1 and 2 litres of urine with a specific gravity of 1012–1018 during the period of hyperelectrolytaemia, did not suggest dehydration. Study of the blood volume showed 70 ml. of blood per kg. of body weight. This figure would appear to exclude severe dehydration.

Other Features

Two other features deserve mention. In the present case, as in other cases, the electrolytes in the blood returned to normal levels, although the urinary content of sodium chloride remained very low. This observation suggests that in addition to salt retention there is an internal shift of electrolytes in this condition. Allott (1957) also inferred that a redistribution within the body is the likely explanation.

The other point worthy of note in our case was the presence of numerous bullae on the body surface. McLardy (1950) considered these bullae to be evidence of a trophic change consequent on cerebral damage. Our patient thus exhibited two distinct manifestations of his brain injury—the trophic lesions and the biochemical changes. In none of McLardy's cases did the lesions appear within two months of the brain injury. In only one case did both uraemia and the trophic changes coexist, and this was in a patient with bilateral involvement of the orbital cortex.

Conclusion

In attempting to discover a mechanism responsible for hyperelectrolytaemia provoked by cerebral dysfunction, it would seem advisable to bear in mind Selkurt's (1954) warning that "in view of the multiplicity of factors concerned with water and electrolyte balance that may be disturbed under the above circumstances, attempts to draw discrete conclusions from clinical observations are extremely hazardous." However, this type of case is not rare, and a planned approach, directed particularly at elucidating renal function, should result in progress in the understanding of this most interesting problem.

Summary

A case of hyperelectrolytaemia following a brain injury is described. Uraemia appears to be an integral part of the biochemical upset.

Evidence is produced that severe dehydration is not responsible for this disturbance, nor is diabetes insipidus thought to be a likely causative factor.

It is pointed out that renal function has not been sufficiently studied to enable an assessment to be made of the role of the kidneys in the syndrome.

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APPENDICITIS AND ACUTE TERMINAL ILEITIS DURING AN OUTBREAK OF GASTRO-ENTERITIS AMONG BRITISH TROOPS IN PORT SAID

BY

D. J. COWAN, F.R.C.S.

Major, R.A.M.C.

During the period of occupation of Port Said from November 5 to December 18, 1956, of the 919 British troops who were admitted to medical units 365 had acute gastro-enteritis. Of the 29 patients admitted with suspected acute appendicitis 14 were subjected to laparotomy: 10 of these had acute suppurative appendicitis and three had acute terminal ileitis. The operations were in every case performed by me. Clinical details of the cases of terminal ileitis are presented.

All patients were young men living under similar conditions, the majority in vacated buildings in Port Said. In view of the damage to water mains and sewage, tap-water was unsafe for drinking, and all water supplied to troops was sterilized, either in bulk or by individual sterilizing tablets (halazone). During the early weeks of the occupation tinned "compo" rations were consumed.

The incidence of gastro-enteritis was higher in units where living conditions and hygiene were below the general standard, flies being particularly prevalent in such areas. In ships anchored in the harbour, where there were few flies, gastro-enteritis was uncommon.

Clinical Description

There appeared to be two different groups of patients with gastro-enteritis. *Group 1.*—A small group of patients presented with the typical picture of bacillary dysentery and had profuse diarrhoea with blood and mucus in the stools; most of these cases occurred in one unit in a forward position. *Group 2.*—The majority of cases occurred among troops stationed in Port Said itself. These presented with high sustained fever, frontal headache, and occasionally neck rigidity. This was followed by severe abdominal colic and profuse diarrhoea 12 to 24 hours later. Few of these cases had blood or mucus in their stools. Recovery usually occurred after four or five days' illness, and relapses were rare. Skin rashes were not observed, nor did rheumatic symptoms occur. Dengue and sandfly fever were excluded on clinical grounds.

Treatment.—All cases received sulphonamides in some form or other, so that it is impossible to predict whether they would have recovered equally quickly without treatment. It was the general impression, however, that sulphonamides had little effect on the course of the disease of the patients in group 2.

Pathology.—Unfortunately only very limited pathological facilities were available. No cultures or agglutination reactions could be performed. Bacillary cellular exudate was found in the stools of patients in group 1 only. Moderate leucocytosis was the rule, with a preponderance of neutrophil granulocytes. Blood slides taken to exclude malaria were consistently negative.

