

## ABDOMINAL ACTINOMYCOSIS

Patchrin PEKANAN, Thitiporn RANGSITPOL

### ABSTRACT

Two cases of abdominal actinomycosis was presented. In one case, the disease involved ileocecum and appendix of a 35-year-old man. Plain film and ultrasonography was suggestive of localized inflammation of the ileocecal area and not specific. In another case, the disease involved left ovary with fistula tract to the anterior abdominal wall. CT scan showed left ovarian mass and soft tissue lesion at anterior parietal peritoneal cavity and muscle of right anterior lower abdominal wall.

### INTRODUCTION

Actinomycosis is a chronic, progressive, suppurative disease characterized by formation of multiple abscesses, draining sinuses, abundant granulation and dense fibrous tissue. The appearance of sulfur granules in the lesions, sinus walls or discharges of involved tissues is characteristic (1).

Two cases of abdominal actinomycosis were shown; the lesion involved ileocecal area and the appendix in one case and ovary in another case.

### CASE REPORTS

#### CASE 1

A 35-year-old man, a labor-worker in Bangkok, admitted to Ramathibodi hospital due to right lower quadrant colicky pain for one day. Watery diarrhea was noted for five times. He also had high fever for 12 days prior to the admission. A mass was palpated at right lower quadrant with signs of peritonitis in this region. Leukocytosis was present with PMN predomination. Ruptured appendicitis was suspected and he was explored. Plain films of the abdomen (Fig.1) showed localized rigidity of the bowel loop of terminal ileum and proximal right colon with short air fluid levels. Intraperitoneal free fluid was noted. Ultrasonography at right lower quadrant (Fig.2) showed thickened bowel wall and surrounding fatty

infiltration with fluid. At operation, 500 cc of serosanguinous fluid was found. There was hyperemia of the terminal ileum, mass at cecum, necrosis of the mesocolon and retroperitoneal area. Fibrin and old hemorrhage was observed. Enlarged nodes were not detected. At pathological examination revealed ulceration of the mucosa of the terminal ileum. Ileocecal valve was markedly swelling. The lumen of the appendix was severely narrow. Intact folds of the cecum and mild swelling of the mucosa was detected. Fibrin coating at serosal surface of the resected bowel was seen. A mass, size 3-4 cm in diameter was seen at mesocolon of the ascending colon; cut surface showed soft yellowish, gray and tan friable tissue with hemorrhagic and necrotic foci. Actinomycosis was suggested, though organisms were not identified by GMS. The patient recovered well.

#### CASE 2

A 26-year-old woman from Yasothorn province (North-eastern part of Thailand), observed a foul smell, bloody greenish discharge from the palpated mass at right lower abdomen for one day. She had chronic lower abdominal pain for 3 months. A 10-cm mass at left adnexa was found by the Gynecologist one month ago. Right lower abdominal pain and palpable mass was observed for 5 days. There was a low grade fever. Twenty-weeks-sized suprapubic mass was noted

---

PMN = Polymorphonuclear

Department of Radiology, Ramathibodi Hospital, Rama 6 Street, Bangkok 10400, Thailand.

