HISTOLOGICAL FINDINGS IN FATAL CASES OF BACILLARY DYSENTERY.

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Introduction.
A histological examination of various organs from fatal cases of bacillary dysentery in the Middle East was commenced in January, 1941, and the following is a report of the findings for the period January 1 to May 31. Brief clinical notes are given in cases where they are of value in co-ordinating the histological picture with the course of the patient's illness.

General.
(a) Incidence.—Tissues from seventeen cases were examined and this number, as compared with the total incidence of bacillary dysentery for the period under review where the figures are available, is shown below under the different nationalities:

<table>
<thead>
<tr>
<th>Nationality</th>
<th>Total Incidence</th>
<th>Fatal Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>B.T.E.</td>
<td>1027</td>
<td>5</td>
</tr>
<tr>
<td>N.Z.E.F.</td>
<td>376</td>
<td>1</td>
</tr>
<tr>
<td>Free French</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Ps.O.W.: Italian</td>
<td></td>
<td>9</td>
</tr>
<tr>
<td>Libyan</td>
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<td>1</td>
</tr>
</tbody>
</table>

Thus, in the first two groups, the death-rate was less than \( \frac{1}{4} \) per cent.
(b) Age.—The age of the fatal cases varied from 22 years to 49 years, but was not stated in eight.
(c) Bacteriology.—The findings in this respect were:
- Isolations: \( B. \text{ dysenteriae} \), Shiga, 6.
- \( B. \text{ dysenteriae} \), Flexner, 1.
- \( B. \text{ dysenteriae} \), mannite-fermenting (non-agglutinating), 1.
- Bacillary exudate, even on repeated examinations, 4.
- Indefinite exudate, often including sigmoidoscopic swabs, 1.
- No investigation, including two fulminating cases, 4.
(d) Duration.—As the onset of bacillary dysentery is usually sudden, the duration of the disease could be stated accurately in all of the cases and they fell into four groups:
- A. 1 week or under, 3, with one Shiga infection.
- B. About 2 weeks (11-15 days), 4, with two Shiga infections.
- C. About 3 weeks (19-21 days), 7, with two Shiga infections.
- D. Over 4 weeks (47, 50, 70 days), 3, with one Shiga infection.

Histological Appearances.
Large Intestine (examined in sixteen cases). In sections of the colon from these cases, the whole range of acute, subacute and chronic inflamma-
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Inflammatory changes was seen in the mucous and submucous coats. The inflammatory process was confined for the most part to the mucosa and superficial area of the submucosa yet it followed the lymphatics and blood-vessels to the deep area and occasionally for a short distance into the muscle layer. The ganglion cells of Auerbach’s plexus were frequently involved in this peri-lymphangitic inflammation, probably explaining the increased irritability of the colon. Sections from only two cases of the series showed general extension of the inflammation into the muscle with oedema, cellular infiltration and changes in the muscle fibres.

The presence of numerous large macrophages was a very prominent feature of the inflammatory exudate. These cells appeared at an early stage in the acute ulcers and were still numerous in the granulation tissue of chronic lesions. In a few cases, they were present under the peritoneum, where an early peritonitis was found. Their size varied from slightly larger than a plasma cell up to 30 to 40 μ in diameter and many contained several red cells and polymorphs plus fragments of others.

Even when desquamation of the mucous membrane was very extensive to naked-eye examination a few tags of epithelium were seen histologically and desquamation of much of the submucosa was rare. In two cases, however, there were areas of desquamation extending right down to the surface of the muscle. In the chronic ulcers, the submucosa was represented by a thick layer of granulation tissue with numerous young capillaries and fibroblasts and a narrow superficial layer showing infiltration of polymorphs, plasma cells, macrophages and lymphocytes.

Congestion of the blood-vessels was very marked and interstitial haemorrhages were frequent. In the fulminating cases, the presence of preformed thrombi in the veins was striking and was probably responsible for changes in the liver and spleen. In one case of the series, the immediate cause of death was repeated severe haemorrhage from the colon as a result of these congested and dilated vessels being exposed by the desquamation and, in another case, haemorrhages played a prominent part in the fatal termination.

The ulceration of the colon was generally more marked in the lower part—sigmoid and rectum—but, in two cases, the cecum and ascending colon showed more chronic changes and evidence of healing while acute lesions were developing at the lower end.

Small Intestine (examined in thirteen cases). Three cases showed acute inflammatory changes in the terminal ileum, four showed merely congestion and there was no change in the rest.

