Pathology of the alimentary tract in *Salmonella typhimurium* food poisoning

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**SUMMARY** The pathology of the alimentary tracts of nine patients dying of *Salmonella typhimurium* infection is reviewed. Two patients had previous gastric operations, supporting previous reports that such patients are more susceptible to food poisoning. Four had no parietal (oxyntic) cells in the gastric mucosa, suggesting hypo- or anacidity. Only one had acute gastritis. None had acute enteritis, but in half of the patients, subtle histological changes suggested an 'enteropathy'. Acute diffuse colitis with abundant crypt abscesses, without stromal abscesses in the lamina propria, was the most constant finding and reparative features started very early, and occurred in later deaths. Under ideal circumstances this crypt abscess is readily distinguished from that of idiopathic ulcerative colitis, but can be confused with the crypt abscess of acute bacillary (sonne) dysentery. While the florid colonic changes may have settled in the late deaths, active inflammation is commonly present in the appendix mucosa on histology. The pathology of the alimentary tract in *S typhimurium* infection differs from that of *S typhi* and *S paratyphi* infections. There is little evidence of gastroenteritis, although subtle changes occur in the stomach and small intestine. The features are those of acute diffuse colitis with histological appendicitis, distinguishable from idiopathic ulcerative colitis.

Statistics suggest that the incidence of food poisoning in the United Kingdom is increasing. Food poisoning is commonly referred to as 'gastroenteritis', implying inflammation of the stomach and small intestine. During an outbreak of *S typhimurium* (phage type 32) infection in the west of Scotland in 1968, it was surprising to find virtually no gastritis, or enteritis, and that the predominant histological lesion was an acute diffuse colitis. Further experience has been gained from *S typhimurium* infection of other phage types and from other salmonella subtypes. Other publications have confirmed the colitis component of *S typhimurium* infection, but the findings in the stomach and small intestine remain un-reported. Indeed, the colonic findings have not been adequately described. In this paper the pathology of the alimentary tract in nine patients dying from *S typhimurium* infection, involving at least four phage types is described. The first necropsy was done before phage typing had been routinely accepted.

**Methods**

**Necropsies**

The nine necropsies were carried out as soon after death as possible. Two loops of jejunum and two of sigmoid colon were sent for microbiological examination and virology. Blood samples from the right auricle were also taken for culture and Widal tests.

The necropsy was standard with the gastrointestinal tract being dissected out and opened with the minimum of handling. Hosing with water and sponging was avoided to allow inspection of the luminal contents and the undisturbed mucosal surfaces.

After formalin fixation standard sets of blocks for histological examination were taken from the gastric fundus, jejunum, jejun-ileal region, ileum, appendix, and from the ascending, transverse and descending colon. These were chosen to include any obvious pathology at these sites. In all 27 blocks of stomach, 43 of small intestine, 27 of appendix and 90 of colon were examined. All tissue was paraffin embedded and stained with haematoxylin and eosin. Standard special stains were carried out when thought necessary.
Results

Patients
Clinical data are shown in Table 1. Patient 1 developed diarrhoea and vomiting when less than one month old. No pathogen was isolated from watery stool specimens during the 10 days in another hospital. After recovery the infant remained well for seven days when less severe diarrhoea developed. On the second admission no pathogen was cultured from a number of stool specimens and the illness settled with gain in weight after two weeks. Pyrexia, however, developed two weeks later— that is, 10 days before death. Penicillin was given, but had no effect. Blood culture four days before death (34 days after the second admission) yielded *S typhimurium*. The stools became loose again on the day before death. These observations, in conjunction with the histology, suggest that the infant’s alimentary *S typhimurium* illness was about one day’s duration.

Patients 4–7 were ill at home, then admitted to other hospitals before being transferred to Ruchill Hospital. Patient 6 became ill on transit to Tunisia, and circulatory complications to her left leg arose while there. Medical attention was given in Tunisia, but an emergency disarticulation at the hip joint was undertaken within two days of her return to Glasgow. She was in a very toxic state and she died six days later.