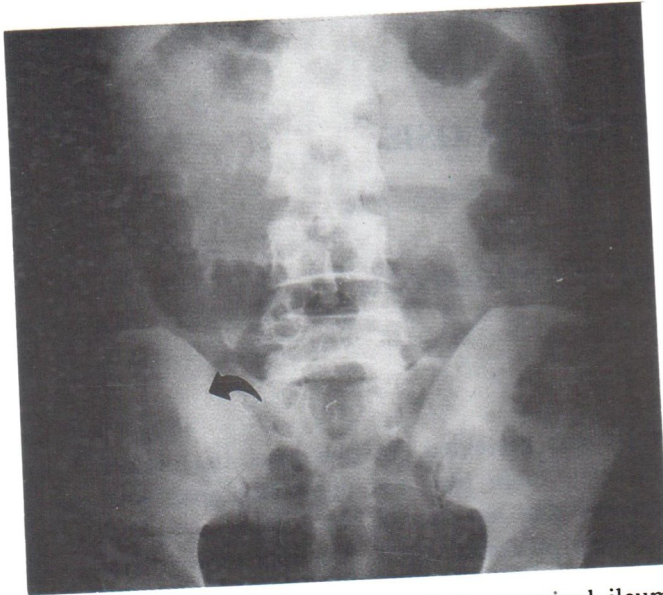


Fig.1 Case 1. Localized rigidity of the terminal ileum at RLQ by plain film

with nodular surface and local cellulitis. A complexed mass was noted by ultrasonography at right lower quadrant, (Fig.3). CT scan showed a solid mass at left adnexa with small cystic areas. Left ovary could not be identified well (Fig.4). An ovoid shape soft tissue lesion was shown at left paramedian anterior intraperitoneal cavity, attached to the parietal peritoneum and was at just anterior to the urinary bladder. An infiltrative border soft tissue lesion was detected at muscle plane of right lower anterior abdominal wall, at mid and lower iliac wing level (Fig.4).

At surgery, left ovary was slightly enlarged, filled with pus. A fistular tract was noted at right side of the abdominal wall connecting to the intraabdominal cavity. Adhesion was present with the omentum and jejunum. At gross examination, the left ovary contained solid, grey-whitish mass in the entire ovary. Section of the left ovary was compatible with actinomycosis.

### DISCUSSION

Abdominal actinomycosis was first described by Bradshaw (2) in 1846. Bolinger (3) demonstrated that a fungus was the primary infective agent in 1877. Actinomyces was derived from the Greek works for ray and fungus, due to the appearance of the filaments radiating from a central tangled mass. Israel (4) noted granules from material, containing mycelia in 1878. Studies of cell wall components resulted in classification of Actinomyces as a bacterium, distinct from the fungi (5,7).

The Actinomyces organism is a fastidious, gram positive, non-spore forming microaerophilic or obligate anaerobe that varies in growth pattern and is somewhat difficult to isolate. Successful isolation requires culture of multiple samples of purulent material in enriched media under anerobic conditions with carbon dioxide, and an adequate time for growth (1,8,9).

There are at least six different species of bacteria which can cause actinomycosis in humans (9,12). Actinomyces israelii, Actinomyces propionicus, Arachnia propionica (13,15). Actinomyces naeslundii

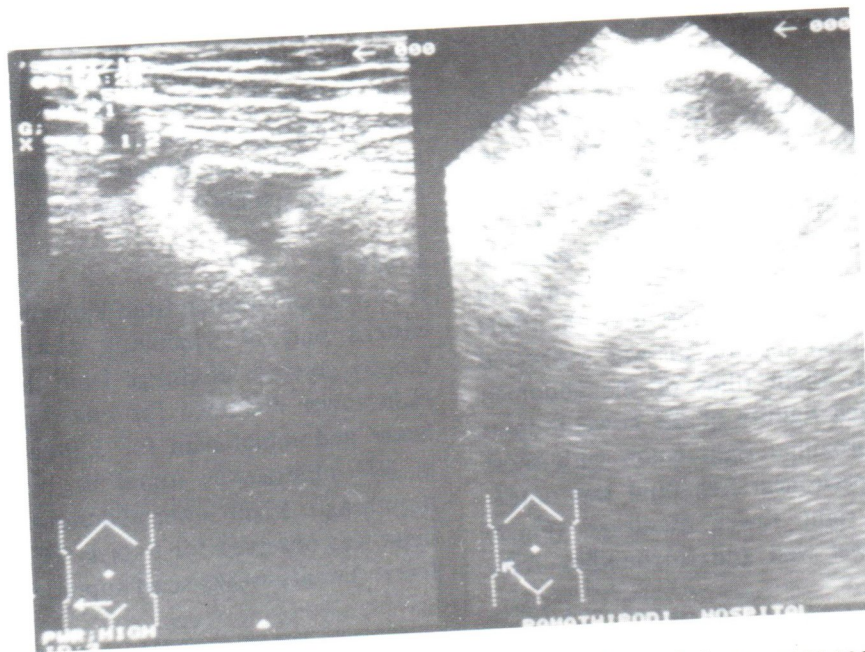


Fig.2 Case 1. Fluid collection at RLQ and around bowel loop, was seen at ultrasonographic examination.

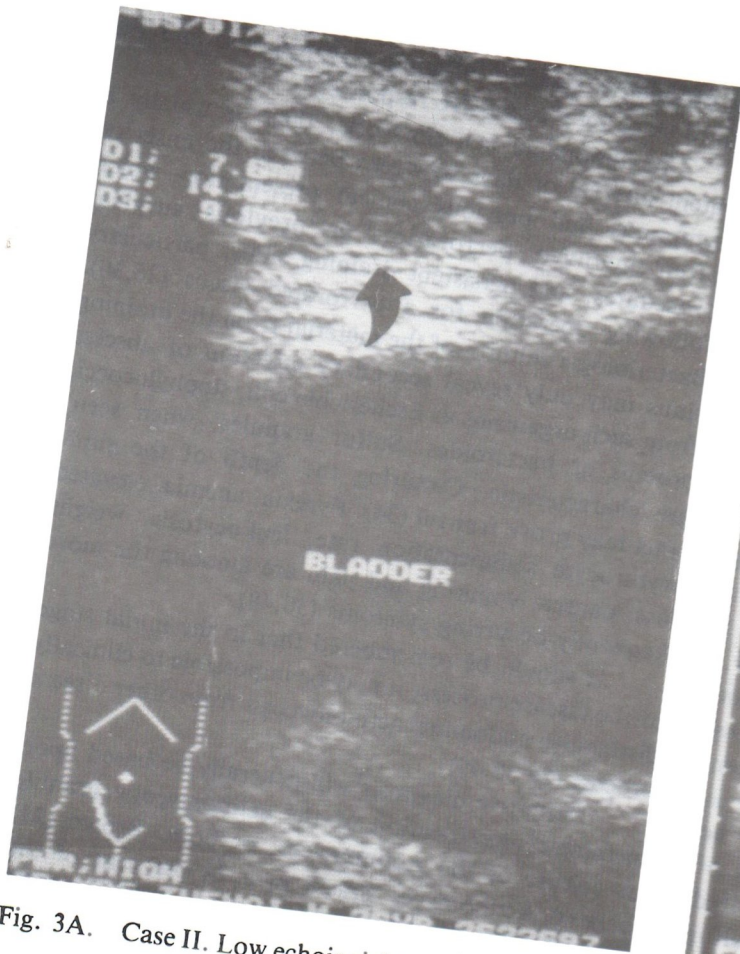


Fig. 3A. Case II. Low echoic right anterior abdominal wall lesion, at ultrasonogram.

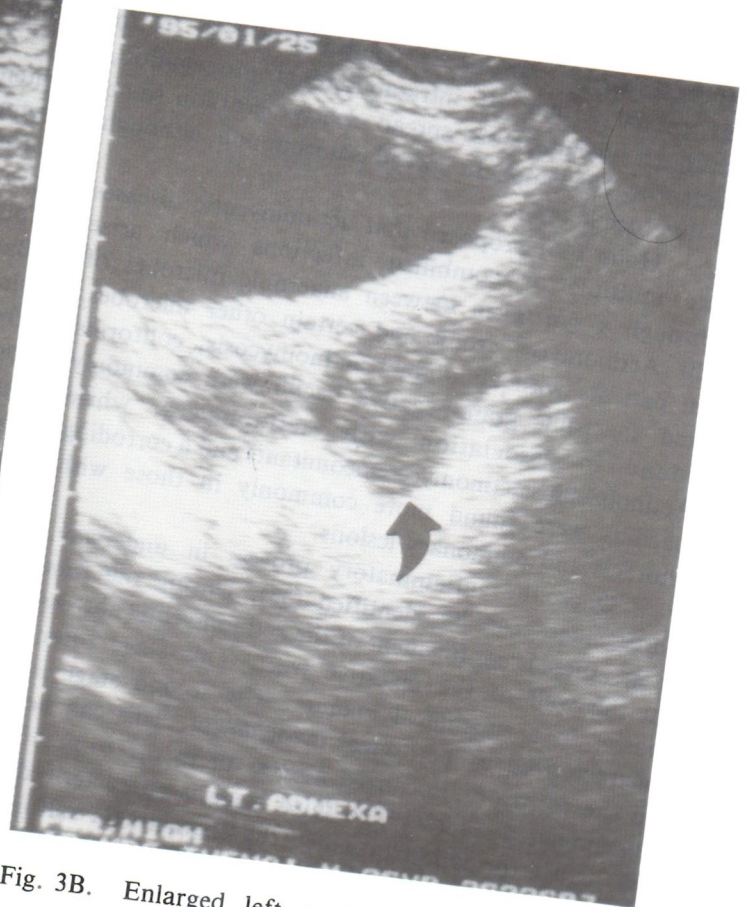


Fig. 3B. Enlarged left ovary with solid lesion at posterior part, at sonography

(16,17), *Actinomyces viscosus* (18), *Actinomyces eriksonii*, *Bifidobacterium* (11), and *Actinomyces odontolyticus* (19,20).

Although the low percentage of recovery of *Actinomyces* organisms by culture, and the high percentage of no growth in culture is generally conceded (12), it is also contended that in order to obtain more fruitful yield, close cooperation between the attending physician and the microbiologist is required (9,15).

Actinomycosis has a world-wide distribution and is present with equal frequency in city and rural dwellers. There is no discernible sex predilection, although males are more frequently infected than females (8). Most instances occur in adolescents and middle-aged individuals.

The frequency with which abdominal actinomycosis occurs as compared with the cervicofacial and thoracic forms of the disease is varied. In some studies, it ranks first (22,23), in others third (22,24-26) and in most second (24,26,27).

The incidence of actinomycosis is difficult to estimate. It would appear that the principal reasons for difficulty lie in the fact that clinical suspicion of the disease is generally low, difficulties are encountered in obtaining bacteriologic cultures, the response of

the microorganisms to penicillin is exquisite and *Actinomyces israelii* is indigenous to the oral cavity and as such, simple recovery does not necessarily establish the diagnosis.

It is accepted that the *Actinomyces* organism is a normal, frequently found inhabitant of the oral cavity and tonsillar crypts (11). Infestation of the gastrointestinal tract appears to come primarily from *Actinomyces* in the oral cavity (28,29).

Abdominal actinomycosis shows a predilection for the right iliac fossa and has frequently followed operations for acute appendicitis or drainage of abscesses in this region. The appendix is by far the most common intra-abdominal organ involved (22,24). This is followed with much lesser involvement of the colon (30), stomach (31), liver (32), gallbladder (33), pancreas (34), small bowel (35), anorectal region (38), pelvis (37), abdominal wall (38), and other less common sites (39,40). Most instances show involvement of a single organ. Disseminated intra-abdominal actinomycosis is less common (21,41). With the introduction of intrauterine contraceptive devices, a number of instances of pelvic actinomycosis have been reported (42,43,44).

Once the organisms have penetrated through the mucosal barrier, spread by continuity appear as to the primary route of intra-abdominal propagation, although hematogenous spread clearly occurs in some instances. The lymphatic spread is uncommon, although it does occur (46).

Holm (46) proposed that actinomycotic diseases are multiple or combined infections which arise through a synergism between anaerobic microbes of the Actinomyces group and certain other microbes. In patients with abdominal actinomycosis, coliforms and anaerobic gram-negative bacilli were commonly found in association with Actinomyces, while Actinobacillus actinomycetemcomitans and a corroding bacillus were found more commonly in those with thoracic and pulmonary lesions.

Once the inflammatory process is underway, three stages can be identified (35). During the first stage, there is confinement of the actinomycotic abscess to the wall of the digestive tract. This stage often passes in a subclinical manner. The second stage is characterized by peritoneal involvement. This can be in the form of a localized abscess or in the form of disseminated disease. The third stage is characterized by fistula formation in which the characteristic sulfur granules are often noted.

The actinomycotic abscess without formation of fistula is generally typified by single or multiple abscess or by indurated masses with hard fibrous walls and soft central loculations containing white or yellow pus.

Minsker and Yegorova (47) demonstrated that the inflammatory response to Actinomyces began during the first hours after inoculation and comprised three stages. The first stage was characterized by a nonspecific exudative inflammatory response, the second stage by

a productive specific inflammatory response and the third stage by a regressive response.

In abdominal actinomycosis, there is usually a latent interval of days to weeks between the onset of symptoms and previous clinical presentation which often involved perforation or previous surgical procedures, and persistent draining sinus, particularly following operation for a perforated viscus (26,30). Bacteriologic cultures of the material from the draining sinus may only reveal secondary infection of abscess from such organisms as Escherichia coli, staphylococci, proteus or bacteroides. Sulfur granules, when seen, are characteristic. Culturing the depth of the sinus tract may prove fruitful (34). Pyrexia, anemia, elevated erythrocyte sedimentation rate, leukocytosis, weight loss, nausea, vomiting and pain are among the more frequently occurring symptoms (30,48).

It should be remembered that in the initial stage of the disease process, it may be impossible to clinically distinguish abdominal actinomycosis from other disease process (26,35,49).

Definitive diagnosis will generally be based upon histologic identification of the actinomycotic granule or culture of the Actinomyces, or both. Brown (21) isolated Actinomyces israelii in a 24 per cent of the instances cultured. There was no growth in about 46 per cent of the cultures and the remaining cultures grew other bacteria or unidentified organisms.

In the abdominal form of actinomycosis, the majority of reported instances have become evident several weeks or even months after an acute episode of perforated gastrointestinal disease, such as acute appendicitis, perforated colonic diverticulitis, perforated peptic ulcer or acute ulcerative disease of the intestinal tract, or after trauma, including operations. However,

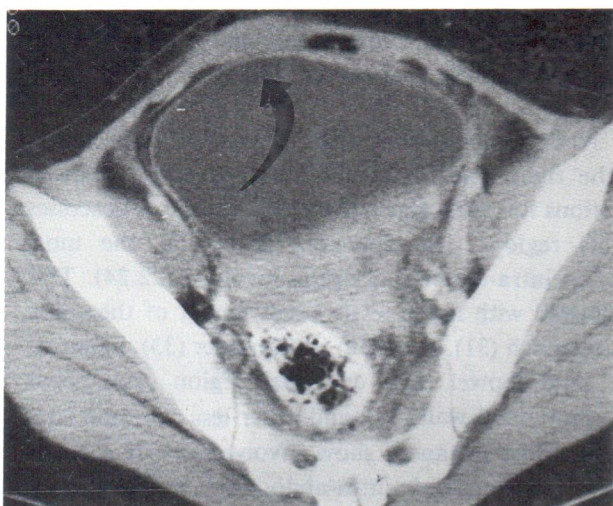


Fig.4A. Case II Right anterior abdominal wall lesion, at CT scan

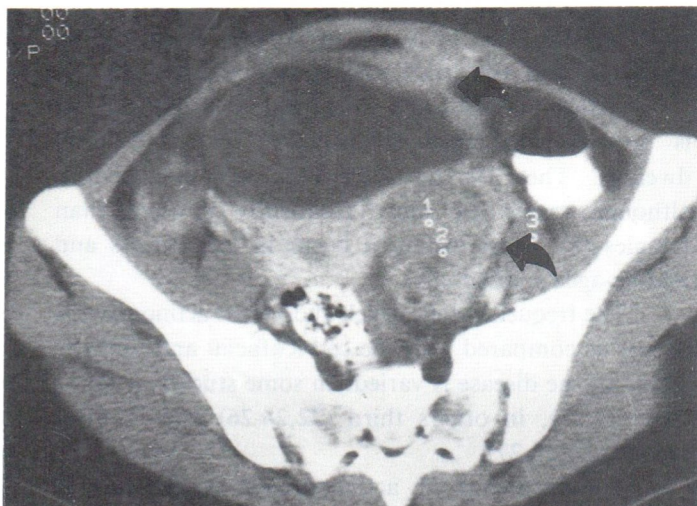


Fig.4B. Left ovarian lesion, at CT scan

in many patients no antecedent disease or operation has been noted (21). Actinomyces organisms are best thought of as opportunist pathogens, although the conditions predisposing to infection are not always apparent. A more frequent association of antecedent disease or trauma, or both, generally noted in the cervicofacial or thoracic forms of actinomycosis. A number of patients with thoracic actinomycosis have pre-existing chronic bronchitis, emphysema, chronic pneumonitis, aspiration or infarction (21).

