

# Giardiasis

Pages with reference to book, From 286 To 288

Rakhshanda Baqai ( PMRC Research Centre, Jinnah Postgraduate Medical Centre, Karachi. )

*Giardia lamblia* is protozoan parasite which occurs as trophozoites and cysts. The trophozoite is pear shaped 14  $\mu$ m by 7  $\mu$ m while the cysts are 12 by 7  $\mu$ m, oval in shape. Trophozoites are localised in the small gut and the cysts are passed in the faeces. In cases with heavy infection of *Giardia lamblia* or during intestinal hurry trophozoites are also passed in the faeces. They survive in the atmosphere for a very short time. There exists no relation between the number of organisms excreted and the patients symptoms.

*Giardia lamblia* as an intestinal parasite has long been recognized. Van Leeuwenhoek in 1681 recognized a motile organism from his own faeces. Dobell (1920) believed that this motile organism was *Giardia lamblia*.

Transmission of *Giardia* is mostly by orofaecal route. House fly as vector also helps in its spread. Contaminated drinking water is also a great source of infection (Craun et al., 1976; Shaw et al., 1977). Previously *Giardia lamblia* was believed to be non-pathogenic but recent evidence indicates that it is a potential pathogen responsible for diarrhoea and abdominal pain. *Giardia* infection is symptomatic in patients with reduced gastric acidity and after gastric resection (Drasar et al., 1969).

Infection with *Giardia* cyst can cause diarrhoea (Rendtorff, 1954). The incubation period is about 2 weeks. Infection often persists for 6 weeks then spontaneous recovery occurs. The symptoms may vary and patients may have eosinophilia (Welch, 1943), intermittent fever (Overton and Hertko, 1963), failure to thrive, retarded growth and weight loss. Malabsorption or lactose intolerance may persist even after the parasite is eradicated (Wolfe, 1975; Hoskin et al., 1967). *Giardia* may alter the host's absorption of nutrients at the epithelial level by direct physical interference and toxin secretion (Alp and Hislop, 1968).

*Giardia* trophozoites gain access to extra intestinal location both intra and extracellular. Allergic reaction to foods and drugs have also been reported in patients harbouring *giardia* (Batko, 1967). *Giardial* infection also depends upon factors as age (Anand, 1980), infecting dose (Rendtorff, 1954), Protein calorie malnutrition (Gracey, 1973), blood groups especially groups A (Barnes and Kay, 1977), and immunological factors. The host immune system may play a protective and pathogenic role (Stevens, 1982). *Giardia* may present as a severe or chronic disease in individuals having dysgammaglobulinemia (Anient and Rubin, 1972).

Recognition of *Giardia* as an invasive parasite was based on the fact that a systemic antibody response was observed when *giardia* antigen was treated with serum from patients by immunofluorescence technique (Visvesvara et al., 1980; Ridley and Ridley, 1976). *Giardia* antibodies may not signify active giardiasis. They are of restricted distribution and are sometimes present in relatively high titres (Moody et al., 1982).

Levels of immunoglobulins are raised in patients harbouring *giardia* before and after treatment when compared to healthy controls. IgG and IgM does not show any significant alteration but IgA is raised (Brown et al., 1972).

Recent evidence indicates that with the possible exception of IgD, a deviation from the normal level of any immunoglobulin does not explain either the presence of giardiasis or the variability of its clinical features (Jokipii and Jokipii, 1982). Giardiasis is not related to the levels of serum IgE (Brown et al., 1973; McLaughlan et al., 1974). No significant haematological changes are produced in giardiasis (Naik et al., 1982). Specific immunoglobulin may play a role in developing resistance to giardiasis. Secretory IgA concentration in individuals who had giardiasis is significantly lower than the controls (Zinneman and Kaplan, 1972).

Immune response to giardiasis may involve both thymus dependent and independent mechanism (Stevens et al., 1978). Diagnosis of giardiasis cannot be made on the basis of symptoms alone as giardia can result in an array of symptoms. It should be suspected in patients with unexplained chronic diarrhoea, vomiting, malabsorption or lowered levels of jejunal disaccharides. Giardiasis is definitely diagnosed by finding trophozoites or cysts in faeces (Paine and Gluck, 1976; Steele and Mcdermott, 1977). A negative stool does not rule out giardia infection. Three stool samples passed on alternate days should be examined. Concentration method is useful for the cysts but will destroy the trophozoites. If repeated stool examinations are negative then duodenal aspirates should be checked for the organisms. Aspiration of duodenal secretion by enterotest capsule is a common method (Townley et al., 1971). Small bowel biopsies may yield giardia trophozoites when examined after staining with giemsa stain. Mucosal impression smears may also be made (Carswell et al., 1973; Tornkins et al., 1974). Anti giardia antibodies in serum of patients can be detected by indirect immunofluorescence test (Radulescu et al., 1976; Ridley and Ridley, 1976).

Giardiasis can be prevented by observing personal hygiene and thorough cleansing of the hands after defecation. Boiled water should be used for drinking purposes. Water iodination, 2-5 phenol and lysol are also effective as cyst disinfectants (Cerva, 1955). Chlorination of water and the process of sedimentation and filtration is also beneficial (Anand, 1980).

Ideal drug for eradicating *Giardia lamblia* is not yet available. Drugs most commonly used are quinacrin, Metronidazole, Tinidazole and furazolidine. Some organisms may escape contact. Our experience with giardiasis patients at PMRC Research Centre, Karachi has shown that with drug by being in extracellular locations.

Fasigyn 1 G given at night for 3 nights eradicates the parasites followed by Diidoquine thrice daily for 7 days and Tetracycline 250 mg 6 hourly for 5 days prevents secondary infection (unpublished data). As Giardiasis is rampant in our population serological studies should be used to detect asymptomatic and symptomatic individuals. An effective Anti giardia antigen should be developed. Methods of drug testing should be studied in vitro to determine drug resistant strains of giardia. Cross infection between animal and man and the role of rodent in human infection should be determined.

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