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I. ABSTRACTPRIMARY PERITONITIS

Abstrs. Ritchie & Leven.

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Historical

1842, Duparcque (ref. 13) reported 5 cases of essential peritonitis in girls 5 to 12 years old under "The Essential Peritonitis of Young Girls." Since then literature has consisted for the most part of analyses of series of cases, some limited to streptococcus and others to pneumococcus peritonitis. First comprehensive work on this subject dates back to Michaut (1901). (Ref. 12) Michaut first suggested haematogenous origin of this type of peritonitis.

In spite of interest in condition, there is still no definite proof as to mode of entrance and mortality is very great. Impression one obtains is that whatever decrease in mortality there has been, is due more to better understanding of treatment of peritonitis per se than to better understanding of etiology of this disease.

By term "primary" is meant type of peritonitis which develops suddenly and is unassociated with pre-existing abdominal inflammation.

It is difficult to separate (in the literature) the two main types, pneumococcus and streptococcus. Discussed together but their essential differences, of which there are some striking ones, will be brought out.

Incidence

One of most serious acute abdo-

inal emergencies of childhood. Lipschutz and Lowenburg (1) in discussing both forms state that they constitute 10% of abdominal emergencies in children. This seems rather high. McCartney and Frazer (6) (discussing pneumococcus type) state that it occurs in 2% of all abdominal emergencies in children. Joseph Schwartz (5) (discussing streptococcic peritonitis) states that in 400 cases of surgical disease in patients under 13 years incidence was 1.5%. Fricke (12) in Lenox Hill Hospital, N.Y.C. with 350 beds found there had been only 10 cases in last 5 years (1925-1930). There have been 2 cases at Minnesota General Hospital in last 12 months. Armstrong in 1903 found 5 cases of primary peritonitis in 102 autopsies. Osler in analysis of 102 earlier autopsies, 12 cases.

In spite of varying reports, one is impressed that the disease is not as uncommon as most believe and is an ever present challenge to one's diagnostic abilities.

Relative Frequency of Both Types.

In the literature more attention is focused on the pneumococcic type than on the streptococcic type. However, it appears that they are rather equally distributed. Duncan (13) reports 66 cases, 33 of which were due to the pneumococcus and 33 to streptococcus.

Lazarus (11) studied 42 consecutive cases of acute peritonitis in children occurring over a period of 3 years (1926-1929). They fell into 3 groups.

1. Pneumococcic - 9
2. Non-pneumococcic - 13 (probably streptococcic as he states that gonorrhreal tuberculous and other forms of peritonitis were not encountered).
3. Acute secondary suppurative peritonitis (ruptured viscus) - 20 cases. (Note high incidence of primary forms 1 and 2).

Zierold (8) however, reported 28 cases and of these 19 had bacteriological studies. Only one case of pneumococcic peritonitis was found and remainder were chiefly streptococcic.

Age Incidence

Disease has a predilection for children up to 14 years. Fricke (12) states that occasionally adults are stricken with pneumococcic peritonitis.

Rischbroth (57 cases) from 8 weeks to 9 years; McCartney and Frazer (6) (56 cases) all under 12 years majority 3 to 7 years; Schwartz (5) (14 cases) 3 weeks - 13 years, only 2 cases above 5 years. Heiman in 125 cases of generalized peritonitis found 15 cases of pneumococcic origin, 2 of which were adults. Fricke (12) reports 10 cases of pneumococcic peritonitis, 2 of which were in adults.

Duncan's 66 cases ranged from 10 weeks to 13 years.

50% were between 3 and 8 years. Of those under 3 years of age, 11 fell in streptococcic group and only 3 in pneumococcic, whereas 14 over 8 years were due to pneumococcic and only 6 to streptococcic.

From this it would seem that the streptococcic form is more common in younger children.

Sex Incidence

Duncan's statistics seem to conform other reports (13). 80% of 66 cases were in females, 90% of pneumococcic type were in females. 66% of streptococcic group were females. McCartney and Frazer (6) found no males in 44 cases of primary pneumococcic peritonitis.

In 22 cases, Lipschutz and Lowenburg reported all but 1 of pneumococcus cases were females, while all streptococcic cases were in males.

Taking all types, females seem to predominate, 3 or 4 to 1.

Etiology

The pneumococcus type I and S. haemolyticus are most common organisms found although the other types of pneumococci and non-hemolytic strepto-

cci have been reported.

The great problem has been, and still is, the mode of invasion.

Four main types of invasion have been considered.