Our first case mimicked acute appendicitis and the disease was at stage two and the second case was at stage three. Imaging diagnosis of these two cases alone could not lead to the proper diagnosis due to the nonspecificity of the findings. Fistula tract could occur in other chronic infections such as tuberculosis, Crohn's disease, fungal infection, and in malignant process.

## REFERENCES

- Berardu RS. Abdominal actinomycosis. *Surgery, Gynecology & Obstetrics* 1979;149:257-266.
- Idem. Visceral actinomycosis. *Br. Med J* 1949; 2:1311.
- Bollinger O. Ueber sine neue Pilzkrankheit beim Rinde. *Centralb f.d. med. Wissensch, Berl* 1877; 15:481.
- Israel J. Neue Beobachtungen auf dem Gebiete der Mykosen des Menschen. *Virchow Arch Pathol Anat.* Berlin 1878;74:15.
- Cummins CS. The chemical composition of cell walls of actinomycetes and its taxonomic application. *Ann Inst. Pasteur*, 1962;103:385
- Idem. Chemical composition and antigenic structure of cell mass of *Corynebacterium*, *Mycobacterium*, *Nocardia*, *Actinomyces* and *Arthrobacter*. *J Gen Microbiop* 1963;28:35.
- Cummins CS, Harris H. Studies on the cell wall composition and taxonomy of Actinomycetales and related groups. *J Gen Microbiop* 1958;18:173.
- Conant MF, Smith DT, Bajer RD and Callaway JL. Actinomycosis. In: *Manual of clinical Mycology*, 3rd ed., chapt i, ppl-37. Philadelphia: W.B. Saunders Co., 1971.
- Dowell VR, Sonnenwirth AC. Gram-positive nonsporeforming anaerobic bacilli. In: *Manual of Clinical Microbiology* 2nd ed, chap 42, pp 396-401. Washington, D.C.: Am Soc Microbiologists, 1974.
- Bowden GH, Hardie JM. Commensal and pathogenic Actinomyces species in man. *Soc Appl Bacteriuk Symposium Series*, London, 1973:2:227.
- Lerner PI. Susceptibility of pathogenic Actinomycetes to antimicrobial compounds. *Antimicrob Agents Chemother* 1974;5:302.
- Wallace RJ, Jr. Musher DM. Actinomycosis; an update. *Int J Dermatol* 1977;16:185.
- Abe PM, Majeski JA, Stauffer LR. Histological changes observed in *Arachnia propionica* infection of mice. *J Surg Res* 1978;25:174.
- Brock DN, Georg LK, Brown Jm, Hicklin ND. Actinomycosis caused by *Arachnia propionica*; report of 11 cases. *Am J Clin Pathol*, 1973;59:96.
- Georg LK, Roberstad GW, Brinkman SA. A new pathogenic Actinomyces species. *J Infect Dis* 1965; 115:88.
- Salkin IF, Gordon MA. Pathogenicity of Actinomyces naeslundii. *Chest* 1974;66:467.
- Varkey B, Kurup PV. Pathogenicity of Actinomyces naeslundii. *Chest* 1974;66:467.
- Gutschik E. Endocarditis caused by Actinomyces Viscosus. *Scand J Infect Dis* 1976;8:271.
- Guillon JP, Durieux R, Dublanchet A, Chevrier L. Actinomyces odontolyticus; premiere etude realisee en France, *CR Acad Sci, Paris* 1977; 285:1161.
- Morris JF, Kilbourn P. Systemic actinomycosis caused by Actinomyces odontolyticus. *Ann Intern Med* 1974;81:700.
- Brofwn JR. Human actinomycosis; a study of 181 subjects. *Hum Pathol* 1973;4:319.
- Harvey JC, Cantrell JR, Fisher AM. Actinomycosis; its recognition and treatment. *Ann Intern Med* 1957;46:868.
- Weed LA, Baggenstoss AH. Actinomycosis; a pathologic and bactriologic study of twenty-one fatal case. *Am J Clin Pathol* 1949;19:201.
- Davis MIJ. Analysis of forty-six cases of actinomycosis with special reference to its etiology. *Am J Surg* 1941;52:447.
- Pulverer G. Problem of human actinomycosis. *Postepy Hig Med Dosw* 1974;28:253.
- Copy VZ. Actinomycosis. London: Oxford University Press, 1938.
- Eastridge CE, Orather JR, Hughes FA, Jr. Actinomycosis; a 24-year-experience. *South Med J* 1972;65-839.
- Kolouch F, Peltier LF. Actinomycosis. *Surgery* 1946;20:401.
- Spilsbury BW, Johnstone FRC. The clinical course of actinomycotic infections; a report of 14 cases. *Can J Surg* 1962;5:33.
- Davies M, Deddie NC. Abdominal actinomycosis. *Br J Surg* 1972;60:18.

