



Radiological diagnosis of pneumoperitoneum in children with typhoid intestinal perforation

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<https://dx.doi.org/10.4314/ecaajs.v22i2.7>

Data from this study were previously presented at the 27th International Congress of Pediatrics; August 24th–29th 2013, Melbourne Australia

Abstract

Background: Typhoid fever is a severe systemic illness caused by the gram-negative bacillus *Salmonella typhi* and transmitted by the faecal-oral route. This study sought to determine the value of plain abdominal and chest radiographs in detecting pneumoperitoneum in children with typhoid intestinal perforation (TIP).

Methods: A retrospective review of plain abdominal and chest radiographs of children who had surgery for TIP between June 2009 and December 2011 in our unit. All the films were reviewed by the same radiologist who was blinded to the intraoperative findings, for the presence or absence of various signs of pneumoperitoneum. Data were collected on a structured questionnaire and analysed using SPSS version 15.0.

Results: Radiographs of 54 children were reviewed. Their ages ranged from 3 years to 13 years (median 7 years). Thirty-three of them were boys and 21 were girls (male-to-female ratio 1.57:1). Pneumoperitoneum was detected in 47 patients (87%). In the erect abdominothoracic films, air under the right hemidiaphragm was detected in 16 of 41 cases (39%), and extraluminal or intraperitoneal air-fluid levels in 31 of 41 cases (75.6%). In the supine abdominal films, the commonest sign of pneumoperitoneum was the right upper quadrant gas sign (23 of 50 cases; 46%).

Conclusions: A careful interpretation of plain abdominal and chest radiographs in the child suspected to have TIP, would lead to more accurate diagnosis of pneumoperitoneum.

Keywords: pneumoperitoneum, typhoid intestinal perforation, radiographs, children, air-fluid levels

Introduction

Typhoid fever is a septicemic illness caused by the gram-negative bacillus *Salmonella typhi*, and constitutes a major public health challenge in developing countries.^{1,2} It is estimated that perforation of the intestine occurs in 2% to 4% of all cases of typhoid fever, and that this is the cause of mortality in about 25% to 40% of patients afflicted with this disease.³ Once perforation has occurred, the resultant peritoneal contamination and peritonitis will depend on the duration, number, location, and size of perforation.⁴ Perforation is heralded by exacerbation of abdominal pain association with tenderness, rigidity, and guarding, most pronounced over the right iliac fossa; experience and a high index of suspicion may however be required for some patients in a



severe toxic state, and for children under 5 years, in whom these signs may be obscured, with resultant delays in diagnosis and institution of appropriate treatment.^{4,5}

Since the early part of the 20th century, erect or left lateral decubitus films of the abdomen have been shown to be valuable for the detection of pneumoperitoneum in patients with suspected perforation of the gut.⁶⁻⁸ In clinical practice, however, many patients with an acute abdomen are too sick or debilitated to stand erect or lie on their side for the time necessary to permit air to migrate to the least dependent portion of the peritoneal cavity, such that supine abdominal radiographs may be the only films feasible.^{7,8} Signs indicating the presence of pneumoperitoneum are based on direct identification of peritoneal ligaments and other structure which are only visible when outlined by air.^{8,9}

This study was aimed at determining the value of plain abdominal and chest radiographs in detecting pneumoperitoneum in children presenting with typhoid intestinal perforation in our hospital.

Methods

We retrospectively reviewed the plain abdominal and chest radiographs of children who were admitted into our ward and had surgery for typhoid intestinal perforation between June 2009 and December 2011. All the films were retrieved from our archives and were reviewed by the same radiologist who was blinded to the intraoperative findings. The films were evaluated for the presence or absence of various signs of pneumoperitoneum. In all there were 41 erect abdomino-thoracic films, 50 supine abdominal films, 5 left lateral decubitus films, 16 erect chest films, and 10 supine chest films.

Data were collected on a structured questionnaire and analysed using SPSS version 15.0. Descriptive statistics were computed for continuous variables, while proportions were used for categorical variables. The study was approved by the research and ethics committee of our hospital.

Results

A summary of the results is as depicted in Table 1.

Radiological signs on erect abdomino-thoracic films

Air under right hemidiaphragm: This is visualised as a radiolucent area separating the right hemidiaphragm above from the dome of the liver below (Figure 1). This was present in 16 of the 41 erect abdominothoracic films (39%).

Air under left hemidiaphragm: Here, a radiolucent area is seen separating the left hemidiaphragm above from the fundus of the stomach below. This was seen in 11 of the 41 of the erect abdominothoracic films (26.8%).