1. Hematogenous route: Lowenburg and Lipschultz (1) are proponents of haematogenous infection. They had 9 positive blood cultures in 11 cases of pneumococcic peritonitis. They felt that the upper respiratory and gastro-intestinal tract were the portal of entry for the blood stream. Others support this view. On the other hand, Rischbreth found only one case of pneumococcic peritonitis in 6000 cases of pneumonia and Rolleston found 11 cases in 4454 cases of pneumonia which suggests improbability of hematogenic spread of organisms to the peritoneum. In 14 cases of streptococcic peritonitis reported by Schwartz (5), 4 had blood cultures and only 2 were positive. In spite of this, he feels that there is a transient bacteremia with localization in peritoneum. Individual susceptibility is brought out by some authors.

Jensen (ref., 12) recovered pneumococci from blood of animals within a few minutes after injecting the organism into the peritoneum of these animals.

The case for haematogenous route of invasion is not established.

2. Gastro-intestinal tract. The usual onset with diarrhea has been a sign for many authors which suggests that the gastro-intestinal tract is point of invasion. Some authors state that the infection is localized in the appendix. The most recent experimental work in this line is by Obadelek (10) who injected 13 guinea pigs with pneumococci, intraperitoneally, and 2 guinea pigs with streptococcic in like manner. Of the 13 developing pneumococcic peritonitis, 11 died and of the 2 with streptococcus both died. In all the deaths, there was found an edema on injection of the terminal ileum and also an inflammation of the appendix. The mesenteric glands in the region of the terminal ileum were enlarged, whereas there was

no other glandular enlargement. Rischbreth (7) on the other hand found that in pneumococcic peritonitis that the mesenteric glands were usually normal, a condition quite unlikely if the peritonitis was of enteric origin.

Jensen failed to produce peritonitis by feeding organisms to animals.

McCartney and Frazer (6) state that no lesions are found in the intestinal tract, i.e. no ulceration of mucosa.

There is certainly no unanimity of opinion on the gastro-intestinal tract as the point of invasion.

3. Lymphatics. Passage of infection from tonsillar, pharyngeal or bronchial glands down into the sub-peritoneal lymphatics is very unlikely in as much as the route would be in an opposite direction to the normal lymph flow. This route seems to have the least support.

4. Genital tract. This route of invasion seems to have more substantiating evidence clinically and experimentally although it cannot explain all the cases. Perhaps we lean more strongly towards this portal of entry because of the obvious higher incidence in females.

Duncan (13) took vaginal cultures in 11 of his 66 cases (6 pneumococcic and 5 streptococcic) and only 5 yielded the causative organism. On the other hand, he states that in those cases which are observed early and the peritonitis is not diffuse the inflammatory process is more extensive in the pelvis.

McCartney (3) was able to reproduce the disease in young female monkeys by vaginal inoculation. Summarizes evidence that infection reaches peritoneum from vagina by way of the genital tract.

- a. Occurs in young girls.
- b. Majority 3 to 7 years. (When vulvitis is more common and vaginal

secretion is neutral or slightly alkaline).

- c. More common in summer months.
- d. Girls belong to lower classes (not substantiated by Lazarus (11)).
- e. Frequently have vulvo-vaginitis. Vaginal secretion is neutral or alkaline, which is not so when vulvo-vaginitis is not present, in which case it is normally acid. Age incidence of valvi-vaginitis usually corresponds to age incidence of disease.

f. Laparotomy usually shows that disease has begun in the pelvis and in earlier cases is usually confined to pelvis. Many flaws can be found in the suggestion of the proponents of this type of invasion, the greatest one being the occurrence of the disease in males.

It is our impression from the review that the only one of the four suggestions which could account for all cases and which has more substantial proof, is a hematogenous type of invasion.

Source of Infection

The relative frequency of peritonitis following pneumonia has been mentioned. (1 in 6000 and 11 in 4,450).

Schwartz (5) showed preceding respiratory infection in 10 out of 14 cases of streptococcic peritonitis.

Lipschutz and Lowenburg (11) showed 90% of 22 cases of pneumococcic peritonitis had preceding respiratory infection.

Palmowitz (4) in 8 cases of streptococcic peritonitis had same experience.

Hamburger found 12 cases in epidemic of sore throat in Baltimore.

Zierold (8) in 28 cases had 10 cases with no known origin as accompanying infection in body, whereas remaining 18 cases were accompanied or preceded by other site of infection such as sore throat, rhinitis, pneumonia or erysipelas.

Fricke (12) states that 5 of the 8 children in his series, had a pertussis infection "sometime before their present illness".

The preceding statements recall the fact that in most instances primary peritonitis is really secondary to some other demonstrable infectious process.

Pathology

About 75-80% of the cases reported have come to autopsy.