31. Cajal SRY, DeLaPena M, Campo C. Actinomycosis gastrica Intramural; observaciones anatomoclinicas sobre un caso y revision de la literatura. *Rev Enfern Apar Dig* 1977;50:445.
32. Golematis B, Hatzitheofilou C, Melissas J. Liver actinomycosis. *Am J Gastroenterol* 1976;65:148.
33. Marrie T, Stiver HG, Molgat A. Actinomycosis of the gallbladder. *Can J Surg* 1977;20:147.
34. Jelen M, Drak AJ, Korolewicz W, Wadowski R. Diagnostic difficulties in an atypical case of actinomycosis of the abdominal cavity. *Patol Pol* 1977;28:227.
35. Mousseau PA, Mousseau-Brodu MC. L' actinomyose abdominale. *J Chir, Paris* 1973;106:565.
36. Brewer NS, Soencer RJ, Nichols DR. Primary anorectal actinomycosis. *JAMA* 1974;228:1379.
37. Chakrabarty D, Wessely Z. Ovarian actinomycosis. *N.Y. State J Med* 1978;5:806.
38. Minocha VR, Sharma MM, Nair SK. Primary actinomycosis of the abdominal wall. *Aust N Z J Surg* 1975;45:66.
39. Anscombe AR, Hofmeyer J. Peranal actinomycosis complicating pilonidal sinus. *Br J Surg* 1954;41:666.
40. Crosse JEW, Soderdahl DW, Schamber DJ. Renal actinomycosis. *Urology* 1976;8:309.
41. Legum LL, Greer KE, Glessner SF. Disseminated actinomycosis. *Southern Med J* 1978;71:463.
42. Barnham M, Burton AC, Copland P. Pelvic actinomycosis associated with IUD. *Br Med J* 1978; 1:719.
43. Csapo Z, Csomor S, Zambo Z. Ovarian actinomycosis developed during the use of a plastic intrauterine contraceptive device. *Acta chir Acad Sci Hung* 1977;18:149.
44. Luff RD, Gupta PK, Spence MR, Frost JK. Pelvic actinomycosis and the intrauterine contraceptive device; a cytohistomorphologic study. *Am J Clin Pathol* 1978;69:581.
45. Spilbury BW, Johnstone FRC. The Clinical course of actinomycotic infections; a report of 14 cases. *Can J Surg* 1962;5:33.
46. Holm P. Studies on the aetiology of human actinomycosis-I, the other "microbes" of actinomycosis and their importance. *Acta Pathol Microbiol Scand*, 1950;27:736.
47. Minsker OB, Yegorova TP. Studies on histogenesis of actinomycosis. *Mykosen* 1974;17:303.
48. Putman HC, Dockerty MB, Waugh JM. Abdominal actinomycosis; an analysis of 122 cases. *Surgery* 1950;28:781.
49. Brongden CJ. Actinomycosis of the gastrointestinal tract; a study of fourteen cases. *J Lab Clin* 1922; 8:180.