Extraluminal or intraperitoneal air–fluid levels: This appears as an air–fluid interface between a radiolucent region of intraperitoneal gas collection above and a radio-opaque region of intraperitoneal fluid collection below (Figure 2). This was noted in 31 (75.6%) of the erect abdominothoracic films.

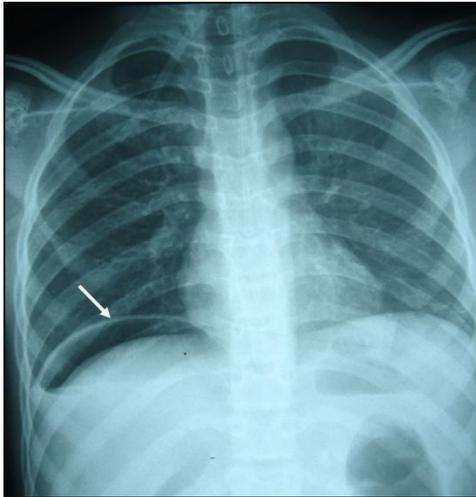


Figure 1: Erect abdominothoracic radiograph showing air under the right hemidiaphragm (arrow)



Figure 2: Erect abdominothoracic radiograph showing massive intraperitoneal air and fluid collection with an air–fluid interface (arrows)



Figure 3: Supine abdominal radiograph showing right upper quadrant gas sign (arrow)

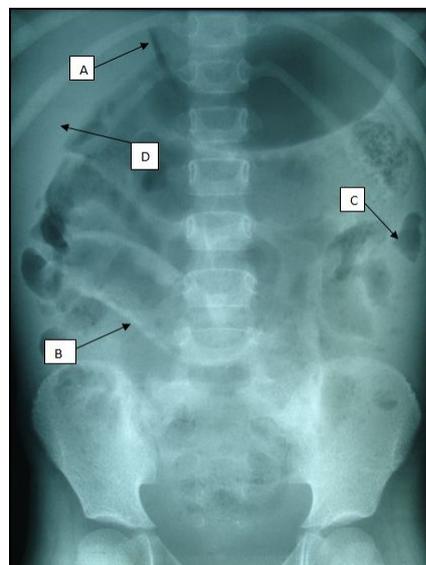


Figure 4: Supine abdominal radiograph showing: A. Air in fissure of ligamentum teres sign; B. Rigler's sign; C. Triangle sign; D. Liver edge silhouette sign.

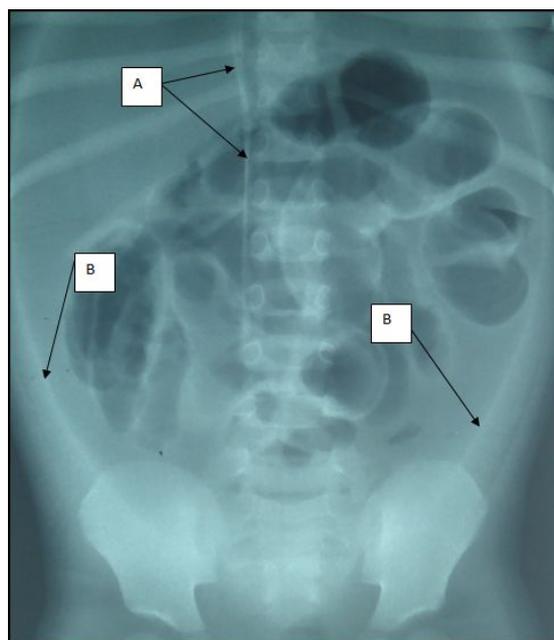


Figure 5: Supine abdominal radiograph showing: A. Falciform ligament sign; B. Football sign

Radiological signs on supine abdominal films

Right upper quadrant gas sign: This appears as a radiolucent pocket (localised gas) in the right upper quadrant.^{7,10} This was visualised in 23 of the 50 supine abdominal films (46%) (Figure 3).

Liver edge silhouette sign: This is visualised as a lucent crescentic area against the liver edge.¹⁰ It was seen in 12 (24%) of the supine abdominal films (Figure 4).

Air in fissure of ligamentum teres sign: This appears radiographically as a vertically oriented, sharply defined, slit-like or oblong area of hyperlucency found about 2.5 to 4 cm right of the vertebral border, between the tenth to twelfth ribs.¹⁰ This was seen in 22 (44%) of the supine abdominal films (Figure 4).

Triangle sign: This appears as a sharply defined triangular area of lucency, extraluminal in location.¹⁰ It was visualised in 2 (4%) of the supine abdominal films (Figure 4).

Falciform ligament sign: Here, gas outlines the falciform ligament, which is radiographically apparent as a vertical band of soft tissue parallel to the right border of the spine.^{7,10} This sign was seen in 9 of the 50 supine abdominal films (18%) (Figure 5).

