Miliary tuberculosis in British Columbia

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Summary: The case records were studied of all 48 patients with miliary tuberculosis registered by the British Columbia Division of Tuberculosis Control between 1967 and 1971. Two groups of patients were found, one group of 30 in whom the diagnosis was made during life and antituberculous therapy instituted, and a second group of 18 patients in whom the diagnosis was made only at autopsy and who had received no antituberculous therapy. In the latter group there was a particularly high incidence of concomitant disease; additionally, tuberculin testing, bacteriological search for acid-fast bacilli and chest radiography were frequently neglected. This group consisted mainly of elderly male Caucasians. The disease was characteristically insidious in onset, frequently with non-specific symptoms and physical findings. Fever was a common presenting symptom. Several patients presented with a fulminant illness which, although diagnosed, was not affected by antituberculous therapy and was rapidly fatal. North American Indians showed a high incidence of disease but a low mortality rate, probably owing to their younger age.

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In recent years many authors have stressed the changing pattern of miliary tuberculosis. Formerly a disease primarily of children, it is now most common in the eighth decade.[1] Diagnosis is frequently made only at autopsy.[2-4] Miliary tuberculosis is found in approximately 10% of patients with a fever of unknown origin.[5]

Varied associated hematological reactions including leukopenia, pancytopenia, leukemoid reactions, agranulocytosis[6] and monocytosis[1] have been described. A high incidence of concomitant disease[1,4,7] further complicates diagnosis. Chest radiography is frequently negative[8] and tuberculin anergy is common.[9]

The present report describes the pattern of miliary tuberculosis in British Columbia over the five-year period, 1967-1971.

Material

The case records of all 48 patients registered with the B.C. Division of Tuberculosis Control, Vancouver as having miliary tuberculosis were studied retrospectively. All cases of miliary tuberculosis occurring in British Columbia are registered, including cases in which the diagnosis is made at autopsy. Since the crude autopsy rate in British Columbia for the year 1970 was only 33.2/100 deaths it is quite possible that the actual number of patients dying of miliary tuberculosis undiagnosed during life may be considerably higher than the registered figures would indicate. There was no appreciable difference in the number of cases reported each year. The patients can be divided into two categories. The first group comprises 30 patients in whom the diagnosis was made during life and who were treated for miliary tuberculosis. The second group consists of the remaining 18 patients in whom the diagnosis was reached only at the postmortem examination and who had received no antituberculous therapy. Cases of tuberculous meningitis with no other evidence of miliary spread were excluded from the present series.

Results

Age and sex distribution in the 48 cases are shown in Table I. In both groups of patients there were twice as many males as females. In the diagnosed and treated group there was fairly uniform age distribution, compared with the autopsy-diagnosed group in which 17 patients (94%) were over the age of 50.

Racial origin

This is shown in Table II. Of the 26 Caucasian patients, 21 were born in Canada, two in Ireland, two in Portugal and one in Finland. In Caucasians and Asiatics over half of the cases occurred in individuals over 60 while half of the North American Indians were below the age of 30. Over 80% of the autopsy-diagnosed group were Caucasians, whereas 43% of the diagnosed and treated group

Table I
Age and sex distribution in the groups of patients

	Diagnosis made during life and treatment given										
Age	0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90+	Totals
Male	3	1	2	3	1	1	4	0	5	0	20
Female	0	1	5	1	1	2	0	0	0	0	10
	Diagnosis made at autopsy										
Age	0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90+	Totals
Male	1	0	0	0	0	4	3	1	2	1	12
Female	0	0	0	0	0	0	3	1	1	1	6

were North American Indians. In 1970 there were just over 50,000 Indians in British Columbia while the total non-Indian population was 2,135,000.[10]

Symptomatology

This was recorded in 42 of the 48 patients. Non-specific symptoms including general malaise and weakness were prominent, being present in 28 (67%) of the patients. Cough was present in 26 (62%) and sputum production in 17 (40%) of the patients. Weight loss was also frequent, occurring in 24 (57%). Less common symptoms included night sweats (four patients), headache (four patients) and urinary symptoms (three patients). Six of the twelve autopsy-diagnosed cases in whom symptoms were recorded had no respiratory symptoms (50%), as compared with 9 of the 29 patients in the diagnosed and treated group (31%).

