PRIMARY PERITONITIS

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INTRODUCTION

The regularly recurring problem of acute abdominal pain in children that faces the general surgeon, has prompted a brief consideration of one of the causes, which, on account of its rarity, is apt to be overlooked.

There is some lack of clarity about what constitutes primary peritonitis. Fraser and McCartney[1] regarded primary peritonitis as peritonitis in which there was no clinical focus of infection elsewhere, while Gross[2] referred to 'primary' or 'idiopathic' peritonitis as 'having no focus of infection within the peritoneal cavity, and which in the vast majority of cases probably arises from a bacteraemia'. Maingot[3] likewise confined the term 'primary' to those cases of acute peritonitis in which no obvious intra-abdominal cause could be found. This definition probably covers the generally accepted concept of the condition. As pointed out by Fowler[4] those cases with obvious primary foci of infection, such as pneumonia or erysipelas, carrying a mortality of their own, should be excluded;

this applies also to the primary peritonitis complicating septicaemia in the newborn.

AETIOLOGY

The causative organisms are the pneumococcus and the haemolytic streptococcus in over 90% of cases, judging by the Boston Children's Hospital series of 158 cases,[2] and the Royal Children's Hospital, Melbourne, series of 97 cases.[4] The paths of invasion are either by haematogenous spread or by direct extension *via* the female genital tract. Although it is taught that the female genital tract is an accepted path of invasion, Gross pointed out that this concept fails to explain the mode of infection in males and, furthermore, that examination of the genital tract at autopsy in females in his series who died has never shown evidence of ascending genital-tract infection.

INCIDENCE

Primary peritonitis is an uncommon condition. Barrington-Ward,[5] writing in 1928, quoted a series of 32 cases over a period of 10 years at the Hospital for Sick Children, Great Ormond Street, while Ladd *et al.*[6] in 1939 could refer to 67 cases during the preceding 10 years at the Children's Hospital, Boston. Gross[2] based his experience on a total of 158 cases from the same institution; the first 120 cases up to 1940, and a subsequent 38 cases from 1940 to 1950.

Fowler,[4] writing in 1957, reviewed 97 cases at the Royal Children's Hospital, Melbourne, over a period of 30 years, and assessed the frequency at that hospital as between 1 and 2% of all abdominal emergencies in children under 14 years of age, which is in keeping with the findings of Fraser and McCartney,[1] who quoted an incidence of 2%.

At the Red Cross War Memorial Children's Hospital, Cape Town,[7] there have been 11 cases of primary peritonitis from June 1956 to March 1962.

CLINICAL FEATURES AND DIAGNOSIS

Classical descriptions of the disease are to be found in the writings of Barrington-Ward,[5] Ladd[6] and Gross.[2] The picture is that of a severe illness in a child, of acute onset, with fever, abdominal pain, nausea and vomiting. There is often a history of a preceding upper-respiratory-tract infection.

In contrast to acute appendicitis, primary peritonitis is seen as frequently in the 1 - 2 year age-period as in later age groups. Diarrhoea is a common accompaniment. In females there may be a vaginal discharge; Ladd *et al.* found this sign in 4 of the 34 girls in their series.[6] The temperature is strikingly raised with a corresponding rise in pulse rate. The abdomen is diffusely tender, with involuntary rigidity which may be board-like, and distension of varying degree is present. In infants the abdomen may present a doughy feel, and on rectal examination there is diffuse tenderness. The leucocyte count is raised to 20 - 50,000 per c.mm. with 80% polymorphonuclear cells. Ladd *et al.*[6] were of the opinion that 'the condition could usually be distinguished from secondary peritonitis' and in their series the correct diagnosis was made in 64% of patients. As pointed out below, this figure is misleading.

Differentiation from Appendicitis

The clinical points stressed in distinguishing primary peritonitis from secondary peritonitis caused by a perforated appendix (which constitutes the real practical problem in diagnosis) are by no means reliable. A preceding upper-respiratory infection is a very common accompaniment of acute non-specific mesenteric adenitis, and it may also be encountered in acute appendicitis in children.

Though uncommon, appendicitis does occur below the age of 2 years,[8,9] so that age *per se* is of little help in arriving at a diagnosis. Likewise, the presence of diarrhoea is not a reliable clinical finding; it is present in about 12% of children with acute appendicitis, when the organ is situated behind the lower ileum or in the pelvis.[10] The child is more acutely ill *from the onset* with primary peritonitis than with appendicitis. This is an important and useful clinical point if a reliable history is obtainable, but, here again, the rapidity with which appendicitis in a young child may progress to perforation and peritonitis should be kept in mind.

Despite a knowledge of the condition and its clinical presentation, it is the exception rather than the rule to be able to arrive at a confident clinical diagnosis and to treat the patient conservatively. The correct diagnoses in 64% of the series of Ladd *et al.* were all *'confirmed by laparotomy or abdominal tap with the object of identifying the causative organism with certainty'*.

The difficulties which may be encountered in diagnosis and management are illustrated in the following case report.

