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1449 concurrent malignant disease (including chronic lymphocytic leu- kaemia and anal cancer). Furthermore, rare case reports have also suggested that B. coli can be associated with osteomyelitis of the cer- vical spine, and urogenital tract infections and kidney failure. Laboratory diagnosis Balantidiasis is most commonly diagnosed by microscopic exam- ination of freshly obtained diarrhoeal stools or colonic mucus obtained at sigmoidoscopy. Examination of wet mount slide pre- parations within less than 6 h after faecal collection shows cysts or motile trophozoites displaying characteristic spiralling move- ments. A preponderance of trophozoites is often found in diarrheal stool, while a greater proportion of cysts is seen in formed stool. Identification is greatly aided by the large size of the parasite, with ovoid-shaped trophozoites of 30–150 µm in length and 40–55 µm in width, or ovoid-shaped cysts of 40–65 µm in size. The large cyst size allows differentiation from the smaller cysts (10–20 µm) of Entamoeba histolytica, which can cause dysenteric symptoms similar to those of severe B. coli infection. Fixation and staining (with Lugol's iodine) can be done, but may obscure cellular details visible by phase-contrast microscopy of fresh specimens, and could lead to misdiagnosis as helminthic ova. Stools in suspected cases should be examined repeatedly over several days because parasite excretion may be intermittent. Histological examination of rectal biopsies may reveal B. coli trophozoites on sections stained with haematoxylin and eosin. Pulmonary balantidiasis can be diagnosed by bronchoalveolar lavage and finding the parasite in the lavage fluid. B. coli has also been detected by PCR in faecal samples, but the technology is not fully developed and not presently available for routine clinical diagnostics. Treatment and prevention Balantidiasis has been treated empirically with various antimicro- bial drugs (Table 8.8.9.2), although available reports are mostly an- ecdotal in regard to the effectiveness of such treatments. One trial in the 1970s showed that metronidazole was effective in eradicating the parasites in all of 20 patients over a range of doses (2.5 g over 5 days or 7.5 g over 10 days in children, or 5 g over 5 days or 12.5 g over 10 days in adults). Several case studies further support the general efficacy of metronidazole against balantidiasis, although at least one report exists in which the drug was apparently inef- fective. As an alternative, tetracycline has been shown to be effi- cacious against B. coli infection. Iodoquinol and nitazoxanide may also have therapeutic benefit, although these drugs have not been employed as widely as metronidazole and

tetracycline. The mech- anisms of action of any of these drugs have not been specifically investigated in B. coli, but in the absence of such information it would be reasonable to assume that they resemble those described in other target microorganisms of the respective drugs. Furthermore, resistance of B. coli to any of the commonly used drugs has not been reported. However, this possibility has not been adequately exam- ined, so it cannot be excluded that reported treatment failures are related to resistance. In rare situations, surgical intervention might be necessary in patients with extracolonic spread, such as liver ab- scess or appendicitis, or with colonic perforation. Prevention of balantidiasis involves avoidance of B. coli cyst ingestion, via filtra- tion or boiling of drinking water, hand washing before handling food, and careful cleaning and adequate cooking of food. A vaccine has not been developed. FURTHER READING Giardiasis Ankarklev J, et al. (2010). Behind the smile: cell biology and disease mechanisms of Giardia species. Nat Rev Microbiol, 8, 413–22. Feng Y, Xiao L (2011). Zoonotic potential and molecular epidemi- ology of Giardia species and giardiasis. Clin Microbiol Rev, 24, 110–40. Fink MY, Singer SM (2017). The intersection of immune responses, microbiota, and pathogenesis in giardiasis. Trends Parasitol, 33, 901–13. Miyamoto Y, Eckmann L (2015). Drug development against the major diarrhea-causing parasites of the small intestine, Cryptosporidium and Giardia. Front Microbiol, 6, 1208. Soares R, Tasca T (2016). Giardiasis: an update review on sensitivity and specificity of methods for laboratorial diagnosis. J Microbiol Methods, 129, 98–102. Upcroft P, Upcroft JA (2001). Drug targets and mechanisms of resistance in the anaerobic protozoa. Clin Microbiol Rev, 14, 150–64. Balantidiasis Arean VM, Koppisch E (1956). Balantidiasis. A review and report of cases. Am J Pathol, 32, 1089–115. Garcia-Laverde A, de Bonilla L (1975). Clinical trials with metro- nidazole in human balantidiasis. Am J Trop Med Hyg, 24, 781–3. Schuster FL, Ramirez-Avila L (2008). Current world status of Balantidium coli. Clin Microbiol Rev, 21, 626–38. 8.8.10 Blastocystis infection Richard Knight ESSENTIALS Blastocystis is an anaerobic unicellular noninvasive colonic parasite of animals and humans. It is transmitted faeco-orally, with human infection associated with travel, institutions, animal handlers, and immunodeficiency. Case reports strongly suggest that it causes a self- limited diarrhoeal illness. Diagnosis is by microscopic examination of Table 8.8.9.2 Oral drug regimens for treating balantidiasis in adults Drug Dose Treatment duration Metronidazole 250–400 mg/dose, 3 × doses/day 5–10 days Tetracycline 500 mg/dose, 4 × doses/day 10 days 8.8.10 Blastocystis infection