Peritoneum. — Changes here varied considerably but were not a consistent feature. In ten cases, no abnormality was found post mortem or histologically (in sections of the bowel). Five cases showed early acute inflammatory lesions, in another case a pint of blood-stained fluid was found in the cavity and, in the seventeenth case, pelvic peritonitis was present with two pints of clear fluid and inspissated fibrin flakes.

Mesenteric Lymph Nodes (examined in three cases). Enlarged nodes
showed the typical picture of sinus catarrh, with macrophage activity developed to a very pronounced degree.

_Liver_ (examined in eleven cases). No changes due to the dysentery were found in seven cases. In three of the others, fatty change with some necrosis of the liver tissue was present, advanced in two and only slight in the third; these cases were of long duration and the lesion could not be considered specific. A specimen of liver was obtained in only one of the cases of short duration. Sections showed small areas of necrosis with macrophage infiltration scattered throughout the organ, probably due to small emboli from the vessels in the wall of the large intestine.

_Spleen_ (examined in thirteen cases). This organ consistently showed the changes usually found in acute septic conditions, i.e. congestion of the pulp and reticulo-endothelial activity. In one fulminating case small areas of necrotic tissue were present in some of the Malpighian corpuscles, due to the same cause as the necrotic areas in the liver of the same case.

_Suprarenals_ (examined in fifteen cases). No histological change was present in this organ. In one case, a gross haemorrhage was found in the left suprarenal.

_Heart Muscle_ (examined in eight cases). No special lesions were found here. In two cases, both of long duration without sudden death, fragmentation and segmentation of the muscle fibres was seen. The capillaries of the skin and subcutaneous tissue showed no histological abnormality.

Thus the circulatory failure which developed terminally in many of the cases was a functional one and not due to any specific lesion in the heart or small blood-vessels.

_Kidney_ (examined in fifteen cases). In describing the lesions in this organ, the cases will be considered in groups according to the duration of the disease and emphasis will be laid on cases due to proved Shiga infections.

A.—Duration of One Week or Less.

The case of Shiga infection in this group developed bilateral bronchopneumonia and died in seven days. His kidney showed patchy glomerular congestion and catarrhal changes in the tubules, of no specific importance in a patient with severe toxæmia.

R.—Duration about Two Weeks.

In one of these cases, an Italian aged 49, the glomeruli showed increase of size and cellularity with thickening of the capillaries and occasionally reduplication of the capsular epithelium. The tubules showed various stages of degeneration most marked in the convoluted and least marked in the collecting parts. This picture was very suggestive of an early glomerulonephritis. In the other Shiga infection post mortem autolysis was very marked and obscured what appeared to be a similar finding.

In a case of this group, in which the causal organism was not isolated,
there was definite acute glomerulonephritis affecting chiefly the capillaries of the tuft—"intracapillary." This case collapsed on the fourteenth day of his illness, his tongue was dry and coated, hiccup developed and he passed into a state of coma vigil. Next day his pulse became rapid and poor, he became semiconscious and died. The syndrome suggested uræmia.

C.—Duration about Three Weeks.

An Italian, aged 28, admitted to hospital on the eighteenth day of his illness in a collapsed condition with albuminuria, died two days later. *B. dysenteriae* (Shiga) was cultured from the colon post mortem. The kidneys showed widespread damage, with increased size and cellularity of the tufts, some with intense congestion, many with reduplication and desquamation of the capsular epithelium. The first convoluted tubules were necrosed or showed extensive cloudy swelling, while the epithelium of the other tubules was not affected although granular or hyaline material was often present in the lumen. This lesion indicated at least a very severe toxic reaction, possibly a genuine glomerulonephritis.

In another case of this group, where the causal organism was not isolated, the kidneys showed an intracapillary glomerulonephritis superimposed upon previous vascular damage. A third case gave evidence of an acute exacerbation of a chronic glomerulonephritis.

In the remaining cases the histological findings were not distinctive.

D.—Duration Over Four Weeks.

The case infected with Shiga in this group gave a very interesting history. After a fortnight's severe illness with diarrhœa, vomiting, weakness and toxæmia, his blood urea was 60 mgm. per 100 c.c. and he showed albuminuria which cleared up in a few days. He did not convalesce in the usual manner: his pulse-rate remained high and nausea and vomiting persisted. After a few weeks, towards the end of which œdema developed in his legs, he became disoriented, the pulse-rate rose still higher and he died on the forty-seventh day of illness. Post mortem, a low-grade peritonitis, with two pints of thin watery pus, was found. Histologically, the kidneys showed a generalized intracapillary glomerulonephritis at a late acute or early subacute stage. The colon showed the usual changes but there was nothing of note in the other organs.