CASE REPORT

A Coloured boy, aged 8 years, was admitted to the Somerset Hospital with a 3-day history of acute illness with generalized abdominal pain and vomiting. No information was available about the mode of onset. The child was ill, with a temperature of 101·8° F. and a pulse-rate proportionately raised.

The abdomen was slightly distended, and diffuse generalized tenderness was present. The leucocyte count was 20,000 per c.mm. with a preponderance of polymorphonuclear cells. A diagnosis of acute appendicitis was made and the abdomen was opened through a McBurney muscle-splitting incision. The findings were as follows:

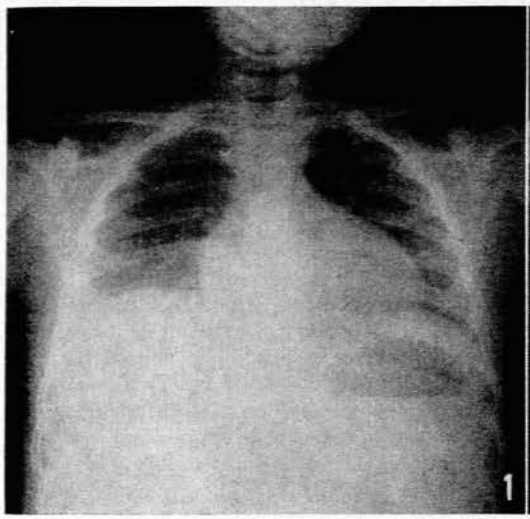
The appendix was normal despite a hyperaemia of the serous coat which was also present in the small bowel. A moderate amount of free fluid was present which was *clear, straw-coloured, non-purulent and odourless*. Numerous enlarged mesenteric lymph nodes were visible and palpable. There was no Meckel's diverticulum and the whole of the small bowel appeared normal.

The following possibilities were considered:

1. Acute non-specific mesenteric adenitis.
2. Acute tuberculous peritonitis.
3. Primary (idiopathic) peritonitis. However, the absence of a purulent or even semi-purulent exudate at this stage of the illness seemed to be against this diagnosis.

A lymph node was taken for histological examination and fluid was collected for bacteriological examination. Since at this stage it seemed that nothing could be gained by drainage, the abdomen was closed without a drain. The appendix was not removed.

It became clear during the early postoperative course that neither non-specific mesenteric lymphadenitis nor acute tuberculous peritonitis could account for the clinical picture. The child became more ill and toxic, the pyrexia increased and showed a 'swing', while the abdominal distension became more pronounced. The report on the culture of the fluid collected at operation showed a growth of coagulase-positive *Staphylo-*



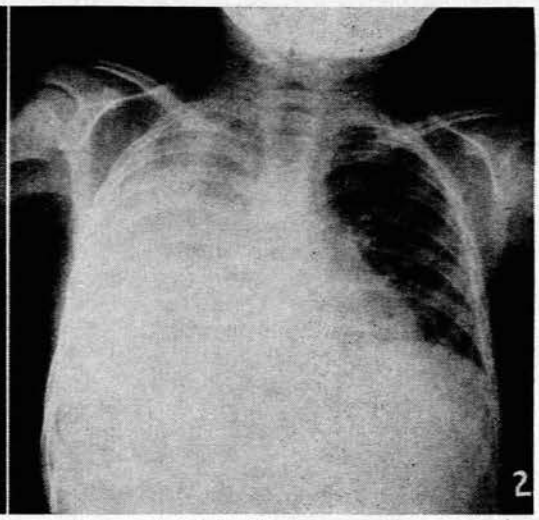


Fig. 1. Chest film taken during the first week of the illness.
Fig. 2. Chest film showing right-sided pneumonia.

coccus pyogenes sensitive to penicillin, chloramphenicol and the tetracyclines.

Despite 48 hours of appropriate and intensive antibiotic therapy, however, the child remained extremely ill and toxic with a swinging temperature and a high leucocyte count. It was therefore decided to reopen the abdomen. A small right paramedian para-umbilical incision was made and profuse frank pus was found to be present in the peritoneal cavity.

The liver edge was palpable, but there was no localized swelling to suggest a possible liver abscess. As much pus as possible was evacuated by suction, and drainage was instituted in the right flank and pelvis.

A specimen of pus was taken for examination. Blood culture showed a growth of coagulase-positive *Staphylococcus pyogenes* sensitive to penicillin and the broad-spectrum antibiotics, and the report on the pus collected at the second operation showed that the same antibiotic-sensitive organism was present in the peritoneal cavity.

The postoperative course was further complicated by a staphylococcal pneumonia (Fig. 2), the sputum examination showing the organism to be identical with that previously cultured, but there was a gradual improvement from the 10th day onwards that progressed to complete recovery.

DISCUSSION

In retrospect the following points are thought worthy of consideration:

1. This was a primary peritonitis, by definition, 'in which no obvious intra-abdominal cause could be found'. The primary focus in this case remains an enigma; the staphylococcal pneumonia was not the primary manifestation as shown by comparison of early and later chest films (Figs. 1 and 2). The presence of a septicaemia recalls to mind Gross' concept, i.e. that the vast majority of cases probably arise as a 'bacteraemia'.