Necropsy findings

1 Stomach
Two patients (5 and 7, Table 1) had undergone gastric surgery. Patient 5 had hypertrophic and patient 9 very atrophic gastric mucosa. Acid-secreting cells were not identified in four (50%) of the stomachs (Table 2). Only one patient (no. 2) had acute gastritis with abundant crypt abscesses (Fig. 1). Very occasional minor crypt abscesses and epithelioid granulomata in the mucosa indistinguishable from Crohn’s disease, were present in patient 7. Thus, gastritis was florid in only one patient who was one of the four patients not showing oxyntic cells.

2 Small intestine
The macroscopic abnormalities were minimal with mild swelling and oedema of some of the mucosal ridges. In four patients (2, 3, 4 and 9) the terminal ileum was reddened and suggested a reflux ileitis.

Histological examination showed that in four cases the small bowel was normal (4, 6, 8 and 9). The villi were normal in height and width. Of the other five cases only one (2) had well established acute inflammatory changes while in the remaining four these were mild (Table 3). This took the form of occasional crypts plugged by mucin and macrophages with ingested nuclear debris. Some crypts were lined by nondescript ‘embryonal-like’ cells with prominent nucleoli and haematoxyphilic cytoplasm (Fig. 2). Occasional capillary thrombi were observed and some perivasculat inflammation in the lamina propria. No granulomas or fissures were seen and Peyer’s patches were not prominent.

Thus only one patient had a well established enteritis with recognisable crypt abscesses and four had mild ‘enteropathic’ changes.

3 Large intestine
Three colons (nos. 2, 3 and 4) were grossly normal, two were collapsed (1 and 7), two (6 and 8) were moderately dilated. Patient 8 had diverticulosis of the sigmoid and a 15 μl abscess in the pouch of
Table 1  Data concerning nine patients dying with Salmonella typhimurium infection ranked by duration of illness

<table>
<thead>
<tr>
<th>Patient no</th>
<th>Sex</th>
<th>Age at Death</th>
<th>Days ill at home/on holiday</th>
<th>Days ill in hospital</th>
<th>Total days ill until death</th>
<th>Previous alimentary surgery</th>
<th>Other conditions antemortem or at necropsy</th>
<th>Organism isolated from</th>
<th>Phage type</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>2mth</td>
<td>&lt;1</td>
<td>10</td>
<td>Nil</td>
<td>Marasmic on admission.</td>
<td>Blood culture</td>
<td>Faeces negative</td>
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<td></td>
<td></td>
<td>At necropsy, dehydrated</td>
<td>Faeces positive</td>
<td>One bl. culture neg.</td>
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<tr>
<td>2</td>
<td>F</td>
<td>65y</td>
<td>&lt;1</td>
<td>38</td>
<td>&lt;39*(1)</td>
<td>Atrial fibrill. Myocarditis</td>
<td>Faeces negative</td>
<td>Bl. culture pos.</td>
<td>32</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>65y</td>
<td>5</td>
<td>2</td>
<td>7</td>
<td>Permanent tracheotomy after laryngectomy. No metastases</td>
<td>Faeces negative</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>6y</td>
<td>&gt;1</td>
<td>7</td>
<td>&gt;12</td>
<td>Lymphatic leukaemia under treatment</td>
<td>Faeces positive</td>
<td>U252</td>
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</tr>
<tr>
<td>5</td>
<td>M</td>
<td>62y</td>
<td>6</td>
<td>3</td>
<td>16</td>
<td>Uraemia due to renal tubular necrosis</td>
<td>Faeces positive</td>
<td>One bl. culture neg.</td>
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<tr>
<td>6</td>
<td>F</td>
<td>43y</td>
<td>11</td>
<td>1</td>
<td>19</td>
<td>Ac. gangrene of L. leg with oper. hip disarticulation</td>
<td>Faeces and bl. culture pos.</td>
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<tr>
<td>7</td>
<td>M</td>
<td>48y</td>
<td>8</td>
<td>13</td>
<td>23</td>
<td>Partial gastrectomy SBE (S. viridans)</td>
<td>Blood culture pos.</td>
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<tr>
<td>8</td>
<td>F</td>
<td>81y</td>
<td>21</td>
<td>8</td>
<td>29</td>
<td>Metastases to brain, cord, liver, lung of ovarian cancer. Involuting zoster</td>
<td>Faeces antemortem, pus pelvic abscess PM both pos. Blood culture not done</td>
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</tr>
<tr>
<td>9</td>
<td>F</td>
<td>67y</td>
<td>1</td>
<td>57</td>
<td>58</td>
<td>Cholecystectomy Asthma, Rheum. arth. Bilat. pulm. art. emb.</td>
<td>Faeces pos. Nine bl. culture neg.</td>
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<td></td>
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* Two separate admissions to hospital, with seven days at home between episodes and histology suggests that S. typhimurium infection was terminal and unrelated to the earlier episodes. (See text.)
Table 2  Summary of histological findings in the stomach

