In seven patients low titre L.K.M. antibodies were found in the absence of any evidence of liver disease and in two of these their appearance was temporary. All but one of these patients suffered from collagenoses or thyroid autoimmunity. In this context it is interesting that in half of all patients having L.K.M. antibodies, irrespective of diagnosis, thyroid or gastric organ-specific antibodies also occurred. The identity of these antibodies could be shown by selective absorption of the thyroid or gastric immunofluorescence, leaving intact that due to the L.K.M. The L.K.M. antibody with its restricted organ reactivity may represent an intermediate form of autoimmunity between the strict organ specificity of the thyroiditis/gastritis/adrenalitis group of disorders and the complete non-organ specificity of such autoantibodies as A.M.A. and A.N.A.

Studies are in progress to attempt a separation of L.K.M.-associated active chronic hepatitis by tissue typing (Mackay and Morris, 1972) and the incidence of high titre viral antibodies, including rubella, measles, and herpes (Triger *et al.*, 1972), from the largest group of active chronic hepatitis patients who show no autoimmunity or HBsAg and from the subgroups associated with high titres of A.N.A. and S.M.A. and those showing A.M.A. in the serum.

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ADDENDUM

Since this paper was submitted further information on the clinical significance of the L.K.M. antibody has come from Dr. J.-C. Homberg, of Paris. He observed this immunofluorescence pattern independently and with other French immunopathologists collected 14 positive cases (11 F., 3 M.) over the past five years, representing about 0.1% of all sera tested. Ten were from patients with either

active chronic hepatitis in young subjects or with unexplained cirrhosis (Homberg *et al.*, 1974).

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Haemolytic-Uraemic Syndrome in Typhoid Fever

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Summary

Among 48 patients with a typhoid infection 6 (12.5%) developed the haemolytic-uraemic syndrome. Neither glucose-6-phosphate dehydrogenase deficiency nor therapy with chloramphenicol could be incriminated as the causal factor. Evidence presented here suggests that the mechanism is localized intravascular coagulation.

The presence of leucocytosis in typhoid fever suggests a complication and should alert one to the possibility of the haemolytic-uraemic syndrome. Furthermore, in our area typhoid should be suspected as a cause in any patient presenting with acute renal failure.

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Introduction

Haemolysis and renal failure have been regarded as rare complications of typhoid fever. Retief and Hofmeyr (1965) found fewer than 40 cases of haemolytic anaemia associated with typhoid noted in the literature. Since then there have been further reports, usually of single cases. That this complication might be more common was suggested when Lwanga and Wing (1970), in a two-year retrospective study, found that of 130 patients with typhoid 7% had evidence of haemolysis, but that was in areas where glucose-6-phosphate dehydrogenase (G-6-PD) deficiency was common. Hersko and Vardy (1967) described haemolysis in five children, all with G-6-PD deficiency. Huckstep (1962) mentioned an incidence of haemolytic anaemia of 2% and of "nephrotypoid" of 1%. Gulati *et al.* (1968) reported two cases of nephritis among 98 patients, and Wicks *et al.* (1971) reported renal complications in six out of 265 patients.

Lwanga and Wing (1970) claimed the first reported case of acute oliguric renal failure after intravascular haemolysis in typhoid fever. That was in a patient with G-6-PD deficiency. Though Allen (1969) reported one case with consumption co-

agulopathy, stating that this had not been previously reported in typhoid fever, Faerman *et al.* (1972), who reported a case with hepatitis, nephritis, and thrombocytopenia, believed that in neither their patient nor Allen's was there sufficient evidence to support a diagnosis of consumption coagulopathy.

Chloramphenicol has been incriminated as the cause of haemolysis (La Grutta *et al.*, 1967), while McCaffrey *et al.* (1971) thought that chloramphenicol intensified the haemolysis seen in white G-6-PD-deficient patients with typhoid fever.

Our aim is to show that haemolysis and uraemia are more common than has been appreciated, and we discuss what we believe to be the underlying mechanism of these complications.

Patients and Methods

The 48 cases of proved typhoid fever in Africans admitted to Mpilo Hospital during the two-year period 1 January 1971 to 31 December 1972 were reviewed. Haemoglobin estimations and full blood counts were made with a Coulter model S electronic cell counter. Blood urea was estimated by a standard diacetylmonoxime method with an Autoanalyzer, serum bilirubin by the Malloy-Evelyn method as described by Wootton (1964), and plasma fibrinogen by a modified Ellis-Stransky method (Burmester *et al.*, 1970). Serum fibrin and fibrinogen degradation products were assessed later in the study by a commercial latex method (Thrombo-Wellcotest), and in the earlier months by a modified Fi test (Hyland Laboratories Ltd.) as described by Merskey *et al.* (1966).