Another case (Flexner infection) which persisted for seventy days showed a similar kidney lesion but patchy in distribution.

In the whole series, it could not be stated dogmatically that so many of the cases showed glomerulonephritis and so many did not. There were two reasons for this difficulty:

(a) Accurate histological interpretation was impossible in some, owing to autolysis or to bad fixation of the tissues.

(b) The borderline, histologically, between appearances due to severe
toxaemia and those which can be called frank glomerulonephritis is very hard to determine. Assistance in recognizing the latter was given in several cases of this series by a sufficient clinical history.

**Kidney Lesions in Non-fatal Cases.**

The following brief notes of two cases, out of a series of 234 (seven Shiga infections) with no deaths, show that a kidney lesion occurs in bacillary dysentery in cases which recover but obviously, in one of them, the organ has suffered very severe damage.

(i) Patient, aged 22, Shiga infection. On the twelfth day of illness, slight generalized oedema developed with suppression of urine for twenty-four hours and the temperature rose to 104°F. Next day, the urine showed albumin (less than a half part Esbach), hyaline and granular casts, polymorphs and epithelial cells, and paracolon bacillus on culture. His temperature fell and rose again in the next few days but it, and also the oedema, had settled by the sixteenth day although there was still a trace of albumin in the urine with scanty granular and hyaline casts. Thereafter, all the signs disappeared and the urine was completely normal by the twenty-third day.

(ii) Patient, aged 23, report on faeces examination—bacillary exudate (causal organism not isolated)—but at the end of the seventh week of illness a culture of *B. dysenteriae* (Shiga) was agglutinated by the patient's serum in a dilution of one in fifty. This patient was admitted with severe toxaemia and had a copious haemorrhage from the bowel on the ninth day of illness. At this time his urine was free from albumin. On the twenty-second day he became drowsy, his heart was enlarged, arthritis developed in both shoulders and elbows and his general condition was very poor. Next day there was a considerable amount of albumin in his urine (four parts Esbach), numerous hyaline and granular casts, red and white cells and epithelial cells, but no growth on culture. The blood urea was 64 mgm. per 100 c.c. On the twenty-fifth day there were seven parts Esbach albumin in his urine, casts and cells not quite so numerous. By the thirty-second day his general condition was greatly improved, his blood urea down to 30 mgm. per 100 c.c. and albuminuria to one and a half parts Esbach, granular casts scanty. One week later the blood urea was 35 mgm. per 100 c.c., albuminuria one part Esbach, casts again numerous. Clinically, this case was considered to be an exacerbation of previous damage.

**Comments.**

From the results obtained so far in this investigation, the main interest lies in the discovery of nephritic lesions in a considerable proportion of these fatal cases of bacillary dysentery. Pyelonephritis due to *B. coli* has been reported (Manson Bahr, 1940) as a complication of this type of dysentery and a similar condition due to *B. dysenteriae* of all three strains (Shiga, Flexner and Sonne) by Neter and Fisher, 1938. But no accounts of the disease (with one possible exception—Wauke, 1938) describe the occurrence of glomerulonephritis. It has been shown above that this complication may develop, usually at the end of the second or third week, and is very probably due to the toxaemia which is most severe in Shiga infections (Rogers and...
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Megaw, 1939). The nephritis may contribute materially to the individual's death in the acute stage by causing uraemia. It may develop gradually, masked by absence of the usual gradual convalescence of the dysentery, and death occur in the subacute stage. In non-fatal cases, lesser degrees of kidney damage may be caused with either complete recovery or the danger of serious illness in later life.

Another point is the occurrence of emboli to the liver and spleen in the fulminating cases. From two cases more recent than this series, it has been found that these emboli may also go to the lungs and cause hemorrhagic infarcts and bronchopneumonia, which is another common complication in fatal cases of bacillary dysentery.

Summary.

The histological findings in a series of seventeen fatal cases of bacillary dysentery have been described.

The occurrence of glomerulonephritis as a complication was noted in several of the cases. It was shown that a similar condition occurred in a few cases which recovered.

N.B.—These cases died before the general introduction of the sulfaguanidine treatment for serious cases of bacillary dysentery and none of the series was given the drug early in their illness. Two were given large doses in the later stages but, by that time, in each case, the kidney was already damaged. Several of the series were given large doses of anti-dysentery serum from the beginning, apparently without effect on the development of the kidney lesion.

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References.


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