<table>
<thead>
<tr>
<th>No</th>
<th>Parietal (oxyntic) cells</th>
<th>Chronic gastritis</th>
<th>Intestinal metaplasia</th>
<th>Acute crypt abscesses</th>
<th>Acute inflammation of lamina propria</th>
<th>Granulomata in lamina propria</th>
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Douglas with no connection to diverticula, from which *S typhimurium* was cultured. Two had toxic dilatation, of the whole colon (no 5) or of the caecum and ascending colon only (no 9). Patient 9 also had diverticulitis.

The mucosa was grossly normal in patients 1, 5, 6 and 9 (apart from the diverticulitis) despite the histology in patient 1, and the toxic megacolon in patients 5 and 9. In patient 8 the mucosa was patchily red. The mucosal ridges were red throughout in patient 2, while the mucosa was diffusely red beyond the splenic flexure in patients 3 and 4. The mucosa of patient 7 was cyanotic from the sigmoid flexure. All these features might have been ignored, if the history of a diarrhoeal illness had not been available. There was no melaena, or bloodstaining in any case.

The histological features are summarised in Table 4. Patient 1 showed universal loss of the mucosal surface and the exposed lamina propria displayed shallow diffuse fibrinous necrosis along the entire bowel wall covered by haematoxyphyllic (Gram negative) bacteria (Fig. 3). The columnar cells lining the lower halves of the crypts had prominent eosinophilic nucleoli, hyperchromic nuclei, raised nuclear/cytoplasmic ratios and abundant mitotic activity. Crypt abscesses were absent. The lamina propria showed oedematous granulation tissue with new capillary loops growing into the zone of fibrinoid necrosis. Some capillaries showed fibrin/platelet thrombi. The deeper lamina propria and the submucosa contained a mixed inflammatory infiltrate.

Varying mucosal surface repair with crypt abscesses occurred in all acute deaths. Patient 2 (three day illness) showed abundant crypt abscesses (Fig. 4) rich in eosinophil leucocytes, neutrophil polymorphs, plasma cells, lymphocytes, mononuclear cells, and desquamated crypt lining cells. A few crypts were ballooned with squamous lining cells at the sides and necks, but at the bases, cells were stratified upon one another. The lamina propria showed no stromal abscesses. Scattered haemorrhages and very scanty capillary thrombi were

Table 3  Summary of histological findings in the small intestine

<table>
<thead>
<tr>
<th>No</th>
<th>Protein-rich vesicles at villous tips</th>
<th>Acute focal enteritis with crypt abscesses</th>
<th>Plasma cells in lamina propria considered to be excessive</th>
<th>Non-differentiated crypt columnar epithelium with no brush border, high nucleo/cyto ratio, prominent nucleoli</th>
<th>Excessive exfoliation of lining cells into crypt lumen</th>
<th>Insipissated mucin plugs in crypt lumina</th>
<th>Capillary thrombosis in villous stroma and/or lamina propria</th>
<th>Perivenous cuffing by lymphocytes and/or neutrophil polymorphs in submucosa</th>
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</table>
Pathology of the alimentary tract in Salmonella typhimurium food poisoning

The submucosa was very oedematous with mild cellulitis and lymphangitis.

Patient 3 (seven days) had a non-differentiated columnar surface epithelium (Fig. 5). Patient 5 (16 days) had ballooned crypts, some with mucin plugs, others with cell debris and a few showed abscesses. Most crypts were rich in goblet cells. Patient 6 (19 days) had tall crypts with abundant goblet cells, but there were isolated ballooned crypts with cell debris in the lumen, and flattened epithelium round their walls. The superficial lamina propria showed persisting necrosis. Patient 7 (23 days) had tall crypts (Fig. 6) rich in goblet cells, a normal quota of goblet cells on the surface and a moderately dense infiltration of lymphocytes superficially in the lamina propria – that is, where fibrinoid necrosis is assumed to have been. Patients 8 (29 days) and 9 (58 days) had no additional features.

