CASE REPORT PROF-072

SCLEROSING PERITONITIS:
A REPORT OF 4 CASES

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SUMMARY

We present 4 cases of a rare disease of sclerosing peritonitis treated by us in last 15 years. Tuberculosis was found out to be the cause in two but no specific cause could be ascertained in other two cases. All were treated by laparotomy, Excision and removal of the thick plastic membrane encasing the whole small intestine. All had perfect recovery without any complication and recurrence.

KEY WORDS: Sclerosing peritonitis- cause and treatment.

INTRODUCTION

Sclerosing peritonitis is a rare disease. A dense white thick membrane encases the constricted, markedly shortened small intestine bundled like a cocoon. It is also called peritoneal fibrosis syndrome and sclerosing encapsulating peritonitis[1]. The bowel is constricted circumferentially and longitudinally. The loops are adherent to each other convoluted and kinked so that the whole of the effected bowel is tightly bound in a mass. The mesentary may also be covered by the thickened tissue which is somewhat shortened. Greater omentum may be opaque, thickened and contracted. Stomach, duodenum, liver, gall bladder, transverse colon, sigmoid colon, caecum and appendix may be covered completely or partially by thick sheet of the opaque membrane in some cases. The pelvic organs may also be covered by thick membrane some times but posterior and antereo-lateral parietal peritoneum is usually spared. There is a space between parietal and visceral peritoneum of the abdomen but loose adhesions may be present between these two.

The thick membrane superficially is formed of a compact fibrinous exudate deep to the

mesothelium. There is a thick lamellar collagen bundle layer 0.5-4 mm thick which forms the bulk of the membrane present. There is a chronic inflammatory exudate consisting of lymphocytes and macrophages in the areolar tissue between the fibrous layer and underlying viscera. Most of the tissue are avascular but may have certain areas of moderate vascularity. It may also get calicified but the underlying viscera and tissues are always normal[2].

It may be due to a non specific reaction of the peritoneal surface to a variety of insults. Any agent that causes irritation of the mesothelial layer and induces serositis resulting into mesothelial loss predisposes the peritonium to fibroneogenesis. It may be characterized by florid reactive hyperplasia of mesothelial mesenchymal cells[3].

It has been a common complication in continuous ambulatory peritoneal dialysis (CAPD) which may be due to organic compounds from plastic bag and tubing[4] or due to the disinfectents used for the peritoneal catheters like formalin[5] and chlorhexidine in alcohal[6] or it may be due to dialysate glucose[1]. Practalol, a beta-blocker has been found to be the cause of this syndrome[7,8,9,10].

It may give rise to haemoperitonium[11] or ischaemic intestinal necrosis[12] besides intestinal obstruction. There are a few investigations which can help to diagnose this disease. Plain X-ray may show some gas fluid levels. Ultrasound scan may show increased small bowel peristalsis, tethering of the bowel to the posterior abdominal wall, intra peritoneal echagenic strands and membrane formation[13]. It can also be diagnosed by positive emission tomogram (PET) using f.19=8 DG (fluorodeoxyglucose) by mass/ muscle radioactivity[14].

Conservative treatment may be tried by immunosuppressive drugs like[15] azothioprine, cyclosporine and predsinolone but treatment of choice is surgery i.e. removal of encapsuling membrane and release of the small intestine from the sac. We are presenting four cases of sclerosing peritonitis treated by us in 17 years.

CASES

All four cases treated from 1979-1994 had almost the same typical history (Table 1). Age ranged between 13-20 years, two male and two were female.

They had intermittent colicky central abdominal pain alongwith vomitting and absolute constipation which lasted for 3-4 days and then had remission for 1-2 weeks. The duration ranged from 2 months to 4 months. The constant feature had been a definite slightly tender mass extending from umbilical region upto the hypogastrium. The mass has been firm, non mobile and non resonant. None of them had any history of any specific medication except one was taking ATT.

Blood examination was almost normal in all of them, X-ray chest did not reveal any abnormality and plain x-ray abdomen showed two fluid levels in one patient.

Three cases were diagnosed as appendicular mass where as one was diagnosed as acute or chronic intestinal obstruction. Laparotomy was under taken for all the cases

Membrane and glands from mesentry was sent for histological examination in all the cases. Two had non specific finding in the glands and two had typical tubercular lesion. The patients were followed up for one month to two years and no recurrence was recorded. The patients with the positive tubercular histology were given full antitubercular medication.

Table 1. Patients Data.

S.No	Sex/Age	Year	Clinical Feature	Finding	Treatment	Cause
1	M/20	1977	Recurrent pain abdomen 3 months vomitting, absolute constipation, lump hypogastrium	Only small intestine involved	Multiple incision & excision of membrane release of small intestinal loop.	Idiopathic
2	F/18	1979	Recurrent pain abdomen 2 months vomitting, absolute constipation, lump hypogastrium	Only small intestine involved	Multiple incision & excision of membrane release of small intestinal loop.	T.B.
3	M/14	1993	Recurrent pain abdomen 4 months vomitting, absolute constipation, lump hypogastrium	Only small intestine involved	Multiple incision & excision of membrane release of small intestinal loop.	T.B.
4	F/17	1994	Recurrent pain abdomen 3 months vomitting, absolute constipation, lump hypogastrium	Small intestine, stomach, transvers colon, greater omentum, liver and gallbladder involved.	Multiple incision & excision of membrane release of small intestinal loop.	Idiopathic

DISCUSSION

Sclerosing peritonitis is a rare disease least expected in the surgical practice. Rarely it is diagnosed before surgery. More cases are reported after the introduction of a beta blocker practolol and also vast usage of C.A.P.D. otherwise, this syndrome is rare and its cause is unknown[16,17,18] as no bacteria or tumour cells have been isolated from any the cases reported so far. Perhaps it occurs as a part of some systemic disorders[19].

Out of our four cases two were idiopathic but other two had tubercular lesion in the mesenteric glands proved on histopathology. One was taking antituberculous drugs for one month before operation. But antitubercular drugs could possibly not the cause of the sclerosing peritonitis as that patient was put on the same regimen after the operation i.e. refampicin isoniazid, pyrazinamide and myambutal, and followed for one year. Patient improved and there was no recurrence of this syndrome, so we believe that tuberculosis could be one of the causes of sclerosing peritonitis.

All were opened by right paramedian incision. There was a clear space between parietal and visceral peritoneum. In three patients, the entire small intestine was encased in a thick, white and opaque membrane whole looking like a cocoon. In last case there was an additional cocoon bag lying transverse superiorly containing transverse colon, greater omentum and stomach. In the same case liver and gall bladder were also covered by the thick membrane so were the pelvic organs except uterus, tubes and overies. Mesentry of the ileum was also involved in all cases and mesenteric glands were moderately enlarged in all, two turned out to be tuberculosing on histolgical examination.

The thick covering was incised and removed by sharp and blunt dissection. No sooner the thick membrane was incised the small intestine loops buldged forcibly outwards. After a long and tedious dissection the whole thick membrane of the cocoon was removed fairly easily and underlying bowel wall looked entirely normal. Thickened mesenteric vessels and thick tissue was bloodless so no much bleeding took place.

One case had a small perforation of the ileum during dissection for which primary closure was

done. In all patients the abdomen was closed without any drain and all had uneventful recovery without any complication. Gentimycin alongwith metronidazole were used a prophylactic antibiotics for all of them.

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