

6-1956

## Acute Miliary Disease Of The Lung: Diagnosis And Treatment

M. A. Blankenhorn

Follow this and additional works at: <https://scholarlycommons.henryford.com/hfhmedjournal>



Part of the [Life Sciences Commons](#), [Medical Specialties Commons](#), and the [Public Health Commons](#)

---

### Recommended Citation

Blankenhorn, M. A. (1956) "Acute Miliary Disease Of The Lung: Diagnosis And Treatment," *Henry Ford Hospital Medical Bulletin* : Vol. 4 : No. 2 , 94-97.

Available at: <https://scholarlycommons.henryford.com/hfhmedjournal/vol4/iss2/8>

This Article is brought to you for free and open access by Henry Ford Health System Scholarly Commons. It has been accepted for inclusion in Henry Ford Hospital Medical Journal by an authorized editor of Henry Ford Health System Scholarly Commons. For more information, please contact [acabrer4@hfhs.org](mailto:acabrer4@hfhs.org).

## ACUTE MILIARY DISEASE OF THE LUNG DIAGNOSIS AND TREATMENT

M. A. BLANKENHORN, M.D.\*

More than 80 conditions have been recorded where disseminated miliary lesions are seen in x-ray examination of the lungs. When such lesions are the sole objective evidence of morbidity differential diagnosis is difficult and any contributing information is welcome.

Dr. Benjamin Felson at the Cincinnati General Hospital has emphasized the help that comes from the observed rate of change, i.e. progression or regression and has arbitrarily set two weeks or less as the period of change in acute diseases—two to eight weeks as subacute—and failure to change after eight weeks as a chronic disease. This is a technique available to less experienced observers and is appreciated by those not specialists in radiology.

Infection due to bacteria, viruses, rickettsia, fungi, protozoa, and inhalation of specific irritants may all cause similar lesions. Such lesions occur also in disorders of unknown etiology such as primary atypical pneumonia or infectious mononucleosis. No inclusive report is attempted here, but typical examples are shown with reference to the rate of change using Felson's standards. It is impossible to show reproductions of the numerous x-ray films demonstrated in the original presentation. In abstract it can be accepted that these films are all much alike—in fact, they were chosen because they could not be distinguished from miliary tuberculosis. Therefore, the rate of change and certain laboratory tests are useful.

Miliary tuberculosis is the most common and perhaps the most important because of the need of early and energetic treatment.

Tuberculosis may make rapid progression and regression under treatment. It is seldom chronic as miliary only.

Felson's experience in 1952 was that one-half of the cases showed other intrathoracic abnormalities due to tuberculosis.

Because pure miliary tuberculosis is perhaps the most important topic in my discourse, I will enlarge upon it later but first go on to some of the other miliary diseases.

Histoplasmosis is being recognized with increasing frequency and resembles tuberculosis in many respects—there is usually slow regression—much slower than tuberculosis. The pure miliary form is rare. The rate of progression depends in large part upon the degree of exposure, i.e. the number of organisms inhaled.

Newer techniques for histopathology show histoplasmosis and tuberculosis to be simultaneous and co-existent in lung lesions.

There is little morbidity from histoplasmosis of the lungs only in adults except in epidemics where severe exposure has occurred in dusty employment.

---

\*Professor of Medicine, University of Cincinnati College of Medicine, Cincinnati, Ohio.  
The material in this paper is an abstract of a presentation before the Henry Ford Hospital Medical Society, February 14, 1956, Detroit, Michigan.

*Diagnostic measures, histoplasmosis:*

1. Histoplasmin skin tests.
  2. Complement fixation tests for histoplasmosis. Said to indicate active infection if positive.
  3. Bone marrow culture.
  4. Culture of abnormal fluids, if present.
  5. Sputum and/or gastric cultures if pulmonary involvement is present.
  6. Biopsy of accessible lymph nodes or of scalene fat pad. Specific diagnosis may be provided by demonstration of organisms in sections.
- The prognosis is good except in those forms in which the blood stream is invaded.

Disseminated miliary lesions are an uncommon form of primary atypical pneumonia and difficult to prove, but they do occur.

Chicken pox and measles may show miliary lung lesions. There is rapid regression in measles but slow regression in chicken pox.

Bronchiolites fibrosa obliterans develops acute miliary disease usually due to the accidents of inhaling irritants. Examples are shown where no known irritant was discovered, but lesions found at autopsy were such. In non-fatal cases there is rapid regression.

Asthmatic miliary pneumonia has been seen 15 times at the Cincinnati General Hospital and published by Felson and Felson<sup>1</sup> as acute diffuse pneumonia in chronic lung disease. Here acute miliary nodulation occurs in a patient who usually has had asthma for a long time. During the attack of pneumonia eosinophilia is absent. The miliary lesions regress rapidly.

A few examples of pneumoconiosis are shown where the lesions are of a disseminated miliary type but different from tuberculosis in many respects—principally in being static.

Blastomycosis is a typical fungus disease with miliary lesions.

I wish now to enlarge upon the diagnosis and treatment of miliary tuberculosis. Miliary tuberculosis, in the experience of the Department of Medicine at the Cincinnati General Hospital, is essentially a problem in internal medicine. This accounts for the emphasis I put upon it and explains why I insinuate tuberculosis into a program for internists. Few doctors, except tuberculosis experts, have been interested until the recent era of chemotherapy. Early diagnosis and energetic treatment depends upon the doctor who first sees the patient. We have had a generous experience at the Cincinnati General Hospital. What was formerly a rapidly fatal disease is now treated with remarkable success. Dr. J. Park Biehl, Assistant Professor of Medicine, has given me reports and treatment schedules for miliary tuberculosis which have not yet been published. From now on I am quoting Dr. Biehl. I concur in his emphasis upon the importance of lumbar punctures.

