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PECULIARITIES OF THE CLINICAL COURSE OF PNEUMONIA FOLLOWING POISONING WITH BERYLLIUM COMPOUNDS

By

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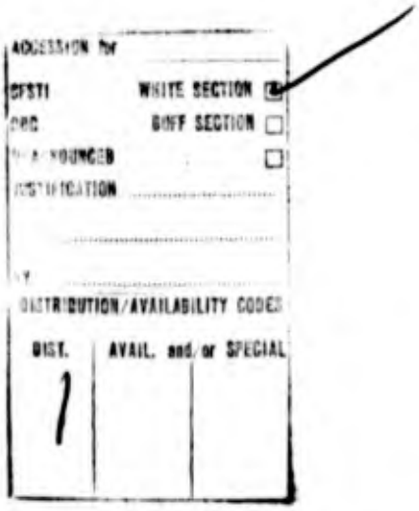

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ABSTRACT Pneumonia, which is the most serious and rather frequent complication of poisoning by beryllium compounds, develops in interstitial tissue against the background of toxic lesion of alveolae and bronchioles, which leads to partial atelectasis and swelling of separate pulmonary areas with considerable increase in the permeability of the pulmonary vessels. Development of pneumonia usually involves association of a secondary pneumotropic infection or activation of the primary one, which then acquires virulent properties.

In view of essential changes of reactance of the organism, most beryllium pneumonias run the course of a hypoergic type without appreciable rise in body temperature, with moderate leucocytosis or normal leucocyte count, and without special shifts in the rods and nuclei. There is observed arterial hypoxemia, hyperventilation, and proctivity towards marked tachypnea, tachycardia, hypotonia and collapse-like conditions. Due to the protracted course of the pneumonic process the functional capacity of the respiratory and hemodynamic systems is slow to recover its normal vigor.

Treatment with antibiotics even of a broad spectrum is not sufficiently effective. Only prolonged combined sulfanilamide and antibiotic mediation used together anti-allergic bronchodilating and cardiovascular agents proves beneficial.

English translation: 6 pages.

U. S. BOARD ON GEOGRAPHIC NAMES TRANSLITERATION SYSTEM

Block	Italic	Transliteration	Block	Italic	Transliteration
А а	*А а*	A, a	Р р	*Р р*	R, r
Б б	*Б б*	B, b	С с	*С с*	S, s
В в	*В в*	V, v	Т т	*Т т*	T, t
Г г	*Г г*	G, g	У у	*У у*	U, u
Д д	*Д д*	D, d	Ф ф	*Ф ф*	F, f
Е е	*Е е*	Ye, ye; E, e*	Х х	*Х х*	Kh, kh
Ж ж	*Ж ж*	Zh, zh	Ц ц	*Ц ц*	Ts, ts
З з	*З з*	Z, z	Ч ч	*Ч ч*	Ch, ch
И и	*И и*	I, i	Ш ш	*Ш ш*	Sh, sh
Й й	*Й й*	Y, y	Щ щ	*Щ щ*	Shch, shch
К к	*К к*	K, k	Ъ ъ	*Ъ ъ*	"
Л л	*Л л*	L, l	Ы ы	*Ы ы*	Y, y
М м	*М м*	M, m	Ь ь	*Ь ь*	'
Н н	*Н н*	N, n	Э э	*Э э*	E, e
О о	*О о*	O, o	Ю ю	*Ю ю*	Yu, yu
П п	*П п*	P, p	Я я	*Я я*	Ya, ya

* ye initially, after vowels, and after ъ, ь; e elsewhere. When written as ё in Russian, transliterate as yё or ё. The use of diacritical marks is preferred, but such marks may be omitted when expediency dictates.

PECULIARITIES OF THE CLINICAL COURSE OF PNEUMONIA FOLLOWING POISONING WITH BERYLLUIM COMPOUNDS

L. N. Belyayeva

Submitted
23 March 1963

There are reports of a number of authors about pulmonary leisons appearing during the influence of beryllium compounds; they are defined as bronchiolitis, bronchiol-alveolitis, acute pneumonitis and chronic pulmonary interstitial granulomatosis (B. I. Martsinkovskiy; De Nardi; Hardy and Tabershaw; Dutra; Aub and Grier). According to the character of its development the disease is divided into two forms; acute and chronic. There remains a controversial question about whether these forms are independent or whether interrelations exist between them.

During the acute form of the disease, characteristic changes in the lungs are repletion of capillaries with blood, separate focal hemorrhages, marked ederna inside alveolae, appearance of cellular exudate and fibrin. During the chronic form the pathological process is also diffuse and equally strikes both lungs. Special attention should be given to development of subsequent pathological processes, played against the background of the primary tissue reaction of pulmonary tissue to the toxic agent. Ederna of bronchi, bronchioles and alveolae and a decrease of blood supply induced by this create favorable soil for introduction of pathogenic microorganisms. Alveolar hypoventilation and anoxia of alveolar tissue cause reflex spasm of pulmonary arterioles - alveolar-vascular reflex - which also promotes decrease of blood supply. All of this favors development of infection and leads to prolonged course of inflammatory process in lungs.

By our information, complication of pneumonia is observed for every fifth patient who has been poisoned. Analysis of clinical manifestation and course of pneumonia following poisoning by beryllium compounds for 60 patients permits noting a number of peculiarities of the disease. First of all one should note that beryllium pneumonia

develops in interstitial tissue of lungs and breaks out as a superimposition on the toxic lesion of alveolae and bronchioles, leading to partial atelectasis and swelling of individual pulmonary areas with considerable increase in permeability of pulmonary vessels. As our preceding observations showed, reactance of organism is essentially changed, and majority of beryllium pneumonias are of the hypoergic type. As a rule, body temperature is subfebrile and rarely exceeds 38°. Development of pneumonia usually involves association of secondary pneumotropic infection or activation of microbic flora, which acquire virulent properties.

The beginning of the pneumonic process is marked by strengthening of criteria, peculiar to toxic bronchiolitis. Dyspnea sharply increases, there appears early heavy tachypsnea not corresponding to body temperature, there appears cyanosis, pains in chest take on a compressing character, and coughing increases. The quantity of phlegm, as a rule, is small, and it rarely acquires a purulent character. There is frequently detected in it increased contents of eosinophils and abundant microflora with presence of hemolytic streptococcus and nonhemolytic staphylococcus, which are usually not very sensitive to such antibiotics as biomycin, penicillin and streptomycin. Pulmonary lesion is usually bilateral and at the beginning of the disease is localized in the lower areas. Above the affected sections of lungs percussion tone has a tympanic, flat character. Breathing is rigid with prolonged expiration, in some cases bronchial; there are heard crepitant rales reminiscent of the sounds made during bronchiolitis and preserved after elimination of the pneumonic process. Frequently the inflammatory process spreads to new areas of the lungs, where all the physical symptoms which were mentioned above will appear.

An essential role in the clinic of the disease is played by asthmatic phenomena, which aggravate its already difficult course. There has been noted a marked lability of the cardiovascular system with proclivity towards tachycardia, hypotonia and states resembling collapse. The disease runs its course with moderate leucocytosis or with normal leucocyte count, without shift in rods and nuclei or with small shift: erythrocyte sedimentation rate is usually accelerated and reaches 30-40 mm per hour.

During X-ray investigation there are determined acinous shadows, usually in the range of middle and lower belts, not infrequently confluent among themselves. There also appear areas of emphysema, which mask focuses of blackout. These X-ray changes undergo reverse development very slowly and can be observed for a period of 2-6 months in spite of energetic therapy.

The functional capacity of the pulmonary and cardiovascular systems is considerably disturbed. During such a kind of pneumonias, there is usually arterial hypoxemia and great hyperventilation with the patient breathing shallowly and frequently. As a rule, it is possible to note protracted course of pneumonias and slow recovery of functional capacity of respiratory and hemodynamic systems. Even in those cases in which temperature of body and blood is normalized, the general condition of the patient is improved and there is no shortness of breath in dormancy; shifting him to semibed conditions again

causes shortness of breath. Unlike ordinary pneumonias it is necessary to keep the patient in stationary conditions from 1 to 2 months and longer.

The essential peculiarity of therapy of beryllium pneumonia consists in insufficient effectiveness of antibiotics even with wide range of action. Only prolonged combined sulfanilamide and antibiotic therapy together with antiallergic, bronchodilating and cardiovascular means gives a positive effect. Undoubtedly, there is shown the application of oxygen therapy. In view of the tendency of patients toward hypoglycemia and hypoproteinemia, necessary medical measures are intravenous infusion of glucose and proteinic nutrition (curds with pancreatin).

The question about outcomes of disease deserves serious attention. Pneumonia is especially dangerous when ever new parts of the pulmonary parenchyma become inflamed and total pneumonia sets in. We were able to keep track of the state of health of patients who had contracted pneumonia for a sufficiently long period of time - from 1 year to 10 years. For 30% of them, there was developed pneumoschlerosis, in a number of cases complicated by bronchiectases, frequent pneumonic flareups and asthmatic bronchitis. The disease proceeded with phenomena of chronic pulmonary deficