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CA 125 – ACTIVITY MARKER IN ABDOMINAL TUBERCULOSIS

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INTRODUCTION

CA 125 is a mucin-like glycoprotein found on normal tissue derived from coelomic epithelium and expressed in most epithelial ovarian tumors. CA 125 serum levels are widely used to monitor regression or progression of tumor in patients undergoing treatment for ovarian carcinoma. In the decade since the development of CA 125, conditions other than ovarian carcinoma have been reported to cause elevation of this marker. In 1981, Bast and colleagues detected CA 125, which is a cell surface antigen that was detectable on endocervix, ovaries, tubes, pleura, peritoneal linings endometrium and foetal mullerian lines.[1,2]

Ttuberculosis is associated with poverty, deprivation, and human immunodeficiency virus infection. Abdominal tuberculosis has an insidious onset with a myriad of symptoms and signs. The diagnosis is challenging as many patients do not have evidence of pulmonary tuberculosis or a positive skin test. India has claimed to have 26% of the world's cases of tuberculosis and 37% of the deaths. The worldwide incidence of abdominal tuberculosis is increasing in parallel with other forms of the disease. In India tuberculosis isresponsible for 7% of hospital admissions for intestinal obstruction and 6% of perforations.[3]

MATERIALS AND METHODS

Ca 125 was evaluated in 23 patients with non malignant ascites after meeting the inclusion and exclusion criteria set for the study, whose ascitic fluid analysis were negative for malignant cells. Patients currently, are either being treated for pulmonary tuberculosis, old case or defaulters. Most presented with abdominal distension, tense ascites, loss of appetite and weight loss. ESR &mantoux test along with ultrasonography of abdomen pelvis. Computed tomography of the abdomen were performed as and when indicated. Serum ADA level above 54U/l, ascitic fluid ADA level above 36 U/l and a ascitic fluid to serum ADA ratio >0.985 were found suggestive of tuberculosis and was correlated with CA125 levels. The patients were initiated on anti-tubercular treatment after tissue diagnosis and called for serial follow-up to assess treatment response. CA125 levels were measured at 1, 2 and 6th months of initiating ATT.

RESULTS

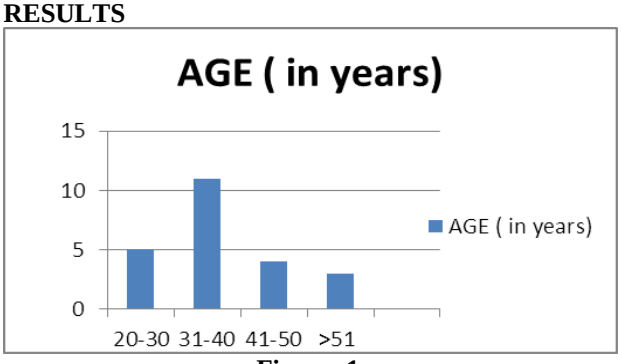

Figure. 1.

The chart clearly shows the age distribution in our study with the maximum number of cases between the third and fourth decade of life, with the mean age being 33 years.

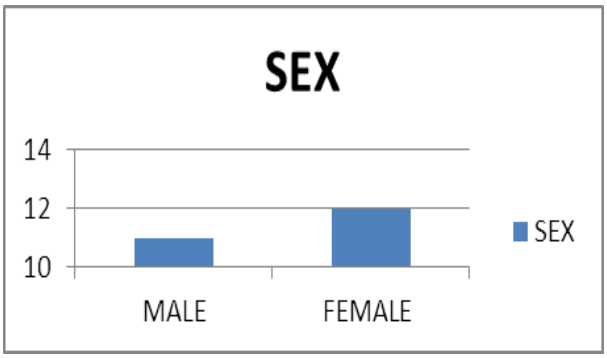

Figure. 2.

In our study males and females are both affected equally.

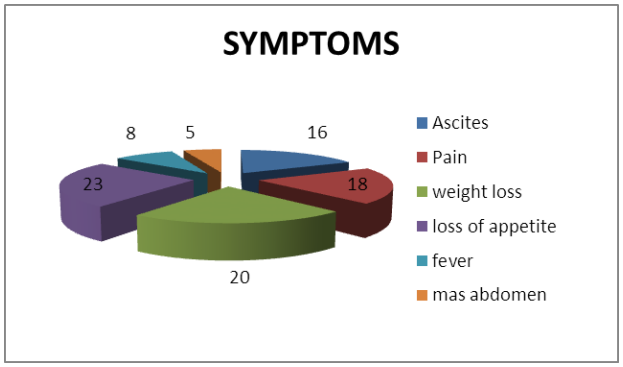

Figure. 3.

The pie chart depicts the symptoms associated with abdominal tuberculosis with loss of appetite virtually present in all the patients, associated with weight loss. Other major symptoms were colicky pain abdomen and Ascites seen in 69% and 78% respectively. Fever was seldom complained, seen in only 34.7% of the patients in our study.

CA 125

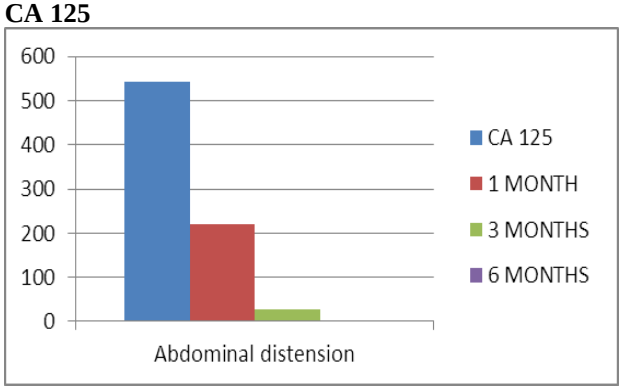

Figure. 4.