Other haematological investigations included platelet counting by the Brecher-Cronkite method with phase microscopy, reticulocyte counting, Schumm's test, and estimation of red cell G-6-PD activity (Motulsky method), prothrombin index by Quick's method (calibrated with Manchester thromboplastin and reported as British corrected ratio. Kaolin activated partial thromboplastin time, whole blood coagulation time (Lee and White's method), and direct Coombs tests were performed by standard techniques as described by Dacie and Lewis (1968). The presence of fragmented red cells was confirmed by one of us (A.E.M.) using multiple fresh blood films. Bacteriological isolation and identification was performed by standard techniques (Kauffman, 1965), as were full Widal titrations (Cruikshank, 1965). Widal screening tests were performed as slide tests with concentrated *salmonella typhi* O suspensions (Hyland Laboratories Ltd.).

As this series was derived from a retrospective study complete laboratory data were not available for all cases.

Results

Evidence of Typhoid Fever.—*S. typhi* was cultured from each of the 48 patients; 42 had positive blood cultures with or without positive stool or urine cultures or both, 4 had positive stool cultures, and 2 had positive urine cultures.

Evidence of Haemolytic-Uraemic Syndrome.—In six patients there was evidence of the haemolytic-uraemic syndrome (table I). The clinical course of these patients is summarized below.

Case 1.—A 45-year-old man had been ill for three weeks with headache and abdominal pains, and for two weeks he had had loose stools and occasional vomiting. On examination he appeared ill, dehydrated, and slightly jaundiced but was afebrile (indeed, there was no significant fever at any stage). The spleen was slightly enlarged. Blood urea was 543 mg/100 ml and haemoglobin 6.5 g/100 ml. There was evidence of intravascular haemolysis. He was treated with intravenous heparin 2,000 U four-hourly for 10 days, blood transfusions, and correction of the fluid and electrolyte balance. Chloramphenicol was begun on the third day and given for 21 days. After an initial deterioration and a haematemesis on the tenth day he slowly improved. He was discharged well two

months after admission. Two years later the haemoglobin, blood urea, and urine were normal.

Case 2.—A 26-year-old man had been ill for eight days with headache, abdominal pain, fever, and, later, vomiting and dark urine. He was ill and febrile and had abdominal tenderness. Typhoid was diagnosed, and chloramphenicol and chloroquine were begun immediately. Two days later he had deteriorated. His haemoglobin had fallen from 12.6 to 7.9 g/100 ml, with evidence of haemolysis, and his blood urea was 320 mg/100 ml. Heparin was not given. He died on the third day. At necropsy typhoid enteritis was confirmed.

Case 3.—An 18-year-old man had been ill with headaches and abdominal pain for two weeks and diarrhoea for two days. On admission he was found to be very ill, with anaemia, acidotic breathing, and uraemic frost. Temperature was normal. Blood urea was 840 mg/100 ml and haemoglobin 4.1 g/100 ml, with evidence of haemolysis. He was treated with peritoneal dialysis, blood transfusion, chloramphenicol, and heparin 1,000 U four-hourly. One month after admission he was discharged fully recovered.

Case 4.—A 20-year-old man had a two-week history of headache, abdominal pain, constipation, and epistaxis. He was confused, emaciated, dehydrated, jaundiced, and anaemic. Blood urea was 416 mg/100 ml and haemoglobin 6.3 g/100 ml, with evidence of haemolysis. Heparin 4,000 U six-hourly and chloramphenicol were begun at once. Treatment also included correction of fluid and electrolyte balance, frusemide, and blood transfusions. He recovered completely within 15 days.

Case 5.—A 36-year-old woman had been unwell and febrile for one week and had abdominal pain and diarrhoea for one day. She was pyrexial, ill, and anaemic. The progression of the anaemia, uraemia, and thrombocytopenia and response to peritoneal dialysis, heparin, and chloramphenicol are shown in fig. 1. She was discharged fully recovered six weeks after admission.

FIG. 1—Case 5. Clinical course showing clearly main features of haemolytic-uraemic syndrome.

Case 6.—A 15-year-old girl complained of headache, vomiting, and diarrhoea for two days. She was ill, febrile, dehydrated, and drowsy. Haemoglobin was 11.6 g/100 ml and blood urea 91 mg/100 ml. She was treated with chloroquine and, as typhoid was suspected, chloramphenicol. Despite an apparently adequate urine output the blood urea rose to 260 mg/100 ml over the next four days. Haemoglobin fell to 8 g/100 ml and fibrin degradation products and fragmented erythrocytes were found in the blood. Intravenous heparin 1,000 U four-hourly was added to her treatment and two weeks later she had recovered.

