Severe strongyloidiasis: a systematic review of case reports

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Abstract
Background: Strongyloidiasis is commonly a clinically unapparent, chronic infection, but immuno suppressed subjects can develop fatal disease. We carried out a review of literature on hyperinfection syndrome (HS) and disseminated strongyloidiasis (DS), in order to describe the most challenging aspects of severe strongyloidiasis.

Methods: We conducted a structured search using PubMed to collect case reports and short case series on HS/DS published from 1991 to 2011. We restricted search to papers in English, Spanish, Italian and French. Case reports were classified as HS/DS according to given definitions.

Results: Records screened were 821, and 311 were excluded through titles and abstract evaluation. Of 510 full-text articles assessed for eligibility, 213 were included in qualitative analysis. As some of them were short case series, eventually the number of cases analyzed was 244. Steroids represented the main trigger predisposing to HS and DS (67% cases): they were mostly administered to treat underlying conditions (e.g. lymphomas, rheumatic diseases). However, sometimes steroids were empirically prescribed to treat signs and symptoms caused by unsuspected/unrecognized strongyloidiasis. Diagnosis was obtained by microscopy examination in 100% cases, while serology was done in a few cases (6.5%). Only in 3/29 cases of solid organ/bone marrow transplantation there is mention of pre-transplant serological screening. Therapeutic regimens were different in terms of drugs selection and combination, administration route and duration. Similar fatality rate was observed between patients with DS (68.5%) and HS (60%).

Conclusions: Proper screening (which must include serology) is mandatory in high-risk patients, for instance candidates to immunosuppressive medications, currently or previously living in endemic countries. In some cases, presumptive treatment might be justified. Ivermectin is the gold standard for treatment, although the optimal dosage is not clearly defined in case of HS/DS.

Keywords: Strongyloidiasis, Strongyloides, Hyperinfection, Disseminated strongyloidiasis, Review

Background
Strongyloidiasis is a neglected condition caused by Strongyloides stercoralis, a soil-transmitted helminth mainly diffused in tropical and subtropical regions, but also present in small areas of low endemicity in temperate climates [1]. Most infected individuals are asymptomatic or may present intermittent symptoms, mostly affecting intestine (from mild abdominal pain, intermittent or persistent diarrhea to more severe conditions that can mimic inflammatory bowel disease), lungs (cough, wheezing and asthma, chronic bronchitis) and skin (pruritus, rash). Systemic symptoms such as weight loss and cachexia may also occur [2]. Immune suppressed subjects tend to develop hyperinfection syndrome (HS) and disseminated strongyloidiasis (DS), that are potentially fatal [3]. Therefore, it is mandatory to diagnose and treat the chronic infection, in order to prevent the life-threatening form. Unfortunately, the index of suspicion of health care providers seems to be low, especially in non-endemic countries [4]. Moreover, there are still gaps in knowledge regarding many aspects of the infection, such as diagnosis and treatment response [2].
Our aim was to systematically review case reports of severe strongyloidiasis, in order to outline the main features of hyperinfection and disseminated strongyloidiasis and the difficulties in their management.

Methods
We carried out a systematic review of case reports/short case series published in PubMed from January 1991 to April 2011. We considered papers available in the following languages: English, Spanish, Italian, French.

The electronic search strategy was as follows: disease (strongyl*, anguillulose) AND severity of cases (disseminat*, hyperinfect*, severe, death, fatal, mortality) OR disease (strongyl*, anguillulose) AND associated conditions (tumor*, cancer, haematolog*, lymphom*, leukem*, leukaem*, neoplas*, malignant*, HTLV*, HIV, AIDS, hypogammaglobulinemia, rheumat*, "biological agents", diabet*, transplant*, COPD, steroid*, glucocorticoid*, Immunocompromise [MeSH], Immunocompromised Host [MeSH]) and limiting the search to humans. Search was done on March 20th 2011.

Definitions used for case - inclusion [5]: - Dissemination: larvae found in any organ, other than the respiratory and the gastrointestinal tracts. Hyperinfection: infection confined to lungs and gastrointestinal tract, but signs/symptoms of severe diseases in relation to elevated number of larvae; in particular, necessity of intensive care, presence of sepsis/meningitis by enteric bacteria, death (without any other clear underlying cause).

Results
Data synthesis
Our search strategy permitted to identify 821 papers, of which 311 were excluded by title and abstract evaluation. Full-text papers were then assessed for eligibility according to the criteria outlined above. Among the 213 papers included, some were small case series, eventually the number of cases analyzed was 244 (Figure 1).