 faecal smears or concentrates. A trial of treatment with metronida- zole is justified in patients who are immunocompromised, also when symptoms are prolonged. Aetiology and biology of the parasite Molecular and ribosomal RNA studies now indicate that Blastocystis is a Stramenopile (a synonym for kingdom Chromista), currently only one species is recognized. Blastocystis has no flagellae, unlike other stramenopiles, which include slime nets, water moulds, and brown algae. The form commonly described in faeces and also in cultures is spherical, from 4 to 15 µm in diam- eter, with one prominent central vacuole, surrounded by periph- eral cytoplasm (Fig. 8.8.10.1) that electron microscopy shows to contain a nucleus, a Golgi complex, and mitochondrion-like or- ganelles (Fig. 8.8.10.2). It grows readily in cultures with mixed bacteria but axenic cultures can also be established; division is by binary fission. Transmission is by small, resistant, faecal cysts, from 3 to 8 µm in diameter. The basic life cycle alternates between the univacuolar and cystic stages, but electron microscopy of faeces and cultures may also show granular, and amoeboid forms of uncertain significance. Bizarre environmentally induced forms with huge vacuoles may develop in cultures (Fig. 8.8.10.3). The common ‘univacuolar’ form was named Blastocystis by Brumpt in 1912 as a yeast, although it was first described by Alexieff in

1911 as a protozoan cyst. Epidemiology Prevalence may exceed 35% in some human populations associ- ated with high faeco–oral transmission. This infection is associated with travel, institutions, animal handlers, and immunodeficiency. Blastocystis is genetically diverse and occurs in a wide range of domesticated and wild animals. Currently only one species is rec- ognized, but at least 17subtypes are described, with subtype ST3 the most common in humans. Important zoonotic sources are pigs, cattle, nonhuman primates, and birds, including chickens and ducks. The resistant cysts can occur in both sewage influents and effluents. Diagnosis Blastocystis is usually recognized as univacuolar forms in direct wet faecal smears or formol ether concentrates. Wet mounts can be stained with iodine, giving a brownish central body, or with tolui- dine blue. The organism is often numerous in symptomatic subjects. Permanent mounts stain well with trichrome. Blastocystis can re- semble amoebic cysts but lack their characteristic nuclei. In fixed smears stained specifically for Cryptosporidium, there is no oocyst wall. Special techniques are used to concentrate and identify cysts in environmental samples. Inflammatory cells in faecal exudates or inflammation seen at endoscopy should promote search for an additional invasive pathogen.
Fig. 8.8.10.1 Blastocystis from culture showing binary fission; the cytoplasm is at the periphery. v, vacuole. Phase contrast, ×400. Fig. 8.8.10.3 Blastocystis from culture showing the great variation in size. v, vacuole. Dark field, ×400. Fig. 8.8.10.2 Blastocystis. Electron micrograph showing the peripheral cytoplasm (c) and the central vacuole (v); the inclusions in the cytoplasm are mitochondria. ×5000.

Revision #1
Created 2026-01-22 16:46:05 UTC by Omar Ayman
Updated 2026-01-22 16:46:05 UTC by Omar Ayman