The lesions are typical of a generalized peritonitis. The 2 types do not differ a great deal.

In the early cases, there is a sticky, rather shiny exudate and many authors report that it is limited to the pelvis.

In 24-72 hours, the lesion became fibrinous and then purulent. The pus of the pneumococcic type is described as being greenish-watery and odorless. Mischaud feels that the diffuse and localized types are due to different strains of the organism but other authors do not agree.

There is a tendency in some cases for abscess formation and localization at the umbilicus. (3 of our former cases in one family.)

Type I pneumococcus and haemolytic streptococcus are most frequent organism.

In the streptococcic type it is reported that the most advanced changes are found in the region of the cecum and appendix. This substantiates the findings by Obadelek (10) (see preceding) in artificial peritonitis where the most pronounced lesions found were in the terminal ileum and appendix. There have been cases in which the tubes have been particularly inflamed. Fricke (12) reports a case of an adult female 23 years old on whom a diagnosis of ruptured appendix was made and at operation free pus was coming from a bilateral pyosalpinx. Pneumococci were demonstrated in the pus from the right tube whereas the pus from the left tube and peritoneum was sterile.

Complicating conditions are found such as empyema, pneumonia, etc. but none of these can be proven to be primary to the peritonitis.

There is nothing in the pathological picture to substantiate a mode of invasion except a few observations of pelvic peritonitis in the early stage.

Symptoms

Lazarus (11) has compared the symptoms of 9 pneumococcic, 13 non-pneumococcic and 20 cases of acute secondary peritonitis due to a ruptured viscus. As acute ruptured appendicitis is one of most difficult differential diagnosis, this will be reviewed.

Clinically, it was found that the symptomatology between the 1st two groups, (i.e. the primary groups) and the severe cases of secondary acute diffuse suppurative appendicitis could not be differentiated.

The temperature usually started high at the onset 102-107 F. and remained elevated during the entire course with morning remissions of 1 to 2 degrees. Temperature as high as 109 F was recorded and when recovery did occur it dropped by lysis over a period of 7 to 14 days. On the other hand in the 3rd group (secondary suppuration), the temperature rarely went above 102 F.

Distension, except in secondary suppurative appendicitis was found sooner or later. Free fluid was elicited in only 10% while tympanities were present in 2/3 of the cases, whereas in the ruptured appendiceal group, this sign occurred in only one patient who recovered and all 6 who died.

Abdominal pain and tenderness present in all cases. In appendiceal type pain was more spasmoid and cramp-like in character. Most authors describe early pain in the pneumococcic type as being more in lower abdomen.

Abdominal rigidity occurred in less than 1/3 of pneumococcic and non-pneumococcic type, whereas in 80% of the appendiceal group there was definite

rigidity of the abdominal muscles. This is in accord with other authors in which they state that the abdominal rigidity is not marked and it is also mentioned in the pneumococcic type that the abdomen may even have a "doughy feel."

Abdominal localization occurred in 3/4 of the pneumococcic type, the favorite site being in the lower right inguinal and lower left inguinal and umbilical regions.

In the non-pneumococcic group, localization occurred in less than 1/3 of the group.

In the appendiceal group, localization appeared sooner or later in all.

Diarrhea was an inconstant symptom and occurred in only 1/3 of patients.

This is somewhat different from statements by other authors who state that, particularly in the pneumococcic type diarrhea is almost uniformly present.

Vomiting occurred in all of appendiceal type, in 85% of non-pneumococcic type and in 50% of pneumococcic type. This too is somewhat different from other reports of both pneumococcic and streptococcic types in which the vomiting is described as being early, constant, and an outstanding feature.

Toxemia was profound in majority of primary cases and in only 35% of the appendiceal group.

One obtains the impression from the literature that the toxemia of the streptococcic type is more rapid and profound than the pneumococcic type although the toxemia in the latter group is very marked.

Laboratory diagnosis

Besides blood culture and peritoneal cultures, there is little of a laboratory nature that helps in mak-

ing the diagnosis.

Lazarus states that the blood count is neither diagnostic nor prognostic. The average white blood count was 18,000. Other authors feel, however, that the white blood count is of importance in that the count in primary peritonitis is usually a great deal higher than in that of the secondary suppurative type and the differential diagnosis shows a polymorpho-nuclear frequency of about 90%.

Diagnosis

The problem is the recognition of primary peritonitis from peritonitis secondary to ruptured viscus.

Careful histories on children are essential in that a previous history of staphylococcus (abscess) or streptococcic infection (scarlatina, streptococcus, sore throat, erysipelas, etc.) is strongly indicative of associated infection by these organisms. Also a history of an upper respiratory infection is strongly suggestive of pneumococcic peritonitis.