Football sign: Radiographically, this appears as a huge oval shadow outlining the periphery of the peritoneal cavity.^{7,10} This was visualised in 12 (24%) of the supine abdominal films (Figure 5).

Other signs: The inverted V sign (gas outlining the lateral umbilical ligaments),^{7,10} and the anterosuperior oval sign (single or multiple avoid lucent areas, usually over the medial aspect of the liver),¹⁰ were not seen in any of the supine abdominal films.



Figure 6: Intraoperative photograph of intestinal perforation (arrow) in a child with typhoid fever. Note the dilated loops of bowel.

Table 1: Summary of radiographic findings in 54 cases of pneumoperitoneum in children, resulting from typhoid intestinal perforation

Sign	No. of cases	No. of films	%
Erect abdomino-thoracic films			
Air under right hemidiaphragm	16	41	39
Air under left hemidiaphragm	11	41	26.8
Extraluminal/intraperitoneal air–fluid levels	31	41	75.6
Supine abdominal films			
Right upper quadrant gas sign	23	50	46
Liver edge silhouette sign	12	50	24
Air in fissure of ligamentum teres sign	22	50	44
Rigler’s sign	16	50	32
Triangle sign	2	50	4
Falciform ligament sign	9	50	18
Football sign	12	50	24
Inverted V sign	0	50	0
Antero-superior oval	0	50	0

Discussion

Typhoid fever is a severe systemic illness transmitted by the faecal–oral route. After ingestion in food or water, *S. typhi* travels to the stomach, from where it goes to the small intestine, adheres to the mucosal cells, invades the mucosa and is subsequently transported across the intestinal



epithelium to the underlying lymphoid tissues.^{11, 12} Invasion of these lymphoid tissues by *Salmonella* results in a massive inflammatory response, including the recruitment of neutrophils and macrophages into the infected organ. Macrophages ingest the bacilli, and rather than killing them, the bacilli multiply within the macrophages.^{1,12} Bacilli laden macrophages and other free typhoid bacilli are drained into the mesenteric lymph nodes and subsequently into the blood stream via the thoracic duct.^{1,11} Humans do not react clinically to the initial invasion step, as the invading *S. typhi* may follow a simple default pathway favoring limiting activation of the inflammatory response.¹⁴ As a result of this silent primary bacteraemia the pathogen reaches an intracellular haven within 24 hours after ingestion throughout the organs of the reticuloendothelial system (spleen, liver, bone marrow), where it resides during the incubation period, which usually spans between 8 and 14 days, although this may decrease due to a large quantity of inoculum, or due to host factors.¹ At the end of the incubation period, the typhoid bacilli are released into the blood stream to invade the liver, spleen, bone marrow, gall bladder and the intestines, with this secondary bacteraemia heralding the onset of clinical signs and symptoms.¹² Clinical illness is accompanied by a fairly sustained but low level of secondary bacteraemia (about 1 to 10 bacteria per mL of blood).¹

The primary site of *Salmonella* infection occurs at specialised microfold or M cells, which are interspersed among the enterocytes covering the follicle associated epithelium (FAE) of the Peyer's patches (PP).¹¹ Besides PP, M Cells have been described in the FAE of small lymphoid aggregations, termed solitary intestinal lymphoid tissues (SILTs) that exist in the small intestine and colon of numerous species, including mice and humans and constitute another entry route or *Salmonella*.^{11,13} It has been shown that the frequency of SILTs invariable ranges from 40 to 60 structures per cm² throughout the murine small intestine, resulting in more than 1000 SILT structures in the intestine.¹³ Evidence also suggests that SILTs can be important in *S. typhi* infection in humans, as both PP and SILTs displayed inflammation in a study of typhoid patients.^{11,15}

