done in CSF, which came positive *A. cantonenis*. She was started on prednisolone 2 mg/kg/day along with Albendazole 15 mg/kg/day for 2 weeks. Within 48 hours of treatment she became afebrile, her sensorium improved with improvement in activity and appetite. After 2 weeks of hospital stay she was discharged in a stable condition with an absolute eosinophil count of 625/mm^3 on day 20.

Peripheral eosinophilia is an immunologically mediated response to various conditions like allergic (atopy, medications), neoplasms (leukemia, lymphoma, tumor associated) drug induced hypersensitivity and infectious diseases (parasite, fungal). It is essential to look for non infectious causes of hyper-eosinophilia symptoms, before looking for parasitic infection.

Overall parasitic meningitis is rare, but exact incidence and prevalence is not reported. Among the three major helminthes that cause EM [1,2], *A.cantonensis* is the most common. Neurocysticrcosis, although rare, also is known to cause EM in endemic areas.

Similar infections have been described in Southeast Asia, South Pacific, Taiwan, Africa, Caribbean, Australia and North America [1]. Shipboard travel of rats is the most common cause for the spread of parasite to other continents as rats are the definitive host. [2,3] and human beings are the accidental hosts. Infection occurs due to ingestion of third stage larvae in raw or undercooked snails or fish and children who play in the dirt in endemic areas are prone for infection. Eosinophilic meningitis is diagnosed presumptively based on travel or exposure history with CSF analysis showing >10% eosinophils, mildly elevated protein and normal glucose or hypoglycorrhachia [5]. Peripheral eosinophilia peaks about 5 weeks after exposure. MRI brain demonstrates high signal intensities, leptomeningeal enhancement, hyper intense signals on T2W image. For detection of parasite in CSF, ELISA is sensitive and specific but is limited by commercial non availability and cross reactivity between helminthic parasites. PCR based studies are sensitive in detecting the parasite DNA.

Treatment is mainly supportive with analgesics, corticosteroids and anthelminthic drugs [6,2]. There is higher incidence of neurological sequelae among children and prognosis is good with 70% improvement within 1-2 weeks and mortality is <1%.

This case is presented to sensitize the clinician, the rarity of EM, caused by helminthic infection. The possibility of eosinophilic meningitis/parasitic meningitis must be considered in a patient with fever, peripheral eosinophilia, headache with or without meningeal signs. In such patients, CSF eosinophil staining is recommended along with demonstration of parasite antigen through real time PCR will help to establish parasitic etiology and also in prognostication and appropriate follow up.

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Undifferentiated Nasopharyngeal Carcinoma and Paraneoplastic Leukemoid Reaction

Nasopharyngeal carcinoma is a rare pediatric malignancy [1]. In this report, we describe a nasopharyngeal carcinoma in child presenting with paraneoplastic leukemoid reaction (PLR). Adult patients with solid tumors presenting with PLR have been reported in the past, but very few pediatric cases have been described [1].

A 11-year-old boy, with a history of global developmental delay, presented with bilateral neck swelling that progressively increased over two months, associated with loss of weight, increased frequency of fever spikes, tiredness and difficulty in swallowing solid feeds. There was no history of contact with tuberculosis. Developmental age was 4 years and his antenatal, natal and post-natal history was uneventful. On examination, he was awake, alert, cooperative, and responded to verbal commands. He was pale, febrile, and had bilateral cervical lymphadenopathy of 15x10x3 cm on the right side and 12x10x3 cm on the left side, non-tender, immobile and firm to hard in consistency. The child was underweight and stunted and head circumference was below 2 SD when compared to age- and sex-matched controls. At presentation, the child was febrile with

tachycardia (rate 110/min), temperature 101^0F, respiratory rate of 26/min and blood pressure 100/70 mm Hg. Peripheries were warm and well perfused. CNS examination revealed decreased muscle bulk in all 4 limbs with normal tone and reflexes. Other systems were unremarkable.