TABLE I—Clinical and Laboratory Data in 6 Cases of Typhoid in which Haemolytic-Uraemic Syndrome developed. Only the most Abnormal Values are shown

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6
Duration of illness at time of admission ..	3 Weeks	8 Days	2 Weeks	2 Weeks	1 Week	2 Days?
Leucocytes/mm ³ ..	41,300	9,600	13,100	33,700	10,400	7,800
Evidence of typhoid:						
Culture { Blood	Pos. × 1	Pos. × 1	Pos. × 2	—	Pos. × 4	Pos. × 1
{ Stool	Pos. × 1	—	—	Pos. × 1	Neg.	—
{ Urine	—	—	—	—	Neg.	—
Widal test { Screen	—	—	Neg.	—	—	Neg.
{ Titre	O1/16c; H1/320	—	—	O1/160; H1/80	O1/320; H1/20	—
Evidence of haemolysis:						
Haemoglobin (g/100 ml)	3.5	7.9	4.1	4.1	6.2	8.0
Reticulocytes (%)	14	0.5	22	19.8	9.2	2.2
Schumm's test	Pos.	Pos.	Pos.	Pos.	Pos.	Neg.
Serum bilirubin (mg/100 ml)	3.5	1.6	<1	3.3	—	<1
Coombs test	Neg.	—	Neg.	Neg.	—	Neg.
G-6-PD	Normal	—	Normal	—	Normal	Normal
Fragmented R.B.C.s	Present	Not recorded	Present	Present	Present	Present
Evidence of consumption coagulopathy:						
Platelets/mm ³	60,000	—	154,000	85,000	31,000	52,000
Plasma fibrinogen (mg/100 ml)	415	—	—	375	307	242
P.T.T.K. (sec)	—	—	—	—	59.3	60.9
Prothrombin index*	—	—	—	1.24	1.22	1.32
Fibrin degradation products	>40 µg/ml†	—	Neg.‡	Pos.‡	Pos.‡	>40 µg/ml†
Evidence of renal failure:						
Blood urea (mg/100 ml)	633	320	840	416	670	260
Urine output (ml/24 hr)	700	200	100	300	100	Not recorded

*British corrected ratio.

†Modified Fi test (see text).

‡Trombo-Wellcotest (see text).

Pos. = Positive. Neg. = Negative. P.T.T.K. = Kaolin activated partial thromboplastin time.

TABLE II—Outcome of Treatment

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6
Chloramphenicol ..	500 mg × 4 daily	500 mg 4 hourly	500 mg × 4 daily	500 mg × 4 daily	500 mg × 4 daily	500 mg × 4 daily
Heparin ..	2,000 U 4-hourly	—	1,000 U 4-hourly	4,000 U 6-hourly	4,000 U 6-hourly	1,000 U 4-hourly
Other ..	Transfusion	—	Transfusion peritoneal dialysis	Transfusions, frusemide	Transfusions, peritoneal dialysis	—
Chloroquine ..	—	Chloroquine course	—	—	—	Chloroquine course
Outcome	Recovered	Died on 5th day	Recovered	Recovered	Recovered	Recovered

COMMENT

Except for case 1 no evidence of significant gastrointestinal or other haemorrhage was found in these cases. Cases 1 and 3 were virtually apyrexial throughout the illness. In no patient was the blood pressure notably raised at any stage, though this has been reported to accompany the haemolytic-uraemic syndrome in children.

White Blood Count.—We feel that the leucocyte count is of special interest. Instead of the usual leucopenia found in typhoid fever the lowest count was 7,800/mm³ and the highest was 41,300/mm³ (table I).

Treatment.—When the diagnosis of haemolytic-uraemic syndrome was made heparin was given (table II). Low dosages have been advocated (Bernstock and Hirson, 1960), and these were used in cases 1, 3, and 6. The initial dose was roughly assessed by dividing the platelet count by 50 (Hardisty and Ingram, 1965). After obtaining a whole blood clotting time 1,000 or 2,000 U of heparin was given as an intravenous bolus at four-hourly intervals. Heparin was administered in a higher dose in cases 4 and 5, and in the only fatal case (case 2) it was not used at all.

Discussion

Altogether six of our 48 patients showed evidence of both haemolysis and renal failure. Further cases might possibly have been found had the less seriously ill patients, with features such as moderate anaemia, been as fully investigated as those described.