There are several differences in the reaction of pneumococcic and streptococcic type.

1. Pneumococcic is usually milder in course, abdominal findings often less marked.
2. Pneumococcic has more tendency to localize than streptococcic.
3. Pneumococcic prognosis is better.
4. Pneumococcus is found chiefly in girls and in lower classes (?).

Differential diagnosis

1. Acute perforative peritonitis.

- a. History of primary focus, gastric or duodenal ulcer, gall-bladder disease, etc.
- b. Onset severe; pain localized at site of perforation.
- c. Temperature first normal or subnormal. Rigidity localized at first.

2. Acute gonorrhreal peritonitis.

- a. Previous symptoms and signs referable to uterus and adnexae.
- b. May have a positive vaginal smear.

- c. Rigidity not marked.
- d. Symptoms abate in 24 hrs.
- e. Majority recover.
- f. Exudate chiefly in pelvis.

3. Typhoid peritonitis.

- a. Occurs late in disease.
- b. Large spleen.
- c. Positive widal.
- d. Blood culture, etc.

4. Tuberculosis.

Often extremely difficult except it has a milder course. Resembles localized cases of pneumococcic peritonitis.

Prognosis

Pneumococcus.

Rischbreth - 88% mortality in 54 cases; 33% of localized cases.

McCartney and Frazer - 65% mortality over 20 years; 53% in 1920; 42% in 1921.

Jensen - 90 cases, 64% mortality.

Konnecke - 27 cases, 55% mortality.

Streptococcus.

Lipschutz and Lowenburg - 9 cases, 100%.

Schwartz - 14 cases, 79%.

Rabinowitz - 8 cases, 75%.

Zierold - 22 unoperated cases, 100%, 6 operated cases, 66%.

Lazarus states that in his series the streptococcus group offered the worst prognosis with an 85% mortality. The pneumococcus group mortality, about 66%. He noted that the pneumococcus group lived on an average twice as long as the non-pneumococcic group.

Duncan - mortality

Pneumococcic - 3 cases under	
peritonitis	3 years of age - 33%

17 cases,	
3 - 8 yrs.	- 62%

14 cases,	
8 - 13 yrs.	- 57%

General	- 57%
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Duncan - mortality (cont.)

Streptococcic - peritonitis	11 cases under 3 yrs. of age	- 100%
	16 cases, 3 - 8 yrs.	- 87%
	6 cases, 8 - 13 yrs.	- ?%
	General	- 80%

Treatment

Fricke (12) gives good summary of the treatment accepted by most authors.

Recommends operative treatment when an abscess has localized and is well walled off.

The only question about which there can be any doubt is the early cases.

The mortality of the cases operated upon in the first few days is about 90%. Because of this high mortality in the earlier stages the concensus of opinion in the last few years has swung towards waiting in these few cases. American surgeons such as Kahn, Meredith, Syms, Lilienthal, Bevan, all favor this and the German surgeons (Strauss, Budde, Salzer) also condemn early operation. It is their opinion that early operation is contra-indicated because in earlier stages it is a systemic generalized infection and operative interference can do more harm than good.

Those who favor early operation do so because of the difficulty of differentiating from a ruptured appendix. It seems, however, that since the non-operative treatment for ruptured appendix is becoming more popular that the early operation of pneumococcic streptococcic peritonitis will be less frequent.

Abdominal puncture is indicated for diagnostic purposes in suspected cases. Those who operate usually drain the abdomen (which cannot be done?)

Irrigation with saline, ether, diluted bile, optochin and rivanol have had little effect. Some attempt vaginal drainage.

Sepsis has been treated with optochin,

horse serum, autogenous blood and pneumococcic serum injection. This latter has proved of definite value.

Besides operative treatment, all supportive measures possible are indicated.

Impression

1. Primary peritonitis is not uncommon.
2. Pneumococcic and streptococcic types are about equally divided. (Both of our cases are streptococcic type.)
3. Pneumococcus type apparently more discussed in the literature.
4. Disease has a predilection for children up to 14 years with greatest incidence between 3 to 8 years. Prevalence in females roughly 3 or 4 to 1.
5. Route of invasion undetermined. Hematogenous route seems to be the only one of 4 suggested that could explain all cases.
6. Cases usually show same signs of infection elsewhere preceding peritonitis.
7. Mortality is extremely high in streptococcus group. Reported mortality in pneumococcus type is lower.
8. Operation seems to make little difference in early lesions.
9. Concensus of opinion is toward non-operative treatment of early cases.
10. Our 2 cases are good examples of streptococcus type, one with preceding inflammation outside the peritoneum and the other marked inflammatory reaction of the pelvic organs.

