third decades of life [2]. Solitary abscess is more common and seen mostly in the thoracic cord. Abscesses are considered primary when no other infection source can be found. Secondary abscesses (upto 85% cases) arise from another infection site, either contiguous to cord (dermal sinus or neural tube defect) or distant (most commonly from lung) [1, 3]. They are also classified as acute (<1 week), sub-acute (1- 6 weeks) or chronic (>6 weeks) [2]. Our case did not show a congenital malformation of the spine and clinical features were of insidious onset, suggestive of sub-acute primary solitary abscess. Organisms isolated include *Staphylococcus* [4] *and Mycobacterium tuberculosis* [5]. However, 25-40% abscesses are sterile on culture, as in our case [4].

In an acute presentation, symptoms of infection (*e.g.* fever, backache, malaise) are common. Chronic cases might mimic features of intramedullary tumor and show neurological symptoms [6]. The procedure of choice for diagnosis of intramedullary spinal abscess is gadolinium-enhanced MRI that shows rim enhancement of its margins. Spinal cord abscesses produce homogenous enlargement on T1-weighted images and hyperintensity on T2-weighted images [4]. These findings may be seen in intramedullary tumors as well.

Treatment of intramedullary abscesses involves surgical drainage and appropriate antibiotics. Steroids are used to reduce spinal cord swelling and associated edema [7]. Paradoxical increase in size of lesion may occur necessitating surgical intervention [8].

Approximately 70% of patients may have residual neurological sequelae [9]. Some patients may show paraplegia due to recurrent or non-resolving abscess and infarct due to vascular occlusion and inflammation.

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REFERENCES

1. Ditullio MV. Intramedullary spinal cord abscess: case report with review of 53 previously described cases. Surg Neurol. 1997;7:351-4.
2. Menezes AH, Graf CJ, Perret GE. Spinal cord abscess: review. Surgical Neurology. 1977;8:461-7.
3. Benzyl D, Epstein MH, Knuckey NW. Intramedullary epidermoid associated with an intramedullary spinal abscess secondary to a dermal sinus. Neurosurgery. 1992;30:118-21.
4. Murphy KJ, Brunberg JA, Quit DJ, Kazanjian PH. Spinal cord infection: myelitis and abscess formation. AJNR AM J Neuroradiol. 1998;19:341-8.
5. Hoda MF, Prasad R, Singh VP, Maurya P, Singh K, Sharma V. Spinal intramedullary tubercular abscess: a case report. Indian J Tuberc. 2005;52:211-4.
6. Guzel N, Eras M, Guzel DK. A child with spinal intramedullary abscess. Childs Nerv Syst. 2003;19;773-6.
7. Schroeder KA, Mckeever PE, Schaberg DR, Hoff JT. Effect of dexamethasone on experimental brain abscess. J Neurosurgery. 1987;66:264-9.
8. Citow JS, Ammirati M. Intramedullary tuberculoma of the spinal cord: Case report. Neurosurgery. 1994;35:327-30.
9. Kalia V, Vibhuti, Aggarwal T. Holocord intramedullary abscess: clinical brief. Indian J Pediatr. 2007;74:589-91.

Disseminated Strongyloidiasis in a Immunocompromised Host

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Strongyloidiasis in an immunocompromised patient has the potential to be life threatening. We describe a boy who was on steroids for acute demyelinating myelitis and receiving antibiotics for *E.coli* UTI and meningitis. He developed anasarca, malabsorption, malnutrition and left ventricular failure. Duodenal biopsy revealed abundant rhabditiform larvae of *Strongyloides stercoralis*. The diagnosis went unsuspected and proved fatal. This emphasizes the need to have a high index of suspicion and early intervention for *S. stercoralis* in immunosuppressed persons who present with refractory gastrointestinal symptoms.

Key words: *Immunodeficiency, Strongyloidiasis.*

Strongyloidiasis is an intestinal infestation caused by the nematode *Strongyloides stercoralis*, common in endemic areas of tropical and subtropical countries. In an immunocompromised patient, it has the potential to cause life threatening conditions like hyper-infection syndrome and disseminated strongyloidiasis. Severe strongyloidiasis has a high mortality of up to 80% as the diagnosis is often delayed. We describe a boy who was immunocompromised secondary to systemic steroid therapy, in whom the diagnosis was delayed.

CASE REPORT

A thirteen year old boy presented with acute progressive paraplegia and bladder incontinence. MRI revealed affection of spinal cord from the level of T1 to conus medullaris. CSF examination was normal. Diagnosis of acute demyelinating myelitis was made. Child was treated with intravenous methyl prednisolone for five days which was followed by oral prednisolone for three weeks. He recovered completely and steroid was tapered over next three weeks. However, at the end of six weeks after starting steroids, child presented with abdominal pain and distension, and vomiting. Clinical and abdominal examination were unremarkable. Hemogram revealed a total count of 15,000/cmm with 60% neutrophils, 36% lymphocytes and 4% eosinophils. HIV serology was negative. Urine microscopy revealed pyuria and urine culture grew *E.coli* in significant colony count. Child was started on intravenous antibiotics Amikacin and Ceftriaxone as per sensitivity report. While on day seven of antibiotics, child developed headache and meningeal signs. CSF examination revealed polymorphic pleocytosis with normal sugar and mildly elevated protein; Gram staining, AFB staining and culture were negative. CT scan of head was normal. Child continued to have abdominal distention, vomiting and developed persistent diarrhea. Stool routine evaluation was normal, there were no ova, cysts or pus cells, and no fat globules or reducing substances. There was no growth on stool culture. Child was treated with parenteral fluids and electrolytes as serum sodium remained persistently low. Repeat stool routine examination, and USG abdomen and erect *X*-ray abdomen were normal. Child underwent upper GI endoscopy and duodenal biopsy was taken. The condition of the child progressively worsened with development of severe malnutrition, malabsorption and anasarca. Child gradually progressed to hypotension and muffled heart sounds with left ventricular failure. Echocardiography revealed mild to moderate pericardial effusion. However, the child expired before a pericardiocentesis could be done. Duodenal biopsy report later revealed blunting with abundant rhabditiform larvae of *S. stercoralis*.

DISCUSSION

S. stercoralis usually persists and replicates in a host for a decade without symptoms. However when the host becomes immunocompromised, it can lead to fatal conditions like hyper-infection syndrome and disseminated strongyloidiasis. The disease should be suspected in an immunocompromised host who comes from an area endemic for *Strongyloides stercoralis*. In endemic areas, a prevalence of as high as 40% is observed in general population. However, disseminated strongyloidiasis is very rare in immunocompetent host. Clinical manifestations of disseminated strongyloidosis are nonspecific. The onset is usually sudden with generalized abdominal pain, distension and fever, associated with indigestion, vomiting, diarrhea, steatorrhoea, protein losing enteropathy and weight loss. There is remarkable absence of eosinophilia. Steroids may not only affect the host's cellular immunity, but also mimic an endogenous parasitic-derived regulatory hormone. Strongyloides were noticed to produce more eggs in the presence of exogenous steroids. Due to immuno-suppressant therapy, there is a larger proportion of the rhabditiform larvae which mature into the filariform larvae within the host. This leads to a greater larval load and dissemination. It involves widespread dissemination of larvae to extra intestinal organs (CNS, heart, urinary tract, endocrine organs) which are not ordinarily part of parasitic lifecycle. The enteric organisms either carried by the larvae or through intestinal ulcers, cause bacteremia.

Hyperinfection implies confinement of the Strongyloides larvae to the organs normally involved in the pulmonary autoinfection cycle (*i.e.*, GI tract, lungs, and peritoneum). Disseminated strongyloidiasis is defined as larvae migrating to end organs not usually involved in the normal cycle of the parasite, such as brain and skin. The definitive diagnostic test is identification of *S. stercoralis* larvae in stool examination. Single stool examination has low sensitivity (30%). Hence multiple examinations are recommended. In children with hyper-infection syndrome, larvae may be found in samples from sites of potential larva migration like duodenal aspirate, sputum, BAL fluid, lung biopsy and rarely in small intestine biopsy specimens. Stool microscopy done twice, in this child, during the illness did not reveal the larvae. This child developed left ventricular failure on the last day and there was significant pericardial effusion. The reason for heart failure was presumed to be due to disseminated strongyloidiasis. Myocardial involvement in disseminated strongyloidiasis has been described in literature [7].