Incidence of Appendicitis

About one-third of the patients in group 2 had right-sided abdominal pain and tenderness, simulating appendicular pain, at some time during the course of their illness. Apart from the frequency of this appendicular form of gastro-enteritis occurring in group 2, the incidence of true appendicitis was high during this epidemic.

Crohn (1945) noted a similar high incidence of acute suppurative appendicitis in epidemics of acute gastro-enteritis. This has also been reported by Reimann *et al.* (1945). Quigley and Contratto (1942) described four cases of acute appendicitis in 60 cases of acute gastro-enteritis; while Graham (1944) described four cases of acute gangrenous appendicitis in 500 cases of acute gastro-enteritis occurring in troops.

During the epidemic under review 29 patients were admitted with a presumptive diagnosis of acute appendicitis. In 14 the diagnosis was accepted and they were operated on. Of these, 10 had definite acute inflammation of the appendix, three had acute inflammation of the terminal ileum, and in one case generalized hyperaemia of the small bowel only was found. The remaining 15 patients were considered to have acute gastro-enteritis with appendicular symptoms. All recovered with conservative treatment. Three of them had tender sausage-shaped tumours in the right iliac fossa. The configuration and position of these tumours suggested that these patients had some degree of ileitis. Shifting tenderness was present, although there was well-marked residual tenderness in one patient. Free fluid could not be demonstrated clinically. All three subsequently developed diarrhoea.

Tenderness and spasticity of the sigmoid colon was not uncommon, and in some patients the enlarged tender colon could be palpated.

Differential Diagnosis

The differentiation between the appendicular form of gastro-enteritis, acute appendicitis, and acute terminal ileitis was extremely difficult. Shifting tenderness proved unhelpful; it was not elicited in any of the three cases of acute ileitis which were subjected to operation. Diarrhoea occurred at some stage of the illness in 21 out of 29 patients who were sent to hospital with suspected appendicitis. In 15 this was before or at the time of admission, and in 6 it occurred subsequently. Diarrhoea occurred in 5 out of 10 patients who had acute appendicitis, and in all three cases of acute ileitis. Diarrhoea occurred in 12 of the 15 patients who were not operated on (Table I).

TABLE I

	Diarrhoea on Admis- sion	Diarrhoea in Week Prior to Admis- sion	Diarrhoea in Week following Admis- sion	No. Diarrhoea at any Time	Total
All patients referred with suspected acute appendi- citis	8	7	6	8	29
Those not submitted to operation	7	2	3	3	15
Those operated on .. .	1	5	3	5	14
Acute appendicitis .. .	1	3	1	5	10
Acute ileitis	1	1	1	0	3
Generalized hyperaemia of small bowel only .. .	0	0	1	0	1

Rebound tenderness was present in all three conditions with remarkable constancy. Rovsing's sign was not helpful, being present in only two cases of acute appendicitis. Rectal tenderness was present in six cases of acute gangrenous appendicitis and in one case of acute ileitis. True abdominal rigidity as opposed to guarding or deep tenderness was most reliable and helpful in differentiating the three conditions, and was found only in acute appendicitis (Table II).

Acute Ileitis

Among 14 patients submitted to laparotomy for supposed acute appendicitis were three cases of acute ileitis. This condition was recognized and described by Crohn *et al.* (1932). All three cases corresponded closely with that description.

Case 1

A signalman aged 22 was admitted to hospital on November 26, 1956, suffering from headache, nausea, and vomiting; he had diarrhoea with generalized abdominal pain and pyrexia of 102.6° F. (39.2° C.). He received 24 g. of sulphaguanidine daily.

On November 29 he had pyrexia of 100.4° F. (38° C.), with severe abdominal pain in the right iliac fossa, and a surgical opinion was sought. He was still vomiting occasionally, but was then constipated. When examined he had no pharyngitis or generalized lymphadenopathy; guarding and rebound tenderness was marked in the right iliac fossa, but there was no rigidity of the abdominal wall, nor was rectal tenderness present on digital examination. Shifting tenderness was absent and free fluid could not be demonstrated on clinical examination. The leucocyte count was 15,000 (neutrophil granulocytes 80%, lymphocytes 15%, monocytes 4%, eosinophils 1%).