II. CASE REPORTPRIMARY STREPTOCOCCIC PERITONITIS. (GENITAL FOCUS?)

Path. Koucky.

Case is white, female infant, 6 years old, admitted to Minnesota General Hospital 10-25-32, expired 10-29-32 (4 days).

Acute Onset

10-21-32 - Came home from school complaining of headache, malaise and abdominal pain.

10-22-32 - Developed severe diarrhea.

Abdominal complaints

10-24-32 - Diarrhea and abdominal pain continued. Stools contained mucus but no blood. Pain remained generalized but was somewhat intermittent in character. Accompanying these symptoms, there has been fever, flushed face and patient appeared very ill. No vomiting but nausea present.

10-25-32 - Admitted. Past history - No special note.

Physical examination

Throat - moderately injected with post-nasal discharge. Chest - normal. Abdomen - distension, rigidity, tenderness and rebound tenderness present. Genitalia - vulvae quite red and excoriated. Rectal - no masses made out.

Laboratory

Urine - 1.025, light cloud of albumen, few wbc's. Blood - Hb. 65%, rbc's 4,000,000, wbc's 8,850, Pmn's 80%, L 20%. Widal - reaction absent, B. paratyphosus A. absent, B. paratyphosus B. absent. Remarks - agglutination with B. dysenteriae Flexner present 1:100, agglutination with B. dysenteriae Shiga present 1:400. culture from vagina on blood agar - streptococci. Blood culture - long and short chains of pleomorphic gram positive cocci in broth. 5 colonies of hemolytic gram positive streptococci per 1 cc. blood on blood plate.

Progress:

Through 4 days stay in hospital, temperature ranged between 103 and 106. Pulse from 130 to 180. Respirations irregular, between 35 and 60. Given Hartman's solution and glucose.

Therapy

10-27-32 - Appears somewhat brighter. Abdomen shows about same condition. Hartman's solution subcutaneously. 250 cc. citrated blood intravenously. 3 A.M. - Appears much worse. Respirations irregular. Through remainder of day, condition better and worse.

10-28-32 - Condition unchanged. Con-

tinuous hot packs applied to abdomen. Paraoral fluids. Stimulants given.

Exitus (4th day)

10-29-32 - Same therapy carried out. Becoming weaker. Cyanosis, difficulty in respirations set in. 9:05 P.M. - Expired.

Autopsy

Examination limited to incision in abdomen with visual examination only. No permission for removal of organs.

Peritoneal exudate

Lengthy, abdominal incision made. Peritoneum incised. 600 to 800 cc. yellowish fluid (slightly turbid but on whole fairly clear) released from abdomen. In dependent parts of abdominal cavity and in pelvis there are masses of yellowish-green, gelatinous, fibrinous material. Abdominal viscerae examined without removal with following points in mind. (1) Perforation of hollow viscus, (2) acute appendicitis, (3) Meckel's diverticulitis, (4) diverticulitis of colon, (5) acute salpingitis. No evidence of any of these conditions present except a possible salpingitis. Bowel very markedly injected. Small amount of greenish fibrin in spaces between coils of bowel but no evidence that any of this involvement is other than part of the generalized peritonitis.

Pelvic genitalia:

On both sides, tubes are edematous, bluish-black, appear coiled upon themselves and held in this position by older fibrin. Ivaries appear to be matted in this coil. Uterus appears edematous. All of adjacent peritoneal surface and sub-peritoneal tissue is of bluish-black color. On incision, subperitoneal tissue is edematous with a greenish color. Involvement of internal genitalia appears much more extensive than in any other portion of abdomen. Adjacent rectum and sigmoid colon does not have this bluish-black discoloration. It is assumed that internal genitalia is a focus for the primary peritonitis. Biopsies of tubes, ovaries and uterus taken.

Viscera

Left kidney lifted from bed. Capsule strips easily. Kidney markedly swollen, very pale and has boiled appearance. Biopsy taken and pedicle ligated. Spleen large for size of body and feels very soft. It is not incised. Liver yellowish and swollen. Edges are rounded. Gall-bladder is peculiar, waxy, white, edematous, swollen organ. Wall seems to be thick and bile does not appear to be present in the lumen as far as can be observed from exterior. No further examination done. Cultures of abdominal fluid show streptococci in short and long chains.

Diagnoses

1. Primary streptococcic peritonitis, possible focus in internal genitalia.
2. Acute splenitis.
3. Cloudy swelling and fatty change of liver.
4. Cloudy swelling of kidneys.
5. Acute edema of gall-bladder.

