. Rhabditiform larvae of Strongyloides stercoralis in Ridley concentrate of faeces (100×).
Strongyloides stercoralis is a nematode with a worldwide distribution but is more common in (sub)tropical regions. It is estimated that, worldwide, over 100 million people are infected with this parasite [1]. The most common type of reproduction is the host–soil–host cycle. Fully grown female worms lay their eggs in the mucus of the intestinal tract, where the eggs hatch and liberate rhabditiform larvae. These larvae leave the body with the faeces and develop into infective filariform larvae that can penetrate the skin of the host and enter the bloodstream. The larvae reach the digestive tract after migration through the lungs. When infection is limited to the digestive tract, treatment with an anthelmintic agent usually clears the parasites from the intestine.

The so-called autoinfective cycle is an alternative reproductive cycle; rhabditiform larvae transform within the intestinal tract into infective filariform larvae, which may penetrate immediately into the gut wall or peri-anal skin and enter the bloodstream. In normal circumstances this autoinfection does not seem to be a problem. The infection can persist for years or even decades with hardly any symptoms. This balance can be disturbed when the host becomes immunocompromised by either disease (haematological malignancies, malnutrition or AIDS) or treatment with corticosteroids or cytostatic agents. An overwhelming systemic parasitic load can result because of the enormous number of larvae produced in these circumstances [1, 2]. A serious medical condition called hyperinfection syndrome or disseminated Strongyloidiasis may develop rapidly, and is characterized by pneumonitis, respiratory failure, cerebral infiltration and a high mortality rate.

Arthritis is a rare feature of a parasitic infection including Strongyloides [3, 4]. We found nine earlier reports, involving patients with either oligo- or polyarthritis in association with intestinal Strongyloides infection [2, 3, 5–10]. In six of these nine patients, aspiration of synovial fluid was performed, and no larvae were seen. All nine patients recovered fully after anthelmintic treatment only, as did our patient. The mechanism of the development of arthritis is unknown. Although Strongyloides larvae have once been observed in a synovial biopsy specimen, suggesting an infectious type of arthritis [7], most authors consider this to be a reactive arthritis [2–10].

The distribution of the symmetrical polyarthritis in this patient was highly suggestive of RA. In view of the early diagnosis and aggressive treatment of RA today, this patient could have been exposed to immunosuppressive treatment with high-dose corticosteroids or a TNF-α-blocking agent, potentially resulting in disseminated Strongyloides. Repeated examination of stool specimens resulted in the diagnosis of reactive arthritis associated with Strongyloides, and treatment with anthelmintic drugs was therefore the appropriate therapy rather than immunosuppressive therapy.

Two lessons can be learned from this case. First, infection with S. stercoralis can present as (poly)arthritis. Secondly, there is a risk of lethal disseminated infection in patients harbouring Strongyloides in circumstances associated with suppression of the host’s immune system. If immunosuppressive treatment is considered, a high suspicion of Strongyloides infection and repeated examination of faeces is necessary in patients who originate from or have travelled in (sub)tropic areas or in those with eosinophilia.

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