On November 30 there was no improvement in his condition, and operation was undertaken for a suspected acute appendicitis. At laparotomy some turbid free peritoneal fluid was present. The last 3 in. (7.5 cm.) of the terminal ileum was deeply congested and oedematous, resembling a thick rather perished rubber hosepipe. The involved area was sharply demarcated from the contiguous healthy caecum and small bowel. The lymphatic nodes in the vicinity of the involved ileum and the intermediate and central nodes were all discretely enlarged, firm, and pinkish in colour. The caecum was normal, but there was some peri-appendicular inflammation. The abdomen was closed without removal of the appendix or drainage of the peritoneal cavity.

Post-operatively, gastric aspiration with intravenous fluid and electrolyte replacement was instituted until December 3, when the obstructive element had subsided. After operation 2 g. of streptomycin was given daily by the intramuscular route, supplemented by 2 g. of chloramphenicol daily when oral administration was possible. On December 6 he had recovered enough to be evacuated by air to Cyprus. On December 12 he was completely symptom-free and was evacuated to the United Kingdom.

TABLE II

Patient	Date of Operation	Clinical Findings						Gross Pathology			
		Temperature on Admission		Tenderness or Guarding in R.I.F.	Abdom- inal Rigidity	Rectal Tender- ness	Rov- sing's Sign	Free Peri- toneal Fluid	Appendix	Terminal Ileum	Mesenteric Lymph Nodes
1	Nov. 19	99	37.2	+	0	0	0	+	Normal	Generalized hyperaemia	Normal
2	" 21	100	37.8	+	0	0	0	0	Acute inflammation	Normal	"
3	" 21	100.4	38	+	+	+	0	+	"	"	"
4	" 23	99.8	37.7	+	+	+	0	+	Acute gangrenous inflammation	"	"
5	" 24	98.6	37	+	+	+	0	+	Acute inflammation	"	"
6	" 24	100	37.8	+	0	+	0	0	"	"	"
7	" 24	101	38.3	+	0	+	+	+	Acute gangrenous inflammation	"	"
8	" 26	102	38.9	+	+	0	0	+	Acute inflammation	"	"
9	" 27	100.4	38	+	+	+	+	+	Acute gangrenous inflammation. Perforated	"	"
10	" 30	101.8	38.8	+	0	0	0	Purulent ++ Turbid ++	Peri-appendicular inflammation	Acutely inflamed	Acutely inflamed
11	Dec. 1	100	37.8	+	+	0	0	++	Acute inflammation	Normal	Normal
12	" 1	102.6	39.2	+	0	+	0	++	Normal	Acutely inflamed	Acutely inflamed
13	" 2	101	38.3	+	0	0	0	++	"	"	"
14	" 5	99.8	37.7	+	+	0	0	+	Acute inflammation	Normal	Normal

Case 2

A gunner aged 19 was admitted to hospital on December 1, 1956, with central abdominal pain which he had had for three days. One week prior to this he had had a brief attack of diarrhoea. Six months previously he had suffered a similar attack of pain, which had subsided without treatment. He had neither nausea nor vomiting, and was afebrile. When examined he was found to have marked tenderness and guarding in the right iliac fossa. Shifting tenderness and free fluid were absent on clinical examination. Rectal tenderness on digital examination was pronounced. A provisional diagnosis of acute appendicitis was made. The leucocyte count was 16,000 (neutrophil granulocytes 75%, lymphocytes 16%, monocytes 6%, eosinophils 2%, basophils 1%).

Operation was carried out on December 1. A small amount of clear free peritoneal fluid was present. The appendix and caecum were high and bound down by dense adhesions. Only after an adherent but uninflamed appendix had been removed and the caecum mobilized was the condition of the ileum discovered. The terminal 2 in. (5 cm.) was deeply congested and oedematous, with a sharp line of demarcation separating it from the healthy ileum. The inflammatory process extended up to the ileo-caecal junction but did not involve the caecum. The mesenteric lymph nodes were discretely enlarged and congested. The abdomen was closed without drainage. Post-operatively the patient began to vomit continuously, and gastric suction with intravenous fluid and electrolyte replacement was necessary. Following operation, 2 g. of streptomycin was administered daily by the intramuscular route, supplemented by 2 g. of chloramphenicol orally on December 3, when gastric aspiration and intravenous fluid replacement were discontinued.