The haemolytic-uraemic syndrome constitutes a condition in which acute renal failure is associated with micro-

angiopathic haemolytic anaemia and in which, therefore, the presence of red cell fragmentation is essential for establishing the diagnosis. Red cell fragmentation was present in five cases and not recorded in the sixth. Microangiopathic haemolytic anaemia was originally described by Brain *et al.* (1962). Since then it has been shown that red cell fragmentation results from mechanical damage to the erythrocytes as they pass through fibrin strands deposited in damaged small blood vessels (Bull *et al.*, 1968). It is now generally recognized that the deposition of fibrin results from intravascular coagulation (Brain, 1969). What is pertinent to this series, however, is the growing awareness that intravascular coagulation need not always be of the widely disseminated type or so-called disseminated intravascular coagulation but may be of a localized type; and recent work suggests that the primary lesion of the haemolytic-uraemic syndrome is intravascular coagulation localized in the renal microvasculature (Gilchrist *et al.*, 1969; Gervais *et al.*, 1971). A proposed scheme for the pathogenesis of the haemolytic-uraemic syndrome in typhoid fever, adapted from Brain (1968), is shown in fig. 2.

FIG. 2—Proposed scheme for pathogenesis of haemolytic-uraemic syndrome in typhoid fever.

The concept of localized intravascular coagulation as a basis for the haemolytic-uraemic syndrome implies that the criteria

for diagnosis differ from those of disseminated intravascular coagulation in that it is unnecessary to obtain evidence of massive consumption coagulopathy in the form of hypofibrinogenaemia, but sensitive procedures to detect activation of the coagulation mechanism, such as tests for increased fibrin degradation products, are usually positive. In four out of five cases in which the level of fibrin degradation products was recorded it was, in fact, increased. The platelet count is often subnormal, as in cases 1, 4, 5, and 6 (table I), and fragmented red cells are invariably present, accompanied by a variable degree of haemolytic anaemia and renal failure. (Though platelet count and red cell fragmentation were not documented in case 2 the patient has been included because he had anaemia with a positive Schumm's test, a high blood urea, and a poor urinary output.) Fibrinogen levels may actually be raised in the haemolytic-uraemic syndrome (Gilchrist *et al.*, 1969), as was found in case 1. Increased plasma fibrinogen levels in cases of typhoid fever were recorded by Ogston *et al.* (1964).

Apart from diagnostic considerations the concept of localized intravascular coagulation may also have therapeutic implications. If the role of heparin is accepted it appears rational to infer that localized intravascular coagulation would be controlled by lower doses of heparin than are required for disseminated intravascular coagulation. The outcome was certainly satisfactory in the three cases in our series in which this low dosage scheme was used.

We have been unable to account for the high incidence of the haemolytic-uraemic syndrome in our cases of typhoid as compared with the published experience of others. G-6-PD deficiency was not detected in the four patients in whom it was sought. Except for case 2 the haemolysis and renal failure occurred before treatment with chloramphenicol was started, and this drug can therefore be excluded as the causal agent in these five cases. We found no positive evidence to implicate the traditional herbal remedies as a precipitating factor.

Attention has been drawn to the absence of leucopenia in our cases. A neutrophil leucocytosis in typhoid suggests a complication and could well be another pointer to the development of the haemolytic-uraemic syndrome in this infection.

Furthermore, typhoid must be suspected in any case of acute renal failure seen in this region, even if the patient is apyrexial, as were two of ours.

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Renal Tubular Obstruction by Mucoproteins from Adenocarcinoma of Pancreas

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Summary

We report a case in which mucoproteins from an adenocarcinoma of the pancreas, released into the ascitic fluid and serum, were filtered through the renal glomeruli to form very viscous casts which obstructed the renal collecting tubules and caused the patient's death from oliguria.

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Introduction

Obstruction of the renal collecting tubules with protein casts occurs in up to 30% of patients with myelomatosis (Heptinstall, 1966). It is most often associated with Bence-Jones proteinuria, but occasionally with other proteinuria—for example, IgA (Hobbs, 1966). Oliguric renal failure sometimes occurs with prominent tubular casts in nephrotic patients with heavy albuminuria (Chamberlain *et al.*, 1966). We report a patient who died because his renal tubules were obstructed by the mucoproteins from an adenocarcinoma of the pancreas.

Case Report

The patient was a civil servant aged 43 years. In mid-April 1967 he developed nausea, vomiting, and abdominal pain. On 27 April he was admitted to a local hospital as an emergency. No abnormalities were found on physical or barium meal examination, the blood urea was 32 mg/100 ml, and haemoglobin 14 g/100 ml. His urine contained protein. The symptoms subsided on a milk diet and he