III. CASE REPORTPRIMARY STREPTOCOCCIC PERITONITIS,
(THROAT FOCUS?)

Path. Koucky.

Case is white female, 3 years of age, admitted to Minnesota General Hospital 3-19-33, expired 3-23-33 (4 days).

Sore throat

3-14-33 - First complained of sore throat. Restless, irritable and appeared to be feverish. Vomited 3 times today.

3-15-33 - Fever persists. Poor appetite.

3-16-33 - Condition seemed better but still somewhat feverish.

Abdominal pain

3-19-33 - Complained of pain in abdomen. Still has fever. No vomiting since first day of illness. Constipated. Scanty urine. Seen by physician throughout illness who made diagnosis of peritonitis and advised hospitalization. Past history: Felt well before onset of previous illness.

Admitted - 5th day

Physical examination: Temperature 104 (R). Listless and flushed. Reflexes -

slightly hyperactive. Some crusting about mouth. Chest - normal. Abdomen - Slight distension with some tympanitis; liver and spleen not palpable; not very tender; no rigidity; no apparent rebound tenderness.

Surgical consultation:

Muscles of abdomen held rigidly; apparent tenderness present in all 4 quadrants but possibly more so in right lower quadrant; no rebound tenderness elicited. Rectal examination - shows questionable tenderness.

Laboratory

Blood - Hb. 75%, rbc's 4,000,000, wbc's 16,500, Pmn's 58%, toxic granulation in polymorphonuclears and marked shift to left. Urine - light cloud of albumen, numerous granular casts, few wbc's.

Packs

3-20-33 - Hot packs placed on abdomen continuously. Temperature frequently runs over 103.5. Tepid sponges given for temperature. Listless, pale and drowsy. Had one small, greenish, fluid emesis. Fluids forced, orally and paraorally.

Blood - suction

3-21-33 - Condition same. Abdomen very much distended. 150 cc. citrated blood given. Nasal suction started. Temperature up to 105. Pulse 152. Respirations up to 65. Abdominal findings changed somewhat - definite distension noted, difficult to localize tenderness although undoubtedly diffuse abdominal tenderness present. Rebound tenderness not made out. Less prominent rigidity noted, not localized. No masses present.

X-ray of abdomen

Shows tremendous distension of all loops of bowel, both small and large, and also the stomach with gas. Clinical impression: Peritonitis, probably primary; may be secondary to appendicitis.

Streptococcus found

3-22-33 - Blood culture - positive

for streptococcus. Treatment carried out as before. Another transfusion of citrated blood. Nasal suction continuously. Temperature continues as before.

Exitus (9th day)

3-23-33 - Child weaker. Temperature ranges to 105. 90 cc. citrated blood given. 9:30 A.M. - Resting more quietly. 100 c.c. citrated blood given at 3:30 P.M. Temperature progressively becomes worse. Catheterized urine sterile. 8:11 P.M. - Expired.

Autopsy

External examination

Body that of well-developed and nourished, white female, 3 years of age, measuring about 95 cm. in length and weighing approximately 40 lbs. Rigor present. Hypostasis purplish and posterior. No edema, cyanosis or jaundice. The pupils are equal, each measuring 3 mm. in diameter. Numerous antecubital punctures present. Similar punctures in the thighs. A puncture wound from an abdominal paracentesis present. A great deal of brownish discharge oozes out of the mouth and nostrils. Subcutaneous fat abundant in amount.

Exudate

Peritoneal Cavity. All of peritoneal spaces are filled with thin, greenish fluid in which there is a great deal of fibrin. In culdesac, this fluid is frankly purulent. Appendix hangs free, slightly injected (undoubtedly secondary to general peritoneal process rather than primary appendicitis).

Exudate

Pleural Cavities - filled with about 500 cc. of yellowish, turbid fluid containing a great deal of fibrin. Pericardial Sac contains a definite excess of fluid which is clear and straw-colored.

Early vegetations?

The Heart weighs 90 grams. On both leaves of the mitral valve, there is reddening and little irregularities about 2 to 3 mm. from edges of valve. These irregularities do not disappear on stretching valve. No thrombosis or ulceration present over these irregular areas. They may be very early vegetations?; A few small petechial hemorrhages are noted under the epicardium of the

heart, chiefly on the right side. The Root of the Aorta and coronaries are smooth and clean.

Collapse

Right Lung weighs 150 grams, Left 140. There is compression collapse (may also be partially active atelectasis) involving lower lobe and posterior part of upper lobe. These areas are bluish in color, quite heavy and on cut surface the lung is red, relatively dry but no areas of induration or nodules can be palpated within substance.