He was initially treated with an empirical 5-day course of amoxicillin for lymphadenitis. As the swelling did not subside, an excision biopsy of the left lymph node was done, which revealed granulomatous caseous necrosis suggestive of tuberculosis. In view of no response to anti tubercular treatment (ATT) after 3 weeks of therapy, and his total lymphocyte count showing neutrophilic predominance, a repeat excision biopsy of his right cervical node was done for further evaluation. His complete blood count revealed a total Hemoglobin of 7 g/dL, leucocyte count of 30,000 cells/mm^3 (86% polymorphs, 9% lymphocytes, and 5% mixed cells) and platelets were 607,000 cells/mm^3. Peripheral smear showed severe hypochromic anisopoikilocytosis and neutrophilic leukocytosis. Basic metabolic panel, liver function test, serum calcium, serum uric acid were normal and LDH of 430 U/L. Retroviral screening, urine, and blood cultures were negative. EBV serology was indicative of past infection. Repeat biopsy from a cervical lymph node showed atypical cellular infiltrate with surrounding fibrosis and inflammation. Immunohistochemistry staining of the biopsy specimen was positive for pan-cytokeratin (pan-CK) but negative for CK5/6, CK7, CK19 (A), CD15, CD30, placental alkaline phosphatase, and CD45 suggestive of metastatic carcinoma. Diagnostic nasal endoscopy (DNE) revealed a polyp in the nasopharynx biopsy which was sent for histopathological examination. CT scan of the neck revealed bilateral II, III, IV, and V cervical lymphadenopathy, enlarged retropharyngeal nodes of 2.6×2.0 cm with multiple necrotic areas. Subsequently, his WBC count on day 10 and 11 of hospital stay increased to 56,000 and 68,200 cells/mm^3, respectively (96% neutrophils, 3% lymphocytes and 1% mixed cells), suggesting a hematological of malignancy. Biopsy from the DNE specimen, however, revealed ill-defined sheets of tumor cells **(**Schmincke pattern [2])**,** and vesicular nuclear chromatin with prominent nucleoli and a high nuclear to cytoplasmic ratio with strong and diffuse positivity for pan-CK. Peripheral smear during this phase of hyperleukocytosis showed neutrophilic leukocytosis with predominantly mature forms of neutrophils, thrombocytosis and no evidence of blast cells. C-reactive protein level was 4 mg/dL and blood and urine cultures for bacteria and fungi were negative. The cervical lymph node biopsy and the nasopharyngeal specimen stained positive for pan-CK favored the diagnosis of advanced undifferentiated carcinoma of the nasopharyngeal type $T_xN_{3a}M_0$ – stage IVB. The hyper-leukocytosis was explained by a paraneoplastic leukemoid reaction after ruling out other common causes of hyper-leukocytosis. The child was treated with cisplatin and 5-fluorouracil, and subsequently treated with radiation therapy. On treatment the white cell count reduced thereby confirming paraneoplastic leukemoid reaction. His symptoms improved during the first 6 months of therapy, but he subsequently developed bone metastasis and died after 19 months of initial diagnosis.

The most common variant of nasopharyngeal carcinoma in children is the undifferentiated non-keratinizing carcinoma most commonly presenting as a neck mass [1]. Granulomatous response to the tumor may be dominant in a few cases of nasopharyngeal carcinoma [2], which probably led to the misdiagnosis of tuberculosis in the first place. A marked rise in leukocyte count suggested a hematological malignancy but the staining of the DNE specimen with pan-cytokeratin confirmed an epithelial tumor. Paraneoplastic leukemoid reaction (PLR) in this case was diagnosed after ruling out infections, new malignancy, hemorrhage, and use of drugs like corticosteroids, G-CSF, and minocycline [3]. PLR is thought to be caused due to overproduction of cytokines like IL-10, IL-6, and GM-CSF, which stimulate the bone marrow to produce a large number of leukocytes [4]. In children presenting with solid tumors, PLR should be considered after ruling out more common causes of hyperleukocytosis like a hematological malignancy.

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