Physical findings

Detailed records were available for 37 patients. Fever was the single most common finding, being present in 28 (76%) of the patients. Crepitations of varied type and location were observed in 20 (54%). Hepatomegaly was found in nine patients, three of whom were alcoholics; splenomegaly was present in two and generalized lymphadenopathy in one patient. Seven of the patients had impaired consciousness, ranging from confusion to coma, but meningism was observed in only one patient. Six patients (16%) presented with fever of unknown origin and no other significant symptomatology or physical findings; four were in the autopsy-diagnosed group. There were no other major differences between the two groups with respect to the physical findings.

Concomitant disease

This was present in 23 of the patients, of whom 15 were in the autopsy-diagnosed group and 8 in the diagnosed and treated group.

In the former, co-existing diseases included atherosclerotic heart disease (five patients, none of whom had acute coronary thrombosis as cause of death), dementia (two patients), pneumonia and chronic pyelonephritis (each two patients). Conditions affecting only a single patient included chronic myelogenous leukemia, Wegener's granulomatosis, carcinoma of the breast and pulmonary embolism. Two patients died from massive gastrointestinal bleeding, one each from presumed "stress" ulceration of the duodenum and stomach.

In the diagnosed-and-treated group the most common co-existing disease was alcoholism with fatty infiltration of the liver which was present in three patients, one of whom died. Other concomitant illnesses included systemic lupus erythematosus, silicosis, traumatic quadriplegia and atherosclerotic heart disease. One patient had an aortic valve homograft.

Although the incidence of concomitant disease was high (83% in the autopsy-diagnosed group), it was considered to have contributed, directly or indirectly, to death in only seven patients, all of whom were in the autopsy-diagnosed group.

Bacteriology

The results of smear and culture examinations for *Mycobacterium tuberculosis* are shown in Table III.

Of the group of patients in whom the diagnosis was made during life, positive smears were obtained in two thirds and positive cultures in over 85% of the 30 cases. Smears and cultures were negative in three patients in this group.

In 16 of the 18 autopsy-diagnosed cases no smears or cultures for *Mycobacterium tuberculosis* were examined antemortem. In one pa-

Table II
Patient racial origin and age incidence

Racial origin	Total number	0-29	30-59	60+	Number in diagnosed-treated group	Number in autopsy-diagnosed group
Caucasian	26	5(19%)	6(23%)	15(58%)	11(37%)*	15(83%)**
North American Indian	14	7(50%)	4(29%)	3(21%)	13(43%)*	1(6%)**
Asiatic	8	1(13%)	3(38%)	4(50%)	6(20%)*	2(11%)**

*Expressed as a percentage of the total diagnosed-treated group (30 patients)
**Expressed as a percentage of the total autopsy-diagnosed group (18 patients)

Table III
Examination for *Mycobacterium tuberculosis*

			Smear	Culture
		Number examined and cultured	Number positive	Number positive
Diagnosis made during life (30)	Sputum	25	17 (68%)	21 (84%)
	Urine	13	1 (7.7%)	3 (23%)
	Stomach washings	13	1 (7.7%)	7 (54%)
	CSF	2	1	2
	Bone marrow	2	0	2
	Lung biopsy	1	0	1
Diagnosis made at autopsy (18)	Lung tissue	4*	3 (75%)	4 (100%)
	CSF	3**	0	3
	Sputum	1	0	0
	Pleural fluid	1	0	1

*All autopsy specimens
**Two specimens obtained at autopsy

tient in this group cerebrospinal fluid and pleural fluid were both negative for acid-fast bacilli on smear, but positive cultures were obtained after the patient died.

Radiology

Chest radiography was performed in 42 patients, of whom 30 were in the diagnosed and treated group and 12 in the autopsy-diagnosed group. In the diagnosed and treated group there were 28 patients whose chest radiograph showed a characteristic miliary pattern of disease; the remaining 2 patients had negative radiographs.

In the autopsy-diagnosed group no chest radiograph was obtained in six patients. In another six patients, although radiography showed a miliary pattern, a diagnosis of miliary tuberculosis was either considered and rejected or not suspected. Two patients had entirely normal chest radiographs and in the remaining four cases non-miliary abnormalities were found (pleural effusion, basal pleural thickening, opacities in right upper lobe and diffuse nodular opacities).

Tuberculin testing

This was carried out in 30 patients. Using 5 units PPD, 24 positive results were obtained, including two in patients who did not react to initial tuberculin testing.

Hematological investigations

Hemoglobin and leukocyte counts were available for 30 cases and hemoglobin estimation alone in a further two cases. Upper and lower limits for leukocyte count abnormalities were taken from Dacie and Lewis.[11] Results are presented in Table IV. A mild to moderate anemia was present in 72% of the patients.

The most common leukocyte abnormality was lymphopenia which was present in 53% of cases. Polymorphonuclear leukocytosis was found in 27% of cases and was usually associated with a high staff cell count. Other leukocytic abnormalities were relatively uncommon. There were no significant differences in leukocytic abnormalities between the group of diagnosed and treated cases and those diagnosed at autopsy.

Hepatic biochemistry

One or more biochemical tests of liver function were carried out in 16 patients (Table V). Alkaline phosphatase, glutamic oxaloacetic transaminase and lactic dehydrogenase levels were abnormally high in approximately 50% of tested patients. Only one of the patients with an elevated alkaline phosphatase level was an alcoholic.

Hepatomegaly was present in five patients who had one or more tests of hepatic biochemistry performed, and abnormal results were obtained in four of them. Of 11 patients without hepatomegaly, one or more of the liver function tests was abnormal in six.

Outcome

Tables I and VI show the relationship of fatality to age in the two groups of patients. Of the 30 patients in the diagnosed and treated group 23 recovered from their illness, remaining well at follow-up (from six months to five years) with no recrudescence of their disease. In this group four (44%) of the nine patients over the age of 60 died while there were only three deaths (14%) among the 21 patients under the age of 60. Of the seven patients who died, six presented with a fulminant illness which, although correctly diagnosed and treated, led rapidly to death usually within days after admission to hospital. Six of the seven deaths in this group were in men. Of the autopsy-diagnosed group, 13 (72%) of the 18 patients were over the age of 60.

Autopsy material

Autopsies were performed on 23 of the 25 patients. The lung was involved in all cases, the spleen in 16, liver in 14, kidney in 10 and lymph nodes in 9 cases. Brain, meninges and adrenal glands were each involved in five cases. The

Table IV
Hematological findings

Hemoglobin (g./100 ml.)	>14	12-14	10-12	10
Male	5	8	4	5
Female	0	4	4	2
Leukocytosis (>10,000/c. mm.)	9 (30%)			
Polymorphonuclear leukocytosis (>7500/c. mm.)	8 (27%)			
Lymphocytosis (>3500/c. mm.)	1 (3%)			
Monocytosis (>800/c. mm.)	2 (7%)			
Eosinophilia** (>440/c. mm.)	1 (3%)			
Leukopenia (<4000/c. mm.)	2 (7%)			
Neutropenia (<1500/c. mm.)	2 (7%)			
Lymphopenia (<1500/c. mm.)	16 (53%)*			
High staff count (<400/c. mm.)	13 (43%)			

*Includes one patient with leukopenia.
**Patient on prednisone.

Table V
Hepatic biochemistry

	No. of patients	No. of alcoholics
Serum alkaline phosphatase > 35 IU/l. (Normal 9-35 IU/l.)	6 (12)*	1
Serum glutamic oxaloacetic transaminase >35 IU/l. (Normal 5-35 IU/l.)	5 (12)	3
Serum lactic dehydrogenase > 350 IU/l. (Normal 150-350 IU/l.)	6 (12)	3
Serum total bilirubin > 1.0 mg./100 ml. (Normal < 1.0 mg./100 ml.)	2 (5)	2

*Figures in parentheses = number of patients in whom test was performed

Table VI
Fatality and age of treated patients

Age	0-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90+
Total	5	6	4	2	4	4	0	5	0
Dead	0	1	1	0	1	1	0	3	0

pancreas, pericardium, bone and prostate were each involved in two or three cases.

Concomitant disease (see above) was either confirmed or newly diagnosed in 15 of the 23 cases who came to postmortem examination.

Table VII gives, in summary, a profile of the 18 autopsy-diagnosed patients.

Discussion

The present study shows a disconcertingly high rate of failure in diagnosis of miliary tuberculosis during life in British Columbia as in other areas.[2,4,12,13] There are several factors responsible for the failure of recognition. The most important is the insidious nature of the disease as it occurs in the elderly. In the 1946 autopsy series of Chapman and Whorton [14] only 25% of cases were diagnosed ante mortem although the average age was lower and tuberculosis still a common disease. The peak age incidence of miliary tuberculosis has steadily increased over the years;[1,9,13] in the present series it is only in the autopsy-diagnosed group that a marked predominance of elderly patients is found. The high incidence of concomitant disease in these patients is another important factor in the failure to diagnose miliary tuberculosis.[1,15]

Seventeen of the dead patients in the present series had other diseases and 15 belonged to the autopsy-diagnosed group. Atherosclerotic heart disease and alcoholism were particularly common. Corticosteroid therapy appeared to be an important initiating or potentiating factor in four cases; three of these patients died without the diagnosis being made before death. Similar findings have been reported by earlier workers.[16,17] However, concomitant disease significantly contributed to or directly caused only seven of the deaths, all in the autopsy-diagnosed group. It is interesting that two patients died of massive gastrointestinal hemorrhage from acute "stress" gastric and duodenal ulcers.