2. The organism isolated is a most unusual cause of primary peritonitis. None of the cases in Gross' series of 158 could be attributed to the staphylococcus, while in Fowler's series of 97 there was only 1. Among the 11 cases at the Red Cross War Memorial Children's Hospital, Cape Town, 1 was shown to have been caused by *Staphylococcus aureus.*

3. The uncertainty of diagnosis, even at operation, is noteworthy. At the time of the operation, acute non-specific mesenteric lymphadenitis could be legitimately considered in the differential diagnosis, especially in view of the clear exudate and enlarged mesenteric nodes. The raised leucocyte count need be no bar to this diagnosis. Aird[11] commented on the frequent finding of a leucocytosis of 15 - 20,000 per c.mm. in acute non-specific mesenteric adenitis, with 80% polymorphonuclear cells.

4. The presence of clear non-purulent fluid exudate was a disconcerting finding at operation, but this must be accepted as occurring at any rate before the 4th day of the disease in a staphylococcal primary peritonitis.

5. The decision whether or not to perform appendicectomy is a difficult one. On the basis of first principles the answer clearly must be 'no'; removal of a normal organ in a sick child must constitute meddlesome surgery. Gross leaves us in no doubt concerning his opinion — 'appendicectomy is to be condemned' and, he believes, increases the mortality rate. Quite the opposite view is taken by Fowler[4] who states: 'When a right iliac muscle-splitting incision is employed there is then an obligation to remove the appendix, a step which carries no special risk'. His opinion is based on a series of 97 patients of whom 50 underwent appendicectomy. Of these, 29 were in the

TABLE I. A SUMMARY OF SURGICAL PROCEDURES AND RESULTS IN THE PRE-ANTIBIOTIC AND ANTIBIOTIC ERAS*

Surgical procedures	*1926/1939*		*1939/1955*	
	No. of cases	*Deaths*	*No. of cases*	*Deaths*
Laparotomy alone, by lower paramedian incision:				
(*a*) with peritoneal drainage ..	8	3	4	—
(*b*) without drainage	2	1	7	—
Laparotomy and appendicectomy by lower paramedian incision:				
(*a*) with peritoneal drainage ..	2	1	—	—
(*b*) without drainage	1	—	—	—
Appendicectomy through a right iliac muscle-splitting incision:				
(*a*) with peritoneal drainage ..	7	2	2	—
(*b*) without drainage	11	5	27	—
Drainage of presenting intraperitoneal abscess	2	—	—	—
Right iliac muscle-splitting incision without appendicectomy and with peritoneal drainage	—	—	1	—
Abdominal paracentesis	3	3	5	1
Nil	9	9	6	5
Total	45	24	52	6

*From Fowler, R.[4]

(1939 - 1955) chemotherapy and antibiotic era and the mortality was nil, while 21 occurred in the 1926 - 1939 period, of whom 8 died. This high mortality, however, as can be seen from his figures in Table I, cannot be ascribed to removal of the appendix.

CONCLUSION

Primary peritonitis, though an uncommon condition, must be kept in mind when dealing with the problem of acute abdominal pain in children, especially when the very high mortality, even with the use of modern antibiotic therapy, is considered (Table II).

TABLE II. FIVE-YEAR INCIDENCE AND MORTALITY OF PRIMARY PERITONITIS AT THE ROYAL CHILDREN'S HOSPITAL, MELBOURNE, 1926/1955*

Period	*Number of cases*	*Deaths*	
		No.	*%*
1926/1930	13	7	53·8
1931/1935	14	6	42·9
1936/1940	23	10	43·5
1941/1945	2	1	50·0
1946/1950	14	1	7·1
1951/1955	31	5	16·1
Total	97	30	30·9

*From Fowler, R.[4]

Awareness of the condition makes a provisional clinical diagnosis possible, and this must then be confirmed at operation. This should take the form of a limited laparotomy through a paramedian incision, and confirmation of the presence of a normal appendix and a non-offensive exudate. No more need then be done than the collection of pus for bacteriological examination, and drainage. The efficacy of drainage is debatable, but in the presence of frank profuse pus it would seem more than justifiable. The appendix is not removed.

When, however, a right iliac muscle-splitting incision has been made under the misapprehension that one is dealing with acute appendicitis, I tend to agree with Fowler[4] that appendicectomy should be done in view of the real danger subsequently of acute appendicitis being missed because of the presence of an appendicectomy scar.

SUMMARY

1. A case of primary peritonitis caused by *Staphylococcus pyogenes aureus* is described.

2. The incidence and aetiology of primary peritonitis is briefly considered.

3. Problems in diagnosis and management are discussed with particular reference to the question of removal of the appendix.

4. The importance of bacteriological examination for identification of the causative organism and its sensitivity to antibiotics is stressed.

5. It is pointed out that primary peritonitis carries a surprisingly high mortality — considerably higher than that of acute appendicitis.

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