In the Literature

Capillariasis


Capillariasis in humans is caused by 2 species, Capillaria hepatica and Capillaria philippinensis. The former, as its name implies, infects the liver, while the latter infects the gastrointestinal tract. Capillaria hepatica infection, which is rare but has wide global distribution, is acquired by accidental ingestion of ova that have contaminated soil. In contrast, C. philippinensis infection is acquired by ingestion of raw or undercooked small freshwater fish and predominantly occurs in the Philippines and in Thailand, although cases have been reported from other countries in Asia and the Middle East, as well as in Colombia.

Limsrilai and colleagues reviewed the experience with all 26 patients seen with intestinal capillariasis at Siriraj Hospital in Bangkok from January 2001 to June 2013. Twenty (77%) were male, 17 (65%) were rural dwellers, and 14 of the 15 (93%) for whom information was available reported having eaten raw fish. The most frequent (77%) chief complaint was chronic diarrhea that was watery in most cases, whereas in the remainder it was either abdominal pain or edema. Two patients had abdominal pain and edema but no diarrhea. Abdominal pain was usually colicky without specific localization. The duration of symptoms ranged from 1 to 60 months (median, 5.5 months); 5 (19%) had been symptomatic for >12 months.

Hypoalbuminemia was present in all 26 patients, ranging from 0.6 g/dL to 3.0 g/dL (mean, 1.4 g/dL). Hypokalemia was identified in 18 (70%) patients, and 13 (50%) were anemic. Only 2 (8%) had a total eosinophil count >500 cells/µL. This was true despite the fact that 8 patients had additional helminthic infections—Opisthorchis viverrini in 5, Strongyloides stercoralis in 2, and hookworm in 1. Capillaria philippinensis was identified on stool examination in 15 (57.7%) patients, but required examination of a median of 4 stools before diagnosis. The parasite was identified on biopsy specimens obtained by esophagogastroduodenoscopy in 1 of 10 patients; ileal biopsy via colonoscopy was negative in all 7 patients with mucosal abnormalities in whom this procedure was performed. Push enteroscopy and balloon-assisted enteroscopy each provided positive results in 3 of 5 patients, with the proximal ileum was most frequently involved.

Patients were treated with prolonged courses of albendazole or ivermectin and clinical cure was obtained in all, with resolution of hypoalbuminemia within 1–2 months in the majority of patients. For treatment of capillariasis, the US Centers for Disease Control and Prevention (CDC) currently recommends mebendazole 200 mg twice daily for 20 days as the treatment of choice, but indicates that this drug is only available from compounding pharmacies in the United States [1]. Alternatively, the CDC recommends albendazole 400 mg once daily with food (preferably a fatty meal) for 10 days.

Making a diagnosis of intestinal capillariasis can be difficult as a result of a lack of eosinophilia and limited sensitivity of stool examination, as well as the fact that some patients have abdominal pain but do not complain of diarrhea. As a consequence, it is likely the infection is more common than one would judge from the small number of cases seen during more than a decade at this Bangkok hospital. Clearly, the diagnosis should be sought in patients with appropriate epidemiology, a history of eating raw or undercooked small freshwater fish, and unexplained watery diarrhea.

Reference


Acute HIV Infection: When Fitness Is a Bad Thing


The set-point viral load reached after the initial decline in the very high plasma concentrations of human immunodeficiency virus (HIV) after acute infection is a strong predictor of the rapidity of progression of HIV infection. The set-point is the result of the interplay of host immunogenetics and viral replicative capacity. Like the viral set-point, the intensity of immune activation, another important predictor of disease progression, is established early after infection and remains relatively stable over time. Recent observations indicate that the replicative
capacity of the founder virus is associated with the trajectory of the subsequent decay in CD4\(^+\) T-cell counts [1]. Claiborne and colleagues examined the hypothesis that high viral replicative capacity (vRC) drives excess immune activation, immune cell dysfunction, and increased infection of memory T-cell subsets, thus accelerating disease progression.

The investigators, utilizing data derived from a cohort of 127 Zambians who were newly infected (median, 46 days) with HIV-1 subtype C who participated in a study of discordant couples, confirmed this hypothesis. More specifically, they demonstrated that, during the early months of infection, there was a direct correlation between infection with virus with a high vRC and a distinctive proinflammatory cytokine profile (with elevated levels of, eg, interferon α and interleukin 1\(β\)), increased T-cell activation and proliferation, and exhaustion of CD8\(^+\) T cells. The vRC correlated with the viral load within both naive and memory CD4\(^+\) T cells.

The investigators demonstrated that the excess activation of CD8\(^+\) and CD4\(^+\) T cells appears to be attributable to the replicative capacity of the transmitted virus, likely via its induction of an early innate immune response and consequent elaboration of proinflammatory cytokines. The effect of vRC was largely independent of total viral load and of host genetically determined protective characteristics. The latter is consistent with the fact that HLA type, although considered the most important of the host factors, accounts for only approximately 20% of the overall variation in disease progression.

The investigators suggest that these findings have implications for vaccine development in that a successful vaccine may need to only target virus with high vRC.

**Reference**


**Case Vignette: Apparent Transmission of Scedosporium From an Organ Donor Who Experienced Near-drowning**


Pneumonia due to *Scedosporium* species is an often reported complication of near drowning. Kim and colleagues now report apparent transmission of this organism to at least 3 recipients of organs from a near-drowning victim.

A 19-year-old heart transplant recipient developed cerebral embolic events in association with intracardiac vegetations and fungemia due to *Scedosporium aurantiacum*. He died 17 days after transplantation. A 56-year-old renal transplant recipient developed renal failure, wound dehiscence, and pulmonary infection. He died 36 days after transplantation and 4 days after *S. aurantiacum* was detected in his blood cultures. Another renal transplant patient, a 57-year-old woman, developed infection of the transplanted organ with overlying wound infection and subsequently also developed central nervous system and ocular infection believed to be due to *S. aurantiacum*. She died on posttransplant day 58. Genetic analysis found that the fungal isolates from the 3 patients appeared to be indistinguishable. Recognition that all 3 patients had received organs from the same donor led to the administration of preemptive antifungal therapy to 2 patients who had received split-liver transplants from the same donor.

The donor was a 24-year-old man who attempted to drown himself in the Han River of South Korea but was admitted to hospital in cardiorespiratory arrest with diffuse pulmonary infiltrates. Since routine clinical cultures of the donor were negative, the donor who had suffered near-drowning could not be definitively stated to be the source of infection in the organ recipients. The likelihood of this transmission link is nonetheless great.