Routine of the Cincinnati General Hospital Department of Internal Medicine.

*Diagnostic measures, miliary tuberculosis:*

1. Tuberculin testing.
2. Lumbar puncture (to detect presence of meningitis). Culture of CSF x 2 for AFB if abnormality present.



3. Sputum examinations and/or 3 gastrics for culture and guinea pig.
4. Three 24 hour urine collections for AFB culture.
5. Biopsy of any tissue likely to show caseation (L-nodes, endometrium). Biopsy for miliary granulomata (e.g., liver biopsy) less satisfactory since granulomata thus found are not necessarily tuberculous. One frequently may surmise a granulomatous infection from the x-rays alone. Culture of bone marrow has been disappointing. No CSF culture has been positive in the absence of meningitis.

*Treatment schedule, miliary tuberculosis:*

1. Bed rest in hospital 3 - 6 months, according to home situation, then up and about at home for 3 more. Ability to return to work governed by the situation, mainly. Some have returned to light work 4 - 6 months after starting therapy.
2. Streptomycin ½ gm. plus dihydrostreptomycin ½ gm. q.d. for 2 months, then streptomycin 1 gm. twice weekly for a total of 1 year.
3. Isoniazid 20 mg./kg. daily in divided doses, with 300 mg. of pyridoxine daily, both for 2 months; then isoniazid 300 mg. daily alone for a total of 1 year. Pyridoxine is given to prevent peripheral neuritis seen when large doses of isoniazid are given. (Biehl and Vilter)<sup>2</sup>.
4. Tuberculous isolation for 1 month, or until sputa are negative 2 months.

*These factors stand out in the study of 56 cases of miliary tuberculosis in adults observed from 1951 to the present.*

1. Age incidence: over 40% of the group were 60 or over.
  2. Most of the younger women were or had recently been pregnant.
  3. High preponderance of negroes (49/56). This is in keeping with our experience in extrapulmonary tuberculosis generally.
  4. Nonspecific nature of symptoms — those of infection in general; cough, weight loss, weakness, feverishness, anorexia, night sweats, dyspnea, chills and vomiting (chills in 13/51 who give any history). Headache and delirium pointed to meningitis. Duration of present illness was prolonged in some; 12/56 were sick for 10 weeks or more.
  5. Paucity of physical examination: rales, fever. No splenomegaly.
  6. Only 4 had had known tuberculosis previously. Except for 18 with meningitis, 25 (45%) had another form of tuberculosis present concurrently. This was recognized clinically in only 18 (pulmonary, pleural, lymph node, genito-urinary, bone, peritoneal involvement). Thus in 68% there was no clinical evidence of other tuberculosis.
  7. Except when terminal with meningitis, leukocytosis was rare.
  8. Diagnosis established by:
    - (a) demonstration of tuberculosis.
    - (b) demonstration of a grossly caseating granuloma.
- Excluding 6, in whom the diagnosis was never suspected (usually terminal on admission), the diagnosis was made according to the above criteria in 44 of 50 patients. This was best achieved by sputum or gastric cultures, urine cultures, culture of serosal exudates, and biopsy of anything likely to reveal caseation, like nodes or endometrium. CSF was positive in 13 of 16 with meningitis. Tuberculin were negative in 7 of 20. Five of these were terminal cases and died.
9. Treatment: Start tuberculosis treatment in patients with subacute febrile illness, especially in negroes in whom x-ray shows miliary densities and for which no other

good explanation exists. It is not reasonable or necessary to await definite proof before starting therapy. Specimens should be obtained for this, of course, before or during first few days of treatment.

**Response to therapy:**

Fever: normal in from 2 days to 10 weeks — average 5 weeks.

X-ray change: Significant improvement has been noted within 2 weeks in 5, and lesions have cleared completely in 2 after 17 and 19 days treatment. Usually improvement seen after 4 weeks; most cleared by 16 weeks.

**Bad prognosis:**

1. Meningitis present — 6/18 lived.
2. Pulmonary tuberculosis — 0/8 lived.
3. Tuberculin anergy — 2/7 lived.

**Relapse rate:**

2 cases: one with subsequent miliary again, another lapsed treatment after 8 months and died of meningitis.

**BIBLIOGRAPHY**

1. Felson, B., and Felson, H.: Acute diffuse pneumonia of asthmatics, *Am. J. Roentgenol.* 74:235, 1955.
2. Biehl, J. P., and Vilter, R. W.: Effects of isoniazid on pyridoxine metabolism, *J. A. M. A.* 156:1549, 1954.
3. Felson, B.: Acute miliary diseases of the lung, *Radiology* 59:32, 1952.

**AIDS IN DIFFERENTIAL DIAGNOSIS OF THE COMMONER TYPES OF  
ACUTE MILIARY DISEASES**

(Adapted, 1955 from Felson, B.)<sup>3</sup>

<i>Disease</i>	<i>Laboratory Aids</i>	<i>Roentgenologic Aids</i>
1. Miliary tuberculosis	Lumbar puncture; biopsy; cultures	Progression
2. Histoplasmosis	Cultures; serological tests; skin test	Very slow regression; lymphadenopathy
3. Blastomycosis	Cultures; serological tests; skin test	Usually progression
4. Coccidioidomycosis	Cultures; serological tests; skin test	Slow regression; lymphadenopathy
5. Acute diffuse pneumonia in chronic lung disease	No eosinophilia	Rapid regression; hilar enlargement
6. Bronchiolites fibrosa obliterans		Usually rapid regression
7. Loeffler's syndrome	Eosinophilia	Rapid regression
8. Primary atypical pneumonia	Cold agglutination	Rapid regression
9. Infectious mononucleosis	Blood smear; heterophile agglutination	Rapid regression; lymphadenopathy