The common presenting symptoms were general malaise, weakness, weight loss and cough with sputum production, which appeared insidiously over weeks or months; this was also found by Biehl.[9] However, a fulminant course was seen in six patients who, although receiving the correct diagnosis and intensive treatment, died within a few days of admission to hospital. This fulminant course was not seen in the autopsy-diagnosed patients, who usually had an insidious, nonspecific illness, with noticeable absence of respiratory symptoms or signs. Petersdorf and Beeson[18] and Bottiger[5] described tuberculosis (usually miliary) in 11% of the patients admitted for investigation of fever of unknown origin and this mode of presentation was found in six patients (13%) in the present series, four of whom were in the autopsy-diagnosed group.

Fever was the single most frequent physical finding in our cases, being present in 76% of the patients. Crepitations were present in 54%, a figure slightly lower than that found by Biehl.[9] Nine patients (three of them alcoholics) had hepatomegaly. Unlike Litten[19] who found splenomegaly in 70% of cases, our incidence was only 5%. Retinal tubercles, found by Carpenter and Stephenson[20] in 75% of cases of miliary tuberculosis, were only recorded in one patient. This probably represents failure of thorough ophthalmoscopic examination, the importance of which has been stressed by Illingworth and Wright[21] and Chapman and Whorton.[14]

Tuberculin anergy is often associated with miliary tuberculosis.[9] In the present series only six of the thirty patients in whom tuberculin testing was carried out showed negative reactions to 5 units of PPD. Of the 18 patients diagnosed at autopsy, tuberculin testing was carried out in only three, further emphasizing the failure to consider tuberculosis in an undiagnosed illness.

There was a high incidence of leukocytic abnormalities, i.e. lymphopenia, polymorphonuclear leukocytosis and a high staff count, in this series. Other leukocytic abnormalities, e.g. agranulocytosis and leukemoid reactions,[6] neutropenia and moncytosis[1] were not seen. The polymorphonuclear leukocytosis is unexpected and cannot be explained by concomitant bacterial infection, which was definitely present in only two such patients. Oswald,[6] however, described a relatively common transient polymorphonuclear leukocytosis which was usually associated with a secondary bacterial infection, and Chapman and Whorton[14] described polymorphonuclear leukocytosis in 89% of their cases. The latter workers also found lymphopenia and high staff cell counts as common leukocytic abnormalities. The mild to moderate degree of anemia seen in 72% of the patients is in agreement with the findings of Chapman and Whorton[14] and Hegler.[22] Abnormalities of liver biochemistry were demonstrated in over half of the relatively small number of pa-

Table VII
Profile of the 18 cases discovered at autopsy

Age	0-49	1
	50+	17
Sex	Female	6
	Male	12
Racial origin	North American Indian	1
	Caucasian	15
	Asiatic	2
Presented as pyrexia of unknown origin		4
No bacteriological examination ante mortem		16
No chest radiography		6
Chest radiology showing non-miliary changes (including negative radiographs)		6
No tuberculin test performed		15
Concomitant diseases		15
Concomitant diseases contributing to or causing death		7
Patients on prednisone		3

tients in whom one or more tests of liver function were performed. In agreement with the findings of Brunner and Haemmerli[7] the most common abnormality was an elevated serum alkaline phosphatase, but SGOT and lactic dehydrogenase elevations were also seen in a significant proportion of patients.

The great majority of the patients in the diagnosed and treated group had a classic miliary pattern on chest radiography. In the autopsy-diagnosed group six patients had characteristic miliary shadowing in chest radiographs which were incorrectly diagnosed, while six patients had radiological changes other than a miliary pattern or a negative radiograph. A proportion of the autopsy-diagnosed patients probably represent the "cryptic" group of patients described by Proudfoot *et al*,[1] patients who have an insidious illness with the absence of miliary mottling in the chest radiograph. One aspect of the concept of "cryptic" disease is supported by the work of Berger and Samortin[8] who, in reviewing the literature, found 99 normal initial chest radiographs in 290 patients who eventually were proved to have miliary tuberculosis.