Countries
Reports from highly endemic countries were 65/244 (27%), with India ([6–23]), Argentina ([24–28]), Brazil ([29–39]) and Peru ([40,41,42]) accounting for more than two thirds. Only four cases were reported from the whole of Africa [43–46], three of which in South Africa, a state where adequate diagnostic facilities are available. We collected 83/244 (34%) reports from North America (USA [47–109] and Canada [110–116]), 58/244 (24%) from Europe (Belgium [40,117], France [118–129], Germany [130,131], Greece [132–134], Italy [135–141], the Netherlands [142–145], Spain [146–155], Switzerland [156], UK [157–164]) and five (2%) from Oceania.
(Australia [165–168] and New Zealand [169]). In these areas of low/no endemicity, half of the patients were immigrants (70/146, 48%), while a few subjects were veterans (5/146, 3%) who presumably acquired the infection during military service in an endemic country. Other areas of low endemicity where cases have been reported are in Eastern Asia (21 cases, mostly from Japan ([170–177]) and Taiwan ([178–182])), the Arabian peninsula (nine cases, mostly from Kuwait ([183,184]) and Qatar ([185,186])) and Israel ([187,188]) (three cases). Countries such as Iran ([189]), Turkey ([190]) and Venezuela ([191]) that might be presumed at medium to high prevalence, account for only one case each.

**Triggers for development of HS/DS**

According to the case definitions, 171 cases were classified as hyper infection and 73 cases as dissemination.

A high percentage of patients (67%: 164/244) were under corticosteroids: most of them presented clinical conditions causing immune suppression per-se or due to other related therapies (for instance leukemia, rheumatic conditions, transplant), as it is shown in Table 1. On the other hand, a few patients were taking steroids for eosinophilia and/or a specific symptoms caused by *S. stercoralis* itself (data reported in Table 1 too). A patient even underwent bone marrow transplant because of an unexplained eosinophilia misdiagnosed as “idiopathic hypereosinophilic syndrome” [81]; after receiving steroids and immunosuppressive therapy he developed HS (but only limited autopsy was performed, so we cannot rule out DS) and died.

Transplant is surely an event that poses the *Strongyloides*-infected patient at high risk of developing HS/DS. We collected 28/244 (11.5%) cases of HS/DS in transplant patients, of whom 19 (68%) died. A couple of patients who developed hyper infection also had co infection with CMV [21,107]. All the surviving patients received ivermectin, either as single treatment (1 patient) or in combination with albendazole (7) or thiabendazole (1) [29,54,60,71,90,92,94,142,145,150].

HTLV-1 infection is a well known risk factor (sometimes in association with related haematological malignancies), of which we found 24/244 (10%) reports ([214,158,120,159,215,53,54,143,122,12,111,32,216,77,126,163,114,80,35,164,175,155,116]). Ten of the 24 patients (42%) died. One patient had HTLV-1-HIV co infection [122]; he developed an *E. coli* meningitis but successfully responded to ivermectin, two doses given some days (not specified how many) apart.

We found 38/244 (15%) reports on HIV-positive patients, 26 (68%) of whom died (Figure 2). Seven HIV patients were also receiving steroids for suspected Pneumocystis jiroveci pneumonia [24,26], immune reconstitution inflammatory syndrome [105], misdiagnosis of asthma [58], Wegener granulomatosis [36], toxoplasmosis encephalitis [36], cerebral TB with vasculitis [124]; six of them died.

A few reports/case series describe severe strongyloidiasis in patients with alcoholism [178,217] and malnutrition [27,171]. An apparently immunocompetent patient developed hyper infection and died two days after having started therapy with thiabendazole [149]. Unfortunately autopsy was denied.

**Diagnosis**

Eosinophilia was present in 55/244 cases (22.5%) overall, and only in 12/73 cases (16.4%) of dissemination. In all cases *S. stercoralis* was found at microscopy examination of biological samples. Serology was performed only in

| Condition                                | N (%) | References                                                                 |
|------------------------------------------|-------|-----------------------------------------------------------------------------|
| COPD/asthma/lung fibrosis                | 30 (18.3) | [48,49,52,57-59,68,99,101,118,121,123,128,137,146,153,180-183,185,187,188,192-196] |
| Leukemia/lymphoma                        | 13 (7.9) | [9,17,23,25,37,47,56,98,111,126,162,186]                                    |
| SLE                                      | 9 (5.5)  | [41,64,66,86,151,176,197,198]                                              |
| Rheumatoid arthritis                     | 4 (2.4)  | [83,103,199,200]                                                           |
| IBD                                      | 6 (3.6)  | [59,147,148,164,177,201]                                                  |
| Sarcoidiosis                             | 2 (1.2)  | [65,132]                                                                   |
| Cancer                                   | 8 (4.8)  | [30,54,93,97,112,160,169,202]                                              |
| Organ/bone marrow transplant             | 25 (15.2) | [21,25,29,31,39,48,51,54,60,70,71,74,76,81,87,88,90,92,94,142,145,150,184] |
| Glomerulonephritis/CR1                    | 6 (3.6)  | [16,18,20,129,130,154]                                                    |
| “Idiopathic” eosinophilia                | 3 (1.8)  | [7]                                                                        |
| Multiple myeloma/myelodisplasia          | 6 (3.6)  | [72,185,203-206]                                                           |
| Aspecific symptoms                       | 2 (1.2)  | [85,166]                                                                   |
| Other clinical conditions                | 46 (28)  | [17,22,34,36,54,59,66,84,89,100,102,110,113,124,125,127,133-135,140,155,159,171-174,207-213] |
| HIV-related opportunistic infections/IRIS| 4 (2.4)  | [24,26,36,105]                                                            |
16/244 patients (6.5%) (Table 2). In a couple of organ transplant recipients, an ELISA test was negative pre-transplant, but resulted positive in the deceased donors (test performed retrospectively) [60,145]. In other two cases serology (ELISA) was negative: a HIV-infected person, who had larvae in stool and sputum [165] and a patient with dermatomyositis, under chronic treatment with prednisone and methotrexate, who died from disseminated strongyloidiasis (larvae found at autopsy in skin, lungs, small and large bowel, gall bladder, vessels of meninges and cervical spinal cord) [100].

Diagnosis was obtained post mortem in 29 cases (12%).

Therapy

Therapies given were very different in relation to the drugs used and the length of treatment.

In Table 3 we summarize the drugs used. In the “other drugs” group we found mebendazole [9,17,44,48,131,137,181,218], cambendazole [35,36], levamisole [43,199], pyrantel pamoate [75,108], diethylcarbamazine [14].

Albendazole was used as a single drug even in recent case reports; since 2008 we found patients treated with albendazole only in reports from Pakistan [203], Romania [217], Taiwan [182], Israel [187], Kuwait [184], Argentina [25], Malaysia [207], Greece [133], Thailand [208].

In most cases the administration route was oral, but due to severe clinical conditions of patients, administration via nasogastric tube, subcutaneous injection (veterinary formulation) and retention enema were used, too.

A patient who developed disseminated strongyloidiasis after an empiric steroid treatment for pruritic rash was treated with albendazole [166]. Only one dose could be given, as the patient died. After his death, a review of his clinical records showed that he had been previously diagnosed with strongyloidiasis and treated with a 3-day course of albendazole; although serology persisted positive and eosinophilia was still present 6 and 12 months after treatment, the patient did not receive any further therapy. Another patient who died from Strongyloides hyper infection had never been treated previously, despite a positive serology [101].

Outcome

The recorded deaths were 153/244 (62.7%). A similar fatality rate was observed in patients with dissemination (50/73 = 68.5%) and with hyperinfection (102/171 = 60%).

All 42 of 244 patients who did not receive any therapy died. Excluding patients treated with combination therapy, we observe that 25/34 (73%) patients treated with albendazole died, while deaths among patients treated with ivermectin and thiabendazole were 18/38 (47%) and 28/55 (51%), respectively.

Discussion

Considering that a considerable number of case reports are described in non endemic countries, we assume that fatal cases must be quite frequent in endemic countries, although they are not frequently published in the literature.

The main risk factors identified in this review have been reported previously, in particular steroids are frequently the trigger for developing severe strongyloidiasis. Unfortunately it was not possible to extract from the case reports the cumulative dosage and the duration of the corticosteroids treatment. Although the association with steroids should be well known, there are still papers
reporting cases of patients under steroids who had not been previously screened for strongyloidiasis. Moreover, we found papers reporting severe strongyloidiasis in patients who were previously diagnosed with the infection but had not received a proper treatment. Once more, the lack of familiarity with strongyloidiasis by health care providers is the weak link in the chain; this is also highlighted by the fact that in 12% of cases the diagnosis was made post mortem. Eosinophil count is often normal in severe strongyloidiasis, hence this test has a limited excluding power.

Serology was not frequently performed. In fact, in case of hyperinfection and dissemination the diagnosis is easily made by direct examination of the biological samples. Serology would be most useful in chronic infections, before hyperinfection and/or dissemination occur, while in patients who are already immune suppressed its sensitivity is probably lower.

Limits in our results are due to incomplete information in the case descriptions. Moreover, cases in which autopsy was not performed sometimes couldn’t allow a proper classification. Actually, in the 65 cases we classified as hyper infections, autopsy was not done, hence it is not possible to rule out dissemination. Moreover, we found the same fatality rate for patients with hyper infection and with dissemination, but a misclassification might have played a role. In fact, we think that from a clinical, practical point of view the distinction between hyper infection and dissemination is not essential, because they’re both severe conditions requiring immediate assessment and care.

In general, the best drug to treat strongyloidiasis is ivermectin which is effective and well tolerated. There are still some concerns about the treatment schedule of the chronic infection, and this is even more debated in case of hyper infection/dissemination. In fact there are no specific guidelines and the case reports we collected outline a Babylon of different therapeutic schemes. Subcutaneous ivermectin (veterinary formulation) has been used on an empirical basis, when intestinal absorption is decreased or the patient cannot swallow tablets. On the other hand, albendazole is still used even as a single drug, although it has been proved to be poorly effective. In some cases, this might be due to the scarce availability of ivermectin in many countries.

**Conclusions**

The first step to be done to guarantee an adequate management of infected patients is to avoid a delayed diagnosis. Unfortunately, lack of familiarity with strongyloidiasis by health care providers still seems to be the main cause of delay. A better diffusion of the available information is badly needed, and collaboration among different specialists (oncologists, rheumatologists…) is desirable in order to provide common and adequate protocols for screening and treatment of at-risk patients.

It is mandatory to treat patients in the chronic phase, before HS/DS develop. Patients with possible, previous exposure to the parasite should be screened with serology before corticosteroid treatment, chemotherapy or transplant. Considering the high tolerability of ivermectin, it would be probably worth treating high-risk patients irrespective of the result of the screening test, in order to avoid the potential consequences of a possible false negative result.

Ivermectin is currently the gold standard for treatment of strongyloidiasis, so it is simply no more ethical to use any other drug. Moreover, ivermectin is in the WHO model lists of essential medicines [219], so it should be registered and made available everywhere, particularly in endemic countries.

**Competing interests**

The authors declare that they have no competing interests.

**Authors’ contributions**

DB searched PubMed, analyzed the data and wrote the manuscript. ARM analyzed the data and critically reviewed the manuscript. AA created the review and critically reviewed the manuscript. All authors read and approved the final manuscript.

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**Table 3 Treatments**

| Drug            | Albendazole | Ivermectin | Thiabendazole | Other drugs |
|-----------------|-------------|------------|---------------|-------------|
| Used as single treatment | 34          | 38         | 55            | 6           |
| Deaths among patients treated with single drug | 25/34 (73%) | 18/38 (47%) | 28/55 (51%)  | 5/6 (83%)   |
| Total of patients treated (including combination therapy) | 48          | 79         | 60            | 14          |
42. Zavala J, Sanchez L, Carillo L, Cueva A, Balbin G, Quispe V. [Atypical presentations of strongyloidiasis: a report of 8 cases]. Rev Gastroenterol Peru 1994, 14(1):15–21.

43. Adetoye VA. A case of fatal gastrointestinal strongyloidiasis in an otherwise healthy Nigerian, masquerading as gastric outlet obstruction. Trop Geogr Med 1992, 44(1):260–62.

44. Covadonga YM, Rutherford MC, Bhana RH. Disseminated strongyloidiasis in a diabetic patient. Trop Geogr Med 1993, 45(4):179–180.

45. Daubenton JD, Buys HA, Hartley PS. Disseminated strongyloidiasis in a child with lymphoblastic lymphoma. J Pediatr Hematol Oncol Am Soc Pediatr Hematol Oncol 1998, 20(3):260–263.

46. Haddow LJ, Nahikianavare MS, Ramdial PK, Moosa MY. Histopatology of Strongyloides stercoralis hyperinfection during immune reconstitution in an HIV-infected patient. AIDS 2009, 23(12):1601–1601.

47. Jamil SA, Hilton E. The Strongyloides hyperinfection syndrome. New York State J Med 1992, 92(2):67–68.

48. Purvis RS, Beightler EL, Diven DG, Sanchez RL, Tyring SK. Strongyloides hyperinfection presenting with petechiae and purpura. Int J Dermatol 1992, 31(5):169–171.

49. Boken DJ, Leeni PA, Preheim LC. Treatment of Strongyloides stercoralis hyperinfection syndrome with thiabendazole administered per rectum. Clin Infect Dis Infect Dis Soc Am 1993, 16(1):123–126.

50. Celedon JC, Mathur-Wagh U, Fox J, Garcia R, Wiest PM. Systemic strongyloidiasis in patients infected with the human immunodeficiency virus. A report of 3 cases and review of the literature. Medicine 1994, 73(5):266–273.

51. El Masry HZ, Odonnell J. Fatal strongyloidiasis hyperinfection in heart transplantation. J Heart Lung Transpl Int Soc Heart Transpl 2005, 24(11):1980–1983.

52. Foreman EB, Abraham PJ, Garland JL. Not your typical strong infection: a literature review and case study. South Med J 2006, 99(8):847–852.

53. Friedenberg F, Wongpraparut N, Fischer RA, Gubernick J, Zaeri N, Eiger G, Kothary NN, Muskie JM, Mathur SC. Strongyloides hyperinfection syndrome: report of two cases. Ann Diagn Pathol 2003, 7(2):87–94.

54. Lin AL, Kissimmian N, Benditt JO. Restrictive pulmonary disease due to interlobular septal fibrosis associated with disseminated infection by Strongyloides stercoralis. Arch Intern Med 1994, 151(1):205–209.

55. Link K, Orenstein R. Bacterial complications of strongyloidiasis: Streptococcus bovis meningitis. South Med J 1999, 92(7):728–731.

56. Malhotra A, Kochar T, Rangasaty UC. A perfect host. Intern Med J 2008, 38(1):869–870.

57. Saadeh MW, Buell JF, Gupta M, Conway GD, Akhter SA, Waggoner LE. Strongyloides hyperinfection syndrome after heart transplantation: case report and review of the literature. J Heart Lung Transpl Int Soc Heart Transpl 2004, 23(7):905–911.

58. Morrow S, Soiffer FM, Lin CS, Wolfe DE. Central nervous system Strongyloides stercoralis in acquired immunodeficiency syndrome: a report of two cases and review of the literature. Acta Neuropathol 1993, 86(3):285–288.

59. Reiman S, Fisher R, Dodds C, Trinh C, Laucirica R, Whigham CJ. Strongyloides stercoralis hyperinfection in a carrier of HTLV-I virus with evidence of selective immunosuppression. Am J Med 1992, 92(2):202–208.

60. Gupta S, Jain A, Fanning TV, Couriel DR, Jimenez-C, Eapen GA. An unusual cause of alveolar hemorrhage post hematopoietic stem cell transplantation: a case report. BMC Cancer 2006, 6:87.

61. Peraza-Ma SA, Sadigh M. Acute nonspecific illness in an AIDS patients with dysphagia. Hosp Pract (Off Ed) 1999, 24(11):39–47.

62. Phelps AR, Ginsberg SS, Cunningham AW, Tschachler E, Dosik H. A case report: adult T-cell leukemia/lymphoma associated with recurrent strongyloidiasis. Am J Med Sci 1991, 302(4):224–228.

63. Güçtaç AS, Leona NT, Hosing C, De Lima M, Cortes J, Massaro A, Rivera Z, Deavers M, Adachi JA, Chaplin RE. Strongyloides stercoralis hyperinfection during bone marrow transplantation: a case report. Bone Marrow Transplant 2006, 38(5):393–394.

64. Purvis RS, Beightler EL, Diven DG, Sanchez RL, Tyring SK. Strongyloides stercoralis hyperinfection. Int J Dermatol 1992, 31(3):160–164.

65. Krishnamurthy R, Dincer HE, Whittemore D. Strongyloides stercoralis hyperinfection in a patient with rheumatoid arthritis after anti-TNF-alpha therapy. J Clin Rheumatol: Rheumatic & Musculoskeletal Dis 2007, 13(1):150–152.

66. Reddy TS, Myers JW. Syndrome of inappropriate secretion of antidiuretic hormone and nonpalpable purpura in a woman with Strongyloides stercoralis hyperinfection. Am J Med Sci 2003, 325(5):286–291.

67. Reiman S, Craven DE. Images in clinical medicine. Strongyloides stercoralis hyperinfection. New England J Med 2008, 359(11):e12.

68. Reiman S, Fisher R, Dodds C, Trinh C, Laucirica R, Whigham CJ. Mesenteric arteriographic findings in a patient with strongyloidiasis. J Clin Rheumatol: Rhenumatic & Musculoskeletal Dis 2007, 13(1):150–152.

69. Rosby AC, Gottlieb GS, Limaye AP. Strongyloides stercoralis hyperinfection in transplant patients. Clin Inf Dis Infect Dis Soc Am 2009, 49(9):1411–1422.

70. Patel G, Arvelakis A, Sauter BV, Gondolesi GE, Capiolks D, Huprikar S. Strongyloides stercoralis hyperinfection after intestinal transplantation. Transpl Inf Dis J Transplant Soc 2008, 10(2):137–141.

71. Schindzielorz A, Edberg SC, Bia FJ. Strongyloides stercoralis hyperinfection and central nervous system involvement in a patient with relapsing polychondritis. Southern Med J 1991, 84(5):1055–1057.
Fatal adult respiratory distress syndrome: a review of the literature. Buonfrate et al. BMC Infectious Diseases 2013, 13:78

manifestations of Strongyloides stercoralis hyperinfection in an HIV-immunocompromised host. Thorax 2002, 57(7):752.

Fatal adult respiratory distress syndrome following lung transplantation. Transplant Inf Dis: J Transplant Soc 2011, 13(5):e503.

Strongyloides hyperinfection syndrome: a case report. Ann Trop Med Parasitol 2002, 96(2 Pt 1):265–266.

Primary Strongyloides stercoralis eggs in a urethral smear after bone marrow transplantation. Clin Inf Dis: Inf Dis Soc Am 2002, 34(9):1280–1281.

Acute respiratory insufficiency caused by hyperinfestation with strongyloides. BALF diagnosis and favourable outcome. Rev Pneumol Clin 1992, 48(2):75–78.

Strongyloides stercoralis hyperinfection syndrome: a case report. Cytology: Journal of the British Soc Clin Cytol 2010, 21(5):345–347.

Disseminated Strongyloides stercoralis: hyperinfection during medical immunosuppression. J Am Acad Dermatol 2010, 63(5):986–992.

Strongyloides hyperinfection syndrome following lung transplantation. Transplant Inf Dis: J Transplant Soc 2011, 13(5):e503.

Strongyloides stercoralis hyperinfection in patients with human T-cell lymphotropic virus type 1 infection: report of two cases. JID: Int Soc Inf Dis 2009, 193(6):e501–e503.

Fatal adult respiratory distress syndrome. Another cause of focal hepatic lesions. Clin Imaging 1993, 17(4):274–275.

Children with in vivo diagnosis of Strongyloides stercoralis hyperinfection associated with repititive bacterial meningitis and SIADH: a case report. Acta gastro-enterologica Belgica 2008, 71(4):413–417.

Strongyloides stercoralis infection associated with repititive bacterial meningitis and SIADH: a case report. Acta gastro-enterologica Belgica 2008, 71(4):413–417.

Strongyloides hyperinfection syndrome complicating (ectopic) Cushing syndrome. J Int Care Med 2002, 17(11):583–585.

Fatal adult respiratory distress syndrome following lung transplantation. Transplant Inf Dis: J Transplant Soc 2011, 13(5):e503.
137. Marisota S, Pallone G, Li Branci E, Gilardi G, Bisioli A: Strongyloides stercoralis hyperinfection in a case of idiopathic pulmonary fibrosis. Pnamminza Med 1996, 38(1):45–47.

138. Pampiglione S, Pampiglione E, De Stefano MA: Strongyloides stercoralis hyperinfection with encephalitis manifestations. Pathologica, 1993, 85(106):195–204.

139. Scotto U, Scarlata F, Giordano S, Tortorici C, Bono L, Coglitore M, Faraci C, Infrunni L, Rubino R, Romano A: Nephrotic syndrome and Gram-negative sepsis in a patient with strongyloidiasis: a case report. Le infezioni in medicina: rivista periodica di epatologia, epidemiologia, diagnostica, clinica e terapia delle patologie infettive, 2015, 15(1):59–62.

140. Sidoni A, Polidori GA, Scionti L: Strongyloides stercoralis hyperinfection diagnosed by Papanicolaou-stained sputum smears. Pathologica 1994, 86(1):137–90.

141. Lanzafame M, Faggian F, Lattuada E, Antolini D, Vento S: Strongyloides stercoralis as an immune restoration phenomenon in an HIV-1-infected and HTLV-1 carrier with increased proviral load. Int J STD AIDS 1996, 7(1):12–4.

142. Gomez J, Plaza V, Munoz C, Franquet T: Strongyloides stercoralis hyperinfection: difficulties in diagnosis and treatment. Anaesesthesia 2010, 65(3):298–301.

143. Gill GV, Beeching NJ, Khoo S, Bailey JW, Partridge S, Blundell JW, Luksha AR: A British Second World War veteran with disseminated strongyloidiasis. T Roy Soc Troop Med H 2004, 98(6):382–386.

144. Harcourt-Webster JN, Scaselli F, Danwitz AH: Strongyloides stercoralis hyperinfection in an HIV positive patient. J Clin Pathol 1991, 44(6):346–348.

145. Orlet H, Crawley C, Cwynarski K, Dina R, Apperley J: Strongyloidiasis pre and post autologous peripheral blood stem cell transplantation. Bone Marrow Transplant 2005, 32(1):115–117.

146. Pagliuca A: Strongyloides hyperinfection in adult T-cell leukaemia/lymphoma. British J Haematol 1999, 105(1).

147. Rahim S, Daru B, Yavis K, Melville D: Strongyloidiasis: a mistaken diagnosis and a fatal outcome in a patient with diarhoea. Trans R Soc Trop Med H 2005, 99(3):215–217.

148. Heath T, Riminton S, Garcia R, MacLeod C: Systemic strongyloidiasis complicating HIV: a promising response to ivermectin. Int J STD AIDS 1996, 7(4):294–296.

149. Lim L, Biggs BA: Fatal disseminated strongyloidiasis in a previously treated patient. Med J Australia 2001, 174(3):355–356.

150. Mak DB: Recurrent bacterial meningitis associated with strongyloides hyperinfection. Med J Australia 1993, 159(3):354.

151. Mak DB: Recurrent bacterial meningitis associated with strongyloides hyperinfection. Med J Aust 1993, 159(3):354.

152. Thomas MC, Costello SA: Disseminated strongyloidiasis arising from a single dose of dexamethasone before steroidic radiotherapy. Int J Clin Pract 1998, 52(7):520–521.

153. Kanazawa S, Yamaguchi K, Kinoshita Y, Nomura S: Adult T-cell leukaemia and strongyloidiasis. Eur J Cancer Care (Engl) 2008, 1(7):209–210.

154. Kinjo T, Tsuchako K, Nakazato I, Ito E, Sato T, Koyanagi Y, Iwamatsu T: Extensive intra-alveolar haemorrhage caused by disseminated strongyloidiasis. Int J Parasitol 1998, 28(3):332–336.

155. Miyazaki M, Tamura M, Kabashima N, Serino R, Shibata T, Miyamoto T, Furuno Y, Nishio T, Ohara J, Sakurai T, et al: Minimal change nephritic syndrome in a patient with strongyloidiasis. Clin Exp Nephrol 2010, 14(4):367–371.

156. Mori S, Kanishi T, Matsuoka C, Deguchi M, Ohta M, Mizuno O, Iino T, Okinaka T, Nishimura Y, Ito N, et al: Strongyloides stercoralis associated with nephrotic syndrome. Intern Med 1998, 37(7):606–610.

157. Morimoto J, Kaneoka H, Sasatomi Y, Sato YN, Murata T, Oghara S, Sakata N, Takebayashi S, Naito S, Ito T: Disseminated strongyloidiasis in a nephropathic patient. Clin Exp Nephrol 2002, 5(3):356–361.

158. Satoh M, Futami A, Takahisa K, Kodama M, Tanaka T, Kuriki K, Hori E: Severe disseminated strongyloidiasis complicated by meningitis and hydrocephalus in an HTLV-I carrier with increased proviral load. J Infect Chemother 2003, 9(4):335–357.

159. Sotyomaya M, Fukumaru S, Takasaki T, Yoshida H, Kanazaki T: SLE with death from acute massive pulmonary hemorrhage caused by disseminated strongyloidiasis. Scand J Rheumatol 1997, 26(5):389–391.

160. Wong WW, Leung WK, To KF, Sung JJ: Diarrhoea and rash in a retired farmer. HKMO, Xianggang yu xue za zhi/Hong Kong Academy of Medicine 2005, 11(5):397–398.

161. Ghez DD, Wen YY, Chen ML: Minimal change nephrotic syndrome in association with strongyloidiasis. Clin Nephrol 2006, 66(6):459–463.

162. Huang MS, Hwang KP, Chang PC, Hwang J: Pulmonary hyperinfection with Strongyloides stercoralis. J Formos Med Ass, Taiwan yh 1996, 95(4):551–554.

163. Ting YM: Pulmonary strongyloidiasis--case report of 2 cases. Kaohsiung J Med Sci 2000, 16(1):269–274.

164. Liu HC, Hsu YJ, Chang KM: Strongyloides stercoralis hyperinfection presenting with symptoms mimicking acute exacerbation of chronic obstructive pulmonary disease. AMA 2009, 72(9):442–445.

165. Hira PR, Ali Ali F, Sheikhi HM, Abdella NA, Johny M, Francis I, Iqbal J, Thompson R, Nevar F: Strongyloidiasis: challenges in diagnosis and management in non-endemic Kuwait. Ann Trop Med Parasitol 2004, 98(3):261–270.
Strongyloides stercoralis in patients with chronic lymphocytic leukemia and molecular characterization of the isolate. Korean J Parasitol 2008, 46(4):261–263.