By December 6 the patient had recovered considerably and was fit enough to be evacuated by air to Cyprus. On December 12 he was up and about on normal diet, and was evacuated to the United Kingdom.

Case 3

This patient, a private aged 23, was a member of the Norwegian contingent of U.N.E.F. He was admitted to hospital on December 2, 1956, with pyrexia of 101° F. (38.3° C.), central abdominal pain, and colic. He complained of nausea, and vomited repeatedly. The leucocyte count was 12,000 (neutrophil granulocytes 82%, lymphocytes 12%, monocytes 5%, eosinophils 1%).

When examined he had marked tenderness in the right iliac fossa with guarding. Rebound tenderness was elicited. Shifting tenderness and free fluid were absent on clinical examination, rectal tenderness on digital examination was marked. A provisional diagnosis of acute appendicitis was made.

At operation on December 2 a moderate amount of clear peritoneal fluid was present. The appendix was found to be normal. The terminal 5 in. (12.5 cm.) of ileum was thickened and oedematous and of a deep plum colour; the mesenteric lymph nodes were also enlarged and fleshy. The abdomen was closed without removal of the appendix or drainage. Post-operatively he was given 2 g. of streptomycin and 2 g. of chloramphenicol daily. Recovery was uneventful until December 4, when he developed mild diarrhoea. On December 7 he was well enough to be transferred to the Norwegian hospital. He was symptom-free and back to full duty on January 12, 1957.

Discussion

Felsen (1936) followed up 400 cases of bacillary dysentery, among which 29 cases of acute distal ileitis occurred. Crohn (1949), while accepting that a condition similar to acute distal ileitis may occur during epidemics of bacillary dysentery, considered the imputation by Felsen that *Shigella dysenteriae* was the cause of acute ileitis unsubstantiated.

Reports of acute distal ileitis occur not infrequently in the literature, though usually in association with non-specific

mesenteric adenitis. Rosenberg (1937) described two cases occurring among 75 cases of non-specific mesenteric adenitis. Aird (1945) also mentioned the occurrence of this condition in association with non-specific mesenteric lymphadenitis. Jackson (1937) suggested that acute non-specific mesenteric lymphadenitis and acute distal ileitis are phases of the same disease. Crohn (1949) disputed this, but thought that acute ileitis was undoubtedly an individual phase of regional ileitis capable of complete subsidence in 25 to 50% of cases. Warren and Sommers (1948) reported 15 cases of acute ileitis, 12 of which regressed, 2 progressed to the chronic phase, and 1 patient died. Rose (1946) reported three cases diagnosed and operated on for acute appendicitis. Armitage and Wilson (1950) reported 26 cases of acute terminal ileitis submitted to laparotomy in 15 years. Ten of these were followed up, and some of them had been subjected to appendectomy. All recovered completely. They considered it unlikely that Crohn's disease began in this way.

There is obviously great variance of opinion on the aetiology and prognosis of acute terminal ileitis.

Summary

An account of the incidence of appendicitis and acute ileitis during an epidemic of acute gastro-enteritis is given.

Some of the previous literature on acute ileitis is reviewed and three cases of this condition are described.

The patients with acute ileitis were given short courses of streptomycin and chloramphenicol, since a causal organism would neither be incriminated nor excluded. Appendectomy was performed in one case in error. Most authorities consider appendectomy ill advised in this condition, in view of the dangers of perforation and fistula formation.

Early air evacuation for post-operative cases was possible because a short flight at low altitude only was involved.

In the absence of laboratory investigations no conclusions with regard to aetiology in these diseases can be drawn. Nevertheless, their incidence over a period of six weeks in a relatively small population of young healthy adult males is remarkable, and does suggest that a common factor operated to produce all three conditions.

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The muscular dystrophy group of the Central Council for the Care of Cripples announces that, since its inception a little over two years ago, its members have collected over £12,000 to support research on diseases of muscle. Grants have already been made to support work in London, Oxford, Edinburgh, Newcastle upon Tyne, and Birmingham, and other applications for funds are at present under consideration. Workers engaged in studies of normal or diseased human muscle who need financial support are invited to write for information to the Central Council for the Care of Cripples, 34, Eccleston Square, London, S.W.1. The muscular dystrophy group would also be interested to hear from sufferers from muscular dystrophy, and their friends and relatives, who are not at present members.