Splenitis, Cloudy swelling

Spleen weighs 75 grams, is slightly enlarged in proportion to other organs, and it is very soft.

Liver weighs 600 grams. The edges are founded. It is extremely soft. The liver markings appear to be wiped out. The entire liver substance is yellowish.

Gall-bladder has a thin wall and contains bile. No stones. Ducts are patent.

Gastro-Intestinal Tract. No areas of localized inflammation. No areas of ulceration or reddening in bowel. No malformation observed.

Pancreas is small and soft. No tumors or cysts noted.

Adrenals are well formed and show no hemorrhages or adenomata.

Each Kidney weighs 70 grams. Kidneys are swollen and very red. The cut surface is cloudy. The pelves show fine petechial hemorrhages under the mucosa.

Bladder is reddened at the trigone and shows no definite cystitis or malformation.

Focus?

Genital Organs: Entire retroperitoneal tissue and pelvic retroperitoneal tissue is swollen and edematous. This process continues into adnexae. Exclusive of this generalized edema, there is no characteristic change about adnexae to suggest primary focus in these organs. Tubes are not indurated and their color is approximately that of surrounding tissue. Uterus is small and its

cavity contains no pus. No pus can be expressed from the tubes.

The Aorta is soft and smooth.

There is a large plaque of Lymph Nodes in retroperitoneal tissue surrounding the abdominal aorta which is edematous and gelatinous. On left side, they appear to have broken down into frank pus which has extended along psoas muscle for 2 to 3 cm. The mediastinal lymph nodes are enlarged in a similar manner. The lymph nodes of the great mesentery are slightly hyperplastic.

Head and Sinuses - examination not permitted.

Cultures

Blood culture (taken during life) - showed a streptococcus (no other information given).

Cultures (taken postmortem) from peritoneal, pleural and pericardial cavities: Cultures from the peritoneal and pleural cavities show a pure culture, and that from the pericardium shows an admixture of a white staphylococcus in addition to other organisms. The preponderant organism is very small, pale, hemolytic coccus which grew with difficulty on solid media but grew readily in the fluid media. It formed long chains of cocci which appeared to be so characteristic of streptococcus. No further differentiation done.

Diagnoses

1. Upper respiratory infection (clinical).
2. Primary peritonitis (organism - hemolytic streptococcus).
3. Bilateral empyema.
4. Pericardial effusion.
5. Early endocarditis (?).
6. Splenitis, acute.
7. Cloudy swelling of liver and fatty change.
8. Cloudy swelling of kidneys.
9. Retroperitoneal and mesenteric lymphadenitis.
10. Suppurative lymphadenitis, (retroperitoneal).
11. Petechial hemorrhages in epicardium and kidney pelvis.
12. Compression atelectasis of lung.

IV. MEETING

Date: March 23, 1933

Place: Interne's Lounge, 6th Floor, West Building

Time: 12:14 to 1:10

Program: Undulant Fever

Present: 95

Discussion: W. T. Peyton
E. Lardigabal
H. A. Rieman
O. McDaniel
C. P. Fitch
W. L. Boyd

Theme: W.T.P.: We have recognized the necessity of gently handling tumors in this Clinic. I do not believe a rule against internes handling patients is necessary. Patients are liable to traumatize the breast as much as an examination. We object to taking breast and squeezing it in hand. I think it is essential we teach internes the proper way of handling tumors and not to forbid them to do it.

E.L.: It is an honor for me to come here before you today. I would like to say a few words about our medical profession and also my impression of the profession in the states. I have come here for more experience in surgery because in the Islands we do not progress as much in this line. We do not have the modern facilities you have here, besides there is really more material in a big country like this. In the Phillipine Islands we have only two big hospitals in which surgical work is done. More advances in the U.S. and more facilities. I hope sometime in years to come we will be on the same level as the surgeons of the states. We are willing to learn. Thank you very much.

H.A.R.: In the last 5 years there has been no formal discussion

of undulant fever. This meeting is therefore timely. One sees in Ohio and Iowa where the disease is most prevalent that interest has been stimulated by education, and the disease is recognized there. Most of the older observers stated that undulant fever was disease of very obscure symptoms. If one has the disease in mind it is possible to pick it out easily when typical. Chief things to be noted: Weakness, sweating, prostration interspersed with sense of well being. The patients work under pressure. With these few things in mind it is possible to pick out disease without assistance of laboratory. No other disease, however, is so dependent upon laboratory for final diagnosis. First is skin test which has mislead us - positive when disease not present and negative when it is present. Other laboratory tests are difficult. Tried to obtain positive blood cultures but unsuccessful, as it takes good deal of experience. Organisms require special handling (carbon dioxide) sometimes. Secretion in which organisms can be isolated is urine. Stool much more difficult. Agglutination test also difficult without experience. These are sent to State Board laboratory.

