

Co-infection of *Strongyloides stercoralis* and *Balantidium coli* in a patient with pemphigus vulgaris

P L A N Liyanage¹, G M P Sirimanna², S Samarasingha³, R D Silva⁴

Sri Lanka Journal of Dermatology, 2011, 15, 49-50

Introduction

Pemphigus vulgaris is an autoimmune blistering disease in which antibodies are formed against desmoglein-3, a desmosomal cadherin involved in intercellular adhesion in epidermis. Steroids and immunosuppressives are the mainstay of treatment.

Strongyloides stercoralis is a soil transmitted intestinal nematode widely distributed in tropics. *Balantidium coli*, is the largest ciliated protozoan in humans causing intestinal infection. Balantidiasis occurs in temperate and tropical countries. A few cases of strongyloides infection in conjunction with pemphigus vulgaris have been reported in literature¹. But *Strongyloides stercoralis* and *Balantidium coli* co-infection has not been described.

We report a case of co-infection of *Strongyloides stercoralis* and *Balantidium coli* in a patient with pemphigus vulgaris. In addition this is the first case of *Balantidium coli* infection in humans reported in Sri Lanka.

Case report

A 57-year old diabetic female, diagnosed with pemphigus vulgaris in 1999, was successfully treated with repeated cycles of monthly intravenous dexamethasone cyclophosphamide pulse (DCP) therapy and was in remission by 2005. During this period she experienced recurrent molluscum contagiosum and herpes zoster infections. She developed a minor relapse of pemphigus in 2007 and was managed with low dose oral steroids as an out patient.

In May 2011, she was admitted with an exacerbation of pemphigus, involving 60% of body surface area. One cycle of three day intravenous DCP therapy was restarted, and it was followed with daily oral cyclophosphamide and prednisolone.

During the hospital stay she developed an episode of mucoid stools and poor appetite. She had no abdominal pain, fresh blood in the stools,

malaena or perianal pruritus. There was no fever, lymphadenopathy or hepatosplenomegaly. Microscopic examination of the stool sample revealed numerous rhabditiform larvae of *S. stercoralis*. She was managed on oral rehydration solution and was given a course of oral albendazole with which diarrhoea improved. However, repeat stool microscopy revealed persistent *S. stercoralis* larvae and stool culture grew *Balantidium coli*. Though she was started on doxycycline, it had to be withdrawn due to photophobia. Subsequently she was treated with oral ivermectin. Repeat stool examination did not reveal any parasites.

A few days later, she developed wheezy cough and right sided pleuritic chest pain with low grade fever (100°C). Chest radiograph showed right upper lobe consolidation with effusion in interlobar fissure but no migratory or cavitatory lesions. Sputum for acid fast bacilli, blood cultures for both aerobic and anaerobic bacteria were negative. Sputum culture grew coliform bacilli. Repeated full blood count and blood picture revealed neutrophil leucocytosis but no eosinophilia. She responded to intravenous meropenam, gentamicin and metronidazole.

HIV serology was negative and T lymphocyte subset CD₃ (88%), CD₄ (43%), CD₈ (44%) were within normal limits. But CD₁₉ (B cell) was 24/ml (1%), (normal range 100-500/ml). Lymphocyte function tests were not done.

Discussion

Infections are well known to cause increased morbidity and mortality among patients with pemphigus. Patients are susceptible for infections due to the disease itself as well as its treatment. Though staphylococcal infections are described as commonest, severe infections due to unusual organisms can occur.

Strongyloidiasis usually remains asymptomatic or manifest as a mild gastrointestinal infection.

¹Senior Registrar in Dermatology, ²Consultant Dermatologist, National Hospital of Sri Lanka, ³Consultant Parasitologist, Medical Research Institute of Sri Lanka, ⁴Consultant Immunologist, Medical Research Institute of Sri Lanka.

However, disseminated fatal infection is described in the immunosuppressed². Corticosteroid use is associated with a two- to three-fold increase in the risk of being infected by *S. stercoralis*. Corticosteroids induce hyperinfection by acute suppression of eosinophils and lymphocyte activation. The corticosteroids may also accelerate the transformation of rhabditiform larvae into invasive filariform larvae³.

Balantidiasis can manifest as asymptomatic carrier state, chronic symptomatic form and acute bloody diarrhoea. Extraintestinal manifestations are rare, but cavitary lung infection is reported⁴. Our patient had no close contact with pigs although high prevalence of balantidiasis has been seen in human communities that live in close proximity to *B. coli*-infected pigs.

In local experience, pemphigus patients on immunosuppressive medication mostly develop viral diarrhoea that respond to symptomatic treatment. Usually stool full report is negative for parasites.

Finding two kinds of parasites in our patient's stool was highly significant. Though she responded symptomatically to albendazole, parasitological clearance of stool was obtained only with ivermectin.

Immunosuppressed patients who experience any unusual gastro-intestinal or pulmonary symptoms should be suspected of having strongyloidiasis⁵. Since our patient's pulmonary infection responded to intravenous antibiotic therapy we did not perform bronchoalveolar lavage to look for parasitic infections in lung. Interestingly, despite heavy parasitic load, she had no recurrent urticaria or serpiginous eruption "larva currens", which are known cutaneous manifestations due to strongyloidiasis. Rarely widespread petechiae, purpura and vasculitis are also reported in patients with disseminated infections.

Long term steroid use probably compromised her cell mediated immunity as clinically evidenced by multiple viral and fungal infections despite normal T lymphocyte counts. Steroids affect T cell counts as well as function⁶. In addition the effect of cyclophosphamide on B cells was more severe and longer-lasting than the effect on T cells which explains the lymphocyte subset results of our patient.

As a result of prompt treatment she did not develop gram negative sepsis secondary to disseminated strongyloidiasis, or fatal strongyloidiasis. Thus high degree of suspicion, early identification and prompt treatment will reduce the mortality due to strongyloidiasis among immunosuppressed pemphigus patients.

References

1. Sarubbi FA. Hyperinfection with strongyloides during treatment of pemphigus vulgaris. *Arch Dermatol* 1987; **123** (7): 864-5.
2. Reddy IS, Swarnalata G. Fatal disseminated strongyloidiasis in patients on immunosuppressive therapy: report of two cases. *Indian J Dermatol Venereol Leprol* 2005; **71**: 38-40.
3. Keiser PB, Nutman TB. *Strongyloides stercoralis* in the immunocompromised population. *Clin Microbiol Rev* 2004; **17**(1): 208-17.
4. Sharma S, Harding G. Necrotizing lung infection caused by the protozoan *Balantidium coli*. *Can J Infect Dis* 2003; **14**(3): 163-6.
5. Fardet L, Génèreau T, Poirot JL, Guidet B, Kettaneh A, Cabane J. Severe strongyloidiasis in corticosteroid-treated patients: case series and literature review. *J Infect* 2007; **54**(1): 18-27.
6. Ashwell JD, Lu FW, Vacchio MS. Glucocorticoids in T cell development and function. *Annu Rev Immunol* 2000; **18**: 309-45.