The line graph clearly shows the mean values of CA-125 at the time of diagnosis and follow up at 1,3 and 6 months of starting ATT under RNTCP. CA-125 levels were within normal limits at the end of 3 months of starting ATT and undetectable in the serum at the end of 6 months.

DISCUSSION

Mean age of Incidence of abdominal TB was found to be 31 years, with 63% of cases between 20 and 40 years old and a male/female ratio of 1:2.[1] Abdominal tuberculosis is predominantly a disease of young adults. Abdominal tuberculosis includes infection of the gastrointestinal (GI) tract, peritoneum, mesentery, abdominal lymph nodes, liver, spleen, and pancreas. The most common forms being tuberculous peritonitis and lymphadenopathy, small bowel and ileocecal disease,whereasunsual forms being esophageal, gastroduodenal, colonic, and anorectal involvement. Disease within any part of the GI tract itself has two main forms: (1) an ulcerative process that may bleed, perforate, or form fistulas; and (2) a hyperplastic reaction that may cause obstruction or present as a mass.

Classic histologic findings are caseating granuloma, central necrosis with a peripheral area of lymphocytes, plasma cells, and Langhansgiant cells. Secondary intestinal tuberculosis is caused by ingestion of infected sputum. In both primary and secondary intestinal tuberculosis bacteria are found in the mucosa and lymphoid tissue (Peyer's patches), stimulating an inflammatory reaction predominantly in the submucosa. There is lymphatic spread to local nodes. Endarteritis produces edemaand transverse ulceration. A fibroblastic reaction produces thickening of the bowel wall, subsequent involvement of regional nodes and lymphatic obstruction, a large inflammatory mass may form. Mesenteric lymph nodes and the peritoneum are infected during the primary bacteremic phase of pulmonary tuberculosis. Reactivation of this latent infection produces clinical disease, often in the absence of active lung disease. Hematogenous spread to abdominal nodes and peritoneum also occurs during active pulmonary ormiliary disease. Infection can also reach the peritoneum by direct spread from the fallopian tube, ruptured abdominal lymph nodes, or affected regions of the GI tract. Direct spread is uncommon.

The most common site of involvement is the ileocaecal region, possibly because of the increased physiological stasis, increased rate of fluid and electrolyte absorption, minimal digestive activity and an abundance of lymphoid tissue at this site. It has been shown that the M cells associated with Peyer's patches can phagocytose BCG bacillis.[4] The frequency of bowel involvement declines as one proceeds both proximally and distally from the ileocaecal region. Peritoneal involvement may occur from spread from lymph nodes, intestinal lesions or from tubercular salpingitis in women. Abdominal lymph nodal and peritoneal tuberculosis may occur without gastrointestinal involvement in about one third of the cases.[5] Granulomas are often seen just beneath the ulcer bed, mainly in the submucosal layer. Submucosal oedema or widening is inconspicuous. Tubercular ulcers are relatively superficial and usually do not penetrate beyond the muscularis.[6] They may be single or multiple, and the intervening mucosa is usually uninvolved. These ulcers are usually transversely oriented in contrast to Crohn's disease where the ulcers are longitudinal or serpiginous.[7]

Adenosine deaminase (ADA) is an aminohydrolase that converts adenosine to inosine and is thus involved in the catabolism of purine bases. The enzyme activity is more in T than in B lymphocytes, and is proportional to the degree of T cell differentiation. ADA is increased in tuberculous ascitic fluid due to the stimulation of T-cells by mycobacterial antigens. ADA levels were determined in the ascitic fluid of 49 patients by Dwivedi*et al.*[8] The levels in tuberculous ascitis were significantly higher than those in cirrhotic or malignant ascitis. Taking a cut

off level of 33 U/l, the sensitivity, specificity and diagnostic accuracy were 100, 97 and 98 per cent respectively.[9] In the study by Bhargava *et al*, serum ADA level above 54U/l, ascitic fluid ADA level above 36 U/l and a ascitic fluid to serum ADA ratio >0.985 were found suggestive of tuberculosis.[10]

The CA 125 antigen test was developed as a serum tumor marker to correlate with disease activity in patients with ovarian carcinoma. Among the nonmalignantgynecologic causes of serum CA 125 elevations, menstruation in healthy women can increase the levels twofold to threefold.[11] CA125 may be valuable in not only the diagnosis and monitoring of disease activity in ovarian carcinoma, but also in peritoneal disease activity with other neoplastic or inflammatory states. The patients in the current study had ascitic and/or plastic form of tuberculous peritonitis, with free fluid in addition to advanced adhesive disease of the bowels and omentum. As *Bergmann et al*. postulated, CA 125 could be used tofollow disease activity associated with ascites.[12] The decline in the CA 125 level may have indicated improvement in the inflammatory process. Our study illustrates that despite its limited specificity, the high sensitivity of CA 125 allows this tumor marker to be used in the monitoring of disease activity and treatment response in abdominal tuberculosis.

CONCLUSION

Results of our study suggest that serum CA-125 levels in patients with tuberculous peritonitis are as high. By the end of the third month of antituberculous therapy, serum CA-125 levels have returned to normal. We, therefore, suggest that serum CA-125 can be used to evaluate the efficacy of therapy in tuberculous peritonitis.

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