Following the onset of secondary bacteraemia, disseminated *Salmonella* show a tropism for tissues that contain a high number of phagocytic cells, such as the spleen, liver, bone marrow and intestinal lymphoid tissues, with the bacteria inducing further recruitment or expansion of monocytic and macrophage cells and enlargement of these structures.^{11,13} During the first week of the clinical illness when the temperature is rising, and abdominal tenderness and bowel symptoms may develop, the Peyer's Patches (PP) in the distal ileum and the SILTs in the terminal ileum and proximal colon become hyperaemic and hyperplastic.^{3,13} Collectively, a total of several hundred inflammatory foci are present in the entire intestinal mucosa of *Salmonella*-infected mice outside of the PP.¹³ The maximum inflammatory changes occur in the lymphoid tissues about the tenth day of illness, and may resolve or may progress to necrosis and ulceration.³ The diseased gut is characterised by diffuse nonspecific enterocolitis with hypertrophy, necrosis and ulceration of intestinal and mesenteric lymphoid tissues.⁴ It is commonly believed that in those who perforate (about 2% to 4% of all cases of typhoid fever), necrotic ulcerating tissues usually slough off at the end of the second week or during the third week of the clinical illness, and that it is at this time that perforation and haemorrhage occur.³ A short duration of illness (median 9 days) however, has been reported by various workers from various regions of the world for patients who develop intestinal perforation from typhoid fever.^{2,15-18} Perforations may be multiple (3% to 40% of series), especially in younger children.⁴ Nasir et al., had earlier documented the occurrence of 32 perforations in 1 of their patients.¹⁹ The ulcers and perforations characteristically take the shape and positions of PP, being oval in the long axis of the bowel on the antimesenteric margin of the distal ileum, or may involve the SILTs which are smaller and may be round or oval in the long or transverse axis of the bowel.^{3,11,13}



In contrast to other forms of small bowel perforation which produce little free intraperitoneal gas because of the paucity of gas in the small intestine at the time of perforation, the small intestine is distended with gas prior to perforation in typhoid fever, leading to massive pneumoperitoneum upon intestinal perforation.³ Small intestinal dilatation and a large amount of free intraperitoneal gas was common among our patients (Figure 6). Although subdiaphragmatic gas on an erect chest or abdominal radiograph usually indicates the presence of a gastrointestinal perforation, this sign is present in only 60-80% of cases.¹⁰ Due to the mobility of the gas in the peritoneal cavity, it will only be possible to determine that a perforation exists and not to localise the perforation to any particular part of the gastrointestinal tract.⁶ Unless a considerable amount of free air is present in the abdomen, supine abdominal radiographs are generally thought to have limited value in diagnosing pneumoperitoneum.⁷ Although the erect chest radiograph and lateral decubitus views are far more sensitive at detecting pneumoperitoneum than a plain supine film, frequently only a supine abdominal radiograph may be available for examination especially in critically ill patients such as the child with typhoid intestinal perforation.^{8,10,20} In this study, combining the radiographic signs of pneumoperitoneum from both erect abdominothoracic radiographs and supine abdominal radiographs, pneumoperitoneum was detected in 87% of children with typhoid intestinal perforation. Earlier studies in adults with intestinal perforation resulting from typhoid fever have shown that pneumoperitoneum could be detected in between 50% 80% of the patients' erect abdominal thoracic radiographs.^{2,3}

In our study, the most common sign of pneumoperitoneum seen on supine abdominal radiographs was the right upper-quadrant gas sign (46%). This is an agreement with the findings of some workers who believe that it is probably the single best sign of pneumoperitoneum on supine films.^{7,21} The second most frequent sign of pneumoperitoneum on supine abdominal radiographs in our study was "air in fissures of ligamentum teres sign" (44%), which is at variance with the findings of Levine et al., in which the Rigler's sign which also occurred in 32% of their cases was the second most frequent.⁷ The large volume of intraperitoneal fluid collection in some of our typhoid intestinal perforation patients could have made it difficult to visualise the Rigler's sign in which the wall of the intestine is defined when air is present both in the lumen and outside the lumen of the bowel.^{6,7,10,22} With regards to the right upper-quadrant gas sign, it has been said that the linear collections represent gas in the right sub-hepatic space, whereas the triangular collections represent gas in the posterior recess of the right sub-hepatic space, just medial and inferior to the eleventh rib, in an area known as the Rutherford Morrison pouch or the hepatorenal fossa.^{7,10,21}

Given that small intestinal dilatation is a fairly frequent finding in perforated and non-perforated typhoid fever patients, its presence on plain abdominal radiographs may help distinguish this disease form other abdominal conditions.³

A major limitation of this study is that preoperative diagnosis of typhoid intestinal perforation was primarily based on clinical features of a history of fever followed by abdominal pain, distension and tenderness. It was supplemented by radiological findings of pneumoperitoneum and intra-operative findings of an oval perforation disposed longitudinally on the anti-mesenteric border of the jejunum or ileum or between the taenia coli in the colon, as well as an acutely inflamed and oedematous intestine with intra-peritoneal soiling.² Blood cultures for *S. typhi* were not done in any of the patients due to the non-availability of this procedure in our laboratory.



Conclusions

Our study has shown that the combination of different types of plain abdominal and thoracic radiographs improves the detection of pneumoperitoneum in children with typhoid intestinal perforation.

It is however worthy of note that although the radiographic detection of pneumoperitoneum remains a reliable sign of bowel perforation, its absence does not invalidate the diagnosis.

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