

INTRODUCTION

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Ulcerative colitis is a chronic inflammatory bowel disease of unknown etiology. There are fewer reports of ulcerative colitis in the world literature. Most have shown a slightly higher incidence than for Crohn's disease and the incidence has been unchanged over the last few decades (*Yamamoto-Furusho et al., 2003*).

The incidence of ulcerative colitis was 13.9/100 000 per year and for Crohn's disease 8.3/100 000 per year (*Rubin et al., 2000*).

A recent study from Stockholm on 1247 patients diagnosed over a 25-year period shows a pattern similar to that of Crohn's disease. In this study the annual incidence of ulcerative colitis during the 1970 was approximately 1/100 000, during the 1980 approximately 4/100 000, and during the 1990 was approximately 13/100 000. It has been assumed that an increase incidence is caused by the introduction of a new infectious agent to the environment. The opposite is possible; an increase in incidence may be a reflection of an agent disappearing from the environment. Also, the increase may be caused by a decrease in population immunity rather than by an infectious agent. There has been no attempt to demonstrate that one of these hypotheses is more likely than the other (*Nordenvall et al., 1995*).

There are many published series on dietary factors in ulcerative colitis. There is information on sugar and sweets, fruit and fruit juices, cereals, milk edible fat, tea, coffee and alcohol. There is strong evidence associating ulcerative colitis and increased consumption of sugar. The association may, however, be a consequence of the disease rather than a factor in the pathogenesis. Patients may increase their consumption in an attempt to compensate for loss of energy (*Rubin et al., 2000*).

The ulcerative colitis is an inflammatory bowel disease of unknown a etiology, it has an incidence of 13.9 / 100 000 patient Per year. With a male-to-female ratio of 1: 1.5. Although more common in first degree relatives, 90% of patients have no family history. It is primarily a disease of the large bowel (*Rubin et al., 2000*).

Iritis, arthritis, hepatitis, pyoderma gangrenosum are described but these often disappear completely when all of the affected bowel has been removed. In 95% of cases of ulcerative colitis is a diffuse disease (ascending proctocolitis) starting in the rectum and extending proximally for a variable length. The whole colon may become involved, and the so-called backwash through the ileocecal valve can cause ileitis. In 5% of patients the disease is segmental and the rectum is spared. Because of the frequency of the of rectal involvement, sigmoidoscopy is key to the diagnosis. The characteristic histological feature is the formation of crypt abscesses in the depths of the glandular tubules with a surrounding inflammatory infiltrate cryptitis(*Farrell & Peppercorn, 2002*).

The abscesses coalesce to form ulcers which undermine the mucosa and penetrate as far as the muscularis mucosa. The intervening mucosa becomes oedematous and swollen, and in severe cases forms inflammatory pseudopolyps. The bowel wall loses its haustrations and becomes thickened and rigid as a result of muscle hypertrophy. The thickening is less severe than in Crohn's disease and stricture formation is uncommon(*Farrell & Peppercorn, 2002*).

During an acute attack the bowel may become paper-thin and grossly dilated. This complication is believed to be due to destruction of the myenteric nerve plexus. Other complications include perforation,

massive bleeding, anorectal suppuration, and carcinoma of the colon. (*Farrell & Peppercorn, 2002*).

Ulcerative colitis usually presents with attacks of diarrhea and passage of blood and mucus per rectum. With extensive disease these symptoms are severe, the patient passing up to 10-15 motions daily. Urgency to defecate can be incapacitating. Severe attacks may be associated with abdominal pain, tenderness and pyrexia. Most attacks are mild, and patients with rectal bleeding may initially come to the surgeon with a request to assess and treat haemorrhoids. Rectal examination should include careful inspection for anal complications as fissure and fistula. The rectal mucosa may feel thickened and boggy (*Miller & Windosr, 2000*).

On sigmoidoscopy the mucosa is red, granular, and there may be punctuate haemorrhages or spontaneous bleeding. Contact bleeding, produced by gently rubbing the mucosa with a gauze pledged, is a typical feature. A barium enema is of value in assessing the extent of the disease but is contraindicated in the acute phase because of the danger of perforation. Typical changes include loss of haustrations and reduction in the caliber of the bowel, irregular fluffy outline of the mucosa, pseudopolyps and, rarely, strictures (*Cappell & Friedel, 2002*).

The disease must be differentiated from amoebic colitis, Crohn's disease and, in the elderly, ischaemic colitis. The course of ulcerative colitis is characterized by relapses and remissions but a chronic continuous form is also described. In some patients the initial attack is fulminating, and toxic dilatation of the colon with exacerbation of systemic and abdominal symptoms may occur at any time (*Holtmann & Galle, 2004*).

Initial treatment of an attack of ulcerative colitis relies on medical measures. With fluid replacement, correction of anaemia, adequate nutrition and steroid therapy, 97% of patients survive their initial attack, but 70% will develop further attacks. Long-term treatment with sulfasalazine (0.5–1 g orally every 6 hours) has been shown to reduce the incidence of relapse. Topical steroids (administered as suppositories or prednisone retention enemas) will usually control mild attacks, but systemic steroids (prednisone 10-15mg orally every 6 hours) are needed during an acute relapse. 15% of patients eventually require surgery, which may be elective or emergency. It has been calculated that 1 in 50 patients with mild proctitis, 1 in 20 with moderate colitis and 1 in 3 with extensive disease will come to surgery (*Thuraisingam & Leiper, 2003*).