Certain complications to recognize. Frequently affects genitalia in both male and female. Some patients admitted with orchitis, diagnosed incorrectly, ultimately turn out to be undulant fever. Many other complications, i.e. number of patients operated upon for appendicitis. Differential diagnosis on blood leukopenia, relative lymphocytosis higher than is usual in many other diseases. Undulant fever more prevalent in dairy producing countries than typhoid fever.

Vaccine: We tried it in 2 of our patients but it is unfair to say one thing or another. First patient child, 13 years old, temperature appeared to come down. Given 1/4 cc. of dose and gradually worked up to 1 cc. There was a marked rise in temperature which we attributed to vaccine. General pessimistic attitude toward it. We have had not enough experience to know effect.

O.McD.: The southern part of the state is probably our biggest producing area, and we also have a packing plant there. Map shows six years study. Different colors for each year. Black one 1932. The number of cases during the year 1927 - 6, 12 in 1928, 42 in 1929, 72 in 1931, 60 in 1932 with 15 thus far this year. It is well to know the differential diagnosis and also that the agglutination test is not present in all cases. We have recently had the case of young child referred to us. We have not counted it yet as it was atypical. Blood specimen taken on third day negative all the way through. Child died on 6th day. Diagnosis returned undulant fever because they happened to be taking milk from herd in which disease present. Fact that child died six days after onset is against undulant fever.

C.P.F.: I want to call attention to the dates in your manuscript. Organism of brucella 10 years later, not same year Bang organism discovered. We have little definite knowledge as to the extent of disease in animals. It has been stated according to figures in publications disease is 30, 40, and 85% among cattle. Absolutely untrue. Same thing shown in own laboratory. At present time we are attempting to make survey. We have completed this survey in one township in Washington County, a typical dairy country, and found only a trifle more than 6% infection. We are to attempt next tests in other counties, giving a typical cross section of cattle in the state. Then we will have knowledge of distribution of disease. Not as widespread as laboratory gives us to believe. Important organism is in cattle. Some cases apparently coming from hogs may have come more to our attention because of more virulent type in man. Bovine does not give as typical symptoms as does procine variety.

Diagnosis: This is not an easy thing to do unless there is a definite picture. Same thing in animals, i.e.

negative agglutinations in positive infections. On the other hand we have another picture in laboratory--15 workers of these 5 or 6 carry titers which would be diagnosed as undulant fever, i.e. titers above 1:80. If individuals developed symptoms clinician would call case undulant fever.

You might be interested in an article in British medical journal by Dr. Zomet. They have sent for Dr. Huddleson of Michigan, non-antigenic and non-pathogenic immunity agent. They have been working with this strain of organism in Island of Malta. If results can be taken at face value it is marvelous because controls all developed Malta Fever and only one of their vaccinated animals developed disease. Elimination or segregation of animals and breeding new herd is the answer. They can be raised free of the disease. That scheme is in operation in nearly every state in the union.

W.L.B.: I would like to point out one of the phenomenon of the disease in cattle - sterility or reduced fertility in animals. If animals become infected and expulsion of fetus results they become non-breeders and are sold for beef prices. Interested in doing away with this disease for that reason. Activity of disease in reproductive organs. In the male animal we do not see very much pathology but when we do it is usually in the testes producing orchitis. After temperature reaches normal removed affected gland. Agglutination titer dropped back, and remained as head of herd without former trouble. Lesion produced within the testis produced an acute orchitis and destroyed the gland. If we do not resort to removal of affected organ it means permanent sterility even though other gland is not involved at time.

Gertrude Gunn,
Record Librarian.

V. NEWS

Honors --

Chief Pediatrician Irvine McQuarrie leaves this weekend for Philadelphia where he will deliver the Annual Frederick Packer Memorial Address, April 11th, before pediatric and affiliated groups. Dr. McQuarrie joins a distinguished list who have been so honored. Congratulations! Enroute he will attend the Federated Biological Society meeting in Cincinnati.

Congratulations --

To Dubuque.

Lester G. Erickson, fellow in Radiology, left April 1st to become Radiologist for the Medical Society and Hospitals of Dubuque, Iowa. Coming back to us from practice, he completed his special training in good time. While here he impressed all with his excellent ability. We are certain he will be a credit to our institution. He is one young man who knows what he wants, gets it, and goes on. Congratulations!