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REFERENCES

1. Kliegman MR, Behrman RE, Jenson HB, Stanton BF, Editors. Strongyloidiasis (*Strongyloides Stercoralis)*. Nelson Textbook of Pediatrics, 18th Ed, Philadelphia (Pennsylvania): Saunders; 2008. p. 1501-2.
2. Daubenton JD, Buys HA, Hartley PS. Disseminated strongyloidiasis in a child with lymphoblastic lymphoma. J Pediatr Hematol Oncol. 1998;20:260-3.
3. Sarubbi FA. Hyperinfection with strongyloides during treatment of pemphigus vulgaris. Arch Dermatol. 1987;123:864-5.
4. Reddy IS, Swarnalata G. Fatal disseminated *Strongyloidiasis* in patients on immunosuppressive therapy: Report of two cases. Indian J Dermato Venerol Leprol. 2005;71:38-40.
5. Marcos LA, Terashima A, Dupont HL, Gotuzzo E. *Strongyloides* hyperinfection syndrome: an emerging global infectious disease. Trans R Soc Trop Med Hyg. 2008;102:314-8.
6. Nutman TB, Keiser PB. *Strongyloides stercoralis* in the immunocompromised population. Clin Microbiol Rev. 2004**;**17:208-17.
7. Potter A, Stephens d, de Keulenaer B. Strongyloides hyper-infection: a case for awareness. Ann Trop Med Parasitol. 2003;97:855-60.

Spontaneous Pneumomediastinum in H1N1 Infection

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Spontaneous pneumomediastinum is an uncommon pediatric emergency which usually occurs secondary to bronchial asthma in children. We report a case of spontaneous pneumomediastinum in a 7 year child following Swine Flu (H1N1) infection.

Key words: *Complication, Management, Pneumomediastinum, Swine flu.*

Spontaneous pneumomediastinum in children is triggered by asthma, vomiting, situations reproducing the Valsalva maneuver (e.g. shouting, coughing, inhalation of drugs, and intense sport activities [1]. We report an unusual spontaneous pneumomediastinum caused by Swine Flu (H1N1) infection. Very few similar cases are reported till date [2].

CASE REPORT

A 7-year-old female child presented with severe cough, high grade fever and breathlessness for 3 days prior to admission. At admission, she had maculopapular rash all over the body, sore throat and tachypnea. Respiratory system examination revealed fine crepitation bilaterally. All other systemic examination was within normal limit. Hemoglobin was 10.2 g/dL and total leukocyte count was 4200 cells/cumm with lymphocytic (70%) predominance. Blood culture and endotracheal aspirate culture revealed no growth. Chest *X*–ray revealed bilateral streaky opacities. A throat and nasal swab was sent to rule out Novel H1N1. She was put on broad spectrum antibiotics, intravenous fluid and oseltamivir. After 48 hours of admission, she developed severe stabbing chest pain. This was accompanied with subcutaneous emphysema along with deteriorating oxygen saturation. Blood gas analysis revealed (pH 7.51, PaO_2 50mmHg, PCO_2 28, Spo_2 90%, HCO_3 26, BE 2.4, $AaDo_2$ 70 mmHg). Chest *X*-ray revealed underlying pneumomediastinum. The child was put on pressure control mode of mechanical ventilation. Trachostomy was done, as the subcutaneous emphysema was increasing. Following six hours of tracheostomy, there was complete disappearance of mediastinal air with total resolution of subcutaneous emphysema at 24 hourss. The child was weaned off from mechanical ventilation. However, the child developed Acute respiratory distress syndrome (ARDS) on day 7 of admission and died.

DISCUSSION

The index case had no other apparent risk factor apart from vigorous cough in addition to severe H1N1 infection, which is known to cause diffuse alveolar damage and