Bacteriological confirmation of the diagnosis was obtained in 70% of patients and in seven cases this came from autopsy material. In the diagnosed and treated group, positive cultures were obtained in 85% of the cases and there was a surprisingly high incidence of positive smears (66%). The high yield of positive cultures from sputum, urine, stomach washings and cerebrospinal fluid suggests that widespread use of bone or liver biopsy is unnecessary. On the other hand, Brunner and Haemmerli[7] report 10 out of 12 positive liver biopsies in cases with diagnostic difficulty; all these patients were treated and survived as compared with a further 12 cases in which no biopsy had been performed and the diagnosis was disclosed only at autopsy. It is probable that some of the cases not identified before death would have been revealed by liver or bone biopsy.[23]

North American Indians have been shown to have a high disease rate but low mortality from miliary tuberculosis as compared to Caucasians. Tuberculosis is 10 times more common among Indians,[24] and has a particularly high incidence among Indian children.[25] The lower mortality rate in the Indian population is probably due to the younger age at which the disease occurs, and the more obvious clinical presentation in the majority of cases which leads to greater ease of diagnosis. A similar situation has been described by Falk[26] in Negro and Caucasian populations in the United States.

The present report confirms a disturbingly high autopsy diagnosis rate for miliary tuberculosis in British Columbia, in accordance with other Western experience. Insidious onset, lack of diagnostically significant physical findings, and a high incidence of concomitant disease in an increasingly elderly population are largely responsible for this situation.

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Résumé

La tuberculose miliaire en Colombie britannique

La présente étude passe en revue les dossiers cliniques de 48 malades souffrant de tuberculose miliaire enregistrés de 1967 à 1971 par la British Columbia Division of Tuberculosis Control. On a classifié les malades en deux groupes: un groupe de 30 malades dont le diagnostic avait été porté pendant leur vie et qui avaient reçu une médication antituberculeuse et un second groupe de 18 malades dont la maladie n'avait été diagnostiquée qu'à l'autopsie et qui n'avaient reçu aucune médication antituberculeuse. Dans ce dernier groupe, on notait une fréquence particulièrement élevée de pathologies concomitantes. De plus, l'épreuve à la tuberculine, les recherches bactériologiques de bacilles acido-résistants et les radio-

graphies pulmonaires étaient souvent négligées. Ce groupe était surtout composé d'hommes âgés de race blanche. La maladie avait comme caractéristiques un début insidieux, une symptomatologie et des signes cliniques manquant souvent de spécificité. La fièvre était un symptôme courant. Plusieurs de ces malades ont présenté une maladie foudroyante qui, bien que traitée, n'a pas réagi au traitement antituberculeux et qui eut rapidement une issue fatale. La maladie, très fréquente chez les Amérindiens, n'a été frappée que d'une faible mortalité, probablement en raison du jeune âge des malades.

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FORTHCOMING MEETINGS

CANADA

ANNUAL SCIENTIFIC FALL MEETING, ONTARIO MEDICAL ASSOCIATION, ANAESTHETIC SECTION, ONTARIO DIVISION. Sarnia, Ont. September 20-22, 1973. Information: Dr. K. Panwar, Box 2222, Sarnia, Ont. N7T 7L7

ANNUAL MEETING, ONTARIO SOCIETY OF MEDICAL TECHNOLOGISTS. Ottawa. September 27-28, 1973. Information: Dr. Marie Aprile, Dept. of Surgery, Banting Institute (Room 322), 100 College St., Toronto 5, Ont.

OTHER COUNTRIES

9TH INTERNATIONAL CONGRESS OF BIOCHEMISTRY. Stockholm. June 28-July 4, 1973. Information: Mrs. G. Aulin-Erdtman, Swedish Society of Chemistry, Wenner-Gren Center VI, S-113 46 Stockholm, Sweden.

INTERNATIONAL SYMPOSIUM ON THE IDENTIFICATION OF TABLETS. Pont à Mousson, France. June 30-July 1, 1973. Information: J.-F. Lorentz, Centre Anti-Poison, Centre Hospitalier Regional, 54, Nancy, France

THE HANS BERGER CENTENARY SYMPOSIUM ON EPILEPSY. Edinburgh, Scotland. July 1-5, 1973. Theme: The Natural History and Management of Epilepsy. Information: Kurt Fleischmann, Symposium Secretariat, Kurt Fleischmann and Associates, 164 North Gower St., London, NW1 2ND.

VIIITH INTERNATIONAL CONFERENCE ON HEALTH EDUCATION. Paris. July 8-14, 1973. Information: Secretariat, VIIIth International Conference on Health Education, 20 rue Greuze, 75 Paris 16e, France