90. Gullbaas Z, Kebapci M, Pasaoğlu O, Vardareli E. Successful ivm treatment of hepatic strongyloidiasis presenting with severe eosinophilia. J Exp Med 2009, 97:907–910.

91. Incani RN, Hernandez M, Cortez J, Gonzalez ME, Cortez J, Gonzalez ME, Salazar YD. Staphylococcus warneri meningitis in a patient with Strongyloides stercoralis hyperinfection: first report of a case. Rev Inst Med Troop Sao Paulo 2010, 52(2):169–170.

92. Chaudhary K, Smith RJ, Himelright IM, Baddour LM. Case report: purpura in a patient with Strongyloides stercoralis hyperinfection and pneumonia: first report of a case. Rev Inst Med Trop Sao Paulo 2010, 52(2):169–170.

93. Davidson RA. An unusual cause of pulmonary glomerulonephritis. Arch Pathol Lab Med 2009, 133(1):337–339.

94. Roman-Velez JM, Martinez-Camacho RN, Mayon-Lagarie D, Fernandez-Gonzalez R, Reyes-Sosa R, Santos-Llanos G, Colmen-Perez M, Fierrez D. An unusual presentation of alveolar hemorrhage. PCR J Gen Pract Airways 2009, 18(5):517–526.

95. Arsic-Arsenijevic V, Djamic A, Djamic Z, Milobratovic D, Tomic D. Fatal Strongyloides stercoralis infection in a young woman with lupus glomerulonephritis. J Nephrol 2005, 18(6):787–790.

96. Finkleman JD, Grinberg AR, Paz LA, Plana JL, Benchetrit GA, Nicastro MA, Roncoroni AJ. Case report: reactive hemophagocytic syndrome associated with disseminated strongyloidiasis. Am J Med Sci 1996, 312(1):37–39.

97. Yun HR, Yoo DH, Lee HS, Kim TH, Ahn MH, Min DY, Park MH, Kim SY. Fatal strongyloidiasis hyper-infection in a patient with rheumatoid arthritis. Clin Exp Rheumatol 2001, 19(2):224.

98. Koh MS, Leng PH, Eng F, Hwang J. An unusual cause of pulmonary hematoma in a patient with rheumatoid arthritis. Ann Acad Med Singap 2004, 33(3):365–367.

99. Leung VK, Liew CT, Sung JJ. Fatal strongyloidiasis in a patient with ulcerative colitis after corticosteroid therapy. J Am Gastroenterol 1997, 92(8):1383–1384.

100. Shorman M, Al-Tawfiq IA. Strongyloides stercoralis hyperinfection presenting as acute respiratory failure and Gram-negative sepsis in a patient with asperctoma. J Infect 2010, 60(5):566–572.

101. Asztalos EM, Nitschmann M, Shariati S, Abdulla A, Nampoori NR, Iqbal J, Nair PM, Said T. Strongyloides hyperinfection and lymphoma: first report of a case. Eur J Int Med 2004, 18(5):479–485.

102. Baddour LM. Case report: fatal Strongyloides stercoralis hyperinfection in a leprosy patient on treatment for a type II leprosy reaction. Lepr Rev 2004, 75(4):398–403.

103. Wong TY, Serto CC, Lai FF, Mak CK, Li PK. Nephrotic syndrome in strongyloidiasis: remission after eradication with antihelmintic agents. Nephron 1998, 79(3):333–336.

104. Adedayo AO, Grell GA, Bellot P. Case study: Fatal strongyloidiasis associated with human T-cell lymphotropic virus type 1 infection. Am J Med Trop Med Hyg 2001, 65(3):350–351.

105. Foncin J, Genevier I, Lamy A, Robel M. [Aseptic purulent meningitis in two patients co-infected by HTLV-1 and Strongyloides stercoralis]. Med Trop (Mars) 1997, 57(3):262–264.

106. NTdI K, Kousae KE, Safari R, Hoomdel D, Hulin A: [Strongyloides stercoralis hyperinfection syndrome with acute meningoencephalitis associated to HTLV-1 and HTLV-2 virus co-infection]. Med Mal Infect 2008, 38(3):162–164.

107. Drug V, Haliga R, Akbar Q, Nilai C, Cjewkiwc Pieliecapcean C, Stanciu C. Aspects with Strongyloides stercoralis in a patient with acute alcoholic pancreatitis and liver cirrhosis. JGLD 2009, 18(3):367–369.

108. Ho PL, Luk WK, Chan AC, Yuen KY. Two cases of fatal strongyloidiasis in Hong Kong. Pathology 1997, 29(3):324–326.

109. WHO. Model Lists of Essential Medicines. http://www.who.int/medicines/ publications/essentialmedicines/en/.