All patients had subtle acute diffuse colitis with crypt abscesses in most and evidence of repair. Granulomata and fissures were absent. While histological recovery could occur by two weeks (no 4), evidence of continuing infection could still be seen at four weeks (no 8).

4 Appendix

All appendices appeared grossly normal, but were studied histologically in patients 2 to 9 (Table 5).

Appendicitis was most severe in patient 3 with zones of fibrinoid necrosis, scanty crypt abscesses, and excessive mucopus in the lumen. Patient 7 showed an epithelioid granuloma in the lamina propria, similar to the stomach findings. The inflammatory changes were more severe in the appendix than in the colon of the same patient, although in toto there was more inflammation in the colon.

Discussion

This series suggests that the young and the elderly are most likely to die from food poisoning, but it also shows that patients need not be debilitated. The patients had clinical states similar to those reported by Dickinson and Pickens.13

The relative absence of gross features at necropsy

Table 4  Summary of the histological findings in the large intestine

<table>
<thead>
<tr>
<th>No</th>
<th>Superficial fibrinoid necrosis</th>
<th>Crypt abscesses</th>
<th>Ballooned crypts</th>
<th>Non-differentiated epithelium</th>
<th>Reappearance of goblet cells</th>
<th>Serosal inflammation of lamina propria</th>
<th>Lymphocytic infiltration in superficial lamina propria mucosae</th>
<th>Paneth cells per inch of colon wall</th>
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<tr>
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</table>
Fig. 3  Patient 1. Large intestine. Representative field. Incomplete haematoxyphlic layer of bacilli (Gram-negative) overlying a shallow fairly even layer of fibrinoid necrosis into which new capillary loops are migrating from the lamina propria. The crypts of Lieberkühn are lined by non-differentiated columnar cells, squamoid at the crypt necks. The submucosa shows widespread cellulitis and the venules show pavemencing by neutrophil polymorphs. H and E. Original x 250.

Table 5  Summary of the histological findings in the appendix

<table>
<thead>
<tr>
<th>No</th>
<th>Superficial fibrinoid necrosis</th>
<th>Crypt abscesses</th>
<th>Ballooned crypts</th>
<th>Non-differentiated epithelium</th>
<th>Epithelioid cell granuloma</th>
<th>Pus in the lumen</th>
<th>Excess mucin in the lumen</th>
<th>Hyperplastic mucosa</th>
<th>Paneth cells per circumference of organ</th>
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Fig. 4  Patient 2. Colon. Three adjacent crypts of Lieberkühn lined by primitive squamoid epithelium with prominent nucleoli. The more cellular crypt abscess contains neutrophil polymorphs, an eosinophil leucocyte, mononuclear cells and a few lymphocytes. H and E. Original × 1000.

Fig. 5  Patient 3. Colon. Early goblet cell differentiation in the right-hand crypt. Florid repair in the left-hand crypt has caused stratification at the base, with ballooning. The contents are equally neutrophil polymorphs, lymphocytes and cell debris. The surface bears non-differentiated columnar cells, and goblet cells are absent. H and E. Original × 1000.

can be misleading. Histology of the gastric mucosa showed no parietal cells in half the subjects, reinforcing ideas of Hurst in 1934\textsuperscript{14} and others\textsuperscript{15–18} that patients with hypo- or anacidity, or with stomach operations are more susceptible than normal persons to food poisoning. The relative absence of acute gastritis is important.

The absence of swollen ulcerated Peyer’s patches and the paucity of acute inflammation in the small intestine are important but there were changes which were difficult to explain. Generalised enteritis was not recorded in any patient, only one showing focal enteritis, but in about half there was evidence of repair of the crypts and mucosal surfaces by a non-differentiated epithelium. The infection is occasionally choleraic\textsuperscript{19,20} a cholera toxin like enterotoxin has been reported recently,\textsuperscript{21,22} and three patients (nos 2, 3 and 5) had excessive fluid in the bowel to simulate paralytic ileus at necropsy. This discovery fails to explain the observations reported above, because there is no sloughing or ulceration of the small bowel mucosa on biopsy in true cholera.\textsuperscript{23} The small intestines of four patients (nos 4, 6, 8 and 9) showed no histological abnormality, and therefore a cholera like enterotoxin might have been present in these cases. The ‘enteropathy’ of four patients (nos 2, 3, 5 and 7) may be the consequences of the action of the cytotoxin which has been described in the last two years,\textsuperscript{24} and it is interesting that phage type 32 was responsible for the illness of these four patients (Table 1). This illness has been described previously as ‘salmonella gastro-enteritis’, but in the present study gastritis and enteritis were minimal. A more satisfactory term therefore may be salmonella food poisoning.\textsuperscript{13} It is noted that ‘infantile gastro-enteritis’ shows no gastritis or enteritis on
the author's experience
of
Typhimurium
type
dumb-bell
ulcerative
idiopathic
infection.

Colitis
murium.33
megacolon
illcerative
confused
infections12
in
only
age
Styphimurium
tations
serves
severe
enough
coli, commonly
but,
histology,
infections
may
have
the
below
however
6
Fig.
942
infection caused
pathologist
causes
Patients with
this
default
water
in
series,
but
in
patients
other
liver
appendicitis

42
in
this
department31
which
serves
medical
units
only.
Whether
or
not
salmonella
infections
may
cause
chronic
ulcerative
enteritis32
remains
uncertain.

Styphimurium
causes
greatest
histological
damage
in
the
colon.2–11
In
this
country
the
illness
may
be
confused
with
bacillary
dysentery,
or
idiopathic
ulcerative
coli.
Patients
with
idiopathic
ulcerative
coli
may
develop
superinfection
by
Styphimurium.33
The
gross
changes
in
the
colon
of
patients
of
this
series
were
minimal,
and
toxic
megacolon
can
occur
(patients
5
and
9).34

A
pathologist
should
be
able
to
distinguish
idiopathic
ulcerative
coli
from
Styphimurium
infection.
In
untreated
ulcerative
coli
the
inflammatory
cells
are
totally
neutrophil
polymorphs
with
dumb-bell
type
abscesses
with
one
part
in
the
crypt
of
Lieberkühn
and
the
other
in
lamina
propria.
In
the
author's
experience
the
crypt
abscess
in
Styphimurium
infection
is
never
of
dumb-bell
shaped
type.
Most
cells
are
neutrophil
polymorphs,
with
eosinophil
leucocytes,
lymphocytes,
plasma
cells
and
mononuclear
cells
or
macrophages.
This
appearance
is
indistinguishable
from
that
seen
in
Sonne
dysentery
in
Glasgow,
but
Flexner
dysentery
crypt
abscesses
may
involve
the
lamina
propria.
The
author
has
no
experience
with
campylobacter
infections
but
the
description
by
Price
et
al35
suggests
that
the
changes
resemble
those
of
Flexner
dysentery.

Superficial
fibrinoid
necrosis
of
the
colon
of
patient
1
is
unique
but
similar
areas
are
evident
in
the
colon
of
patient
6
and
the
appendix
of
patient
3
(Tables
4
and
5),
as
well
as
in
a
death
from
S
enteritis
infection
infra).
The
sequence
of
repair
in
other
colons
suggests
that
all
experienced
similar
damage.
The
inference
is
that
diffuse
superficial
fibrinoid
necrosis
is
the
earliest
lesion
in
the
colon,
and
is
distinct
from
pseudomembranous
coli.

The
changes
reported
in
this
series
of
fatalities
may
not
be
representative
of
survivors,
but
some
practical
generalisations
may
be
made.
Although
these
patients
had
a
single
episode
of
infection,
diarrhoea
settled
fairly
rapidly
after
admission
to
hospital.
The
colon
could
be
histologically
normal
by
12
days,
(patient
4,
Tables
1
and
4),
and
certainly
by
58
days
(patient
9). Colonial
inflammatory
activity
could
persist
for
19
days,
(patient
6,
Tables
1
and
4)
and
subtle
minor
activity
was
still
present
at
29
days,
(patient
8,
Table
4).
Thus,
histological
normality
may
take
six
weeks
to
return.
This
suggests
that
with
a
continuing
diarrhoeal
illness,
the
possibility
of
idiopathic
ulcerative
coli
should
not
be
considered
until
after
two
months
unless
a
biopsy
within
this
interval
shows
classical
histology
in
conjunction
with
several
negative
faecal
and
blood
specimens.
These
need
not
be
a
community
outbreak
of
food
poisoning,
as
isolated
cases
of
salmonella
infection
occur.

Histological
appendicitis
is
part
of
the
process.
Repair
is
not
as
advanced
in
the
appendix
as
it
is
in
the
colon
of
the
same
patient,
and
given
the
correct
circumstances,
appendicitis
requiring
operative
treatment
may
evolve.37–39

The
four
patients
reported
by
Story
and
Hanbury40
had
different
alimentary
tract
histology.
It
is
possible
that
polyarteritis
nodosa
contributed
more
to
the
pathology
of
their
case
1
than
the
authors
believed.
The
gross
and
histological
pathology
could
be
very
variable,
but
the
colon
appearances
were
not
changed
appreciably.
Their
patient
1
had
a
hospital
acquired
infection,
a
situation
that
has
become
more
common41
and
was
probably
the
case
in
patient
1
of
this
report.
Review
of
the
literature
disclosed
that
colonic
ulceration
(ulcerative
coli)
was
reported
in
some
studies,
but
none
recorded
acute
diffuse
ulcerating
coli.40
Appendicitis
was
identified
as
a
complication.40

Histology,
but,
when
associated
with
specific
types
of
Escherichia
coli,
commonly
shows
acute
ileitis.25–27
Styphimurium
infection
caused
‘gastro-enteropathy’
in
only
half
of
this
series,
but
in
contrast,
Yersinia
infections12
28–30
may
cause
enteritis
and
ileitis
severe
enough
to
need
laparotomy.
No
such
infections
have
been
seen
in
this
department31
which
serves
medical
units
only.
Whether
or
not
salmonella
infections
may
cause
chronic
ulcerative
enteritis32
remains
uncertain.

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may
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with
bacillary
dysentery,
or
idiopathic
ulcerative
coli.
Patients
with
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ulcerative
coli
may
develop
superinfection
by
Styphimurium.33
The
gross
changes
in
the
colon
of
the
patients
of
this
series
were
minimal,
and
toxic
megacolon
can
occur
(patients
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and
9).34

A
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ulcerative
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the
inflammatory
cells
are
totally
neutrophil
polymorphs
with
dumb-bell
type
abscesses
with
one
part
in
the
crypt
of
Lieberkühn
and
the
other
in
lamina
propria.
In
the
author's
experience
the
crypt
abscess
in
Styphimurium
infection
is
never
of
dumb-bell
shaped

Fig. 6 Patient 7. Colon. The crypts are deeper than normal, and possibly more numerous than normal, with a normal quota of goblet cells. There is a condensation of lymphocytes however in the lamina propria immediately below the surface layer, where the layer of fibrinoid necrosis may have existed. H and E. Original × 250.
Pathology of the alimentary tract in Salmonella typhimurium food poisoning

The author has gained experience from seven necropsies with S. aberdeen (1 case), S. brandenburg (1), S. enteritidis (1), S. heidelberg (2), S. panama (1) and S. virchow (1) infections and has examined colonic biopsies from patients surviving S. typhimurium infection. All had features similar to those reported here. Scanty gastric crypt abscesses showed enteritis only. S. enteritidis infection failed to show enteritis (or enteropathy), whereas the two examples of S. heidelberg food poisoning showed extensive small bowel damage as well as colitis. Thus, the combined series of 16 patients suggests that not all salmonellae carry the enterotoxin or cytotoxin and that yet other possible mechanisms have to be sought to explain the undoubted gastric and small intestinal clinical features. Superficial fibrinoid necrosis occurred in the colon with S. enteritidis as described above in patients 1, 3 and 6. The lack of gross features in the alimentary tract of all these patients is re-emphasised, and some patients with infective diarrhoea will inevitably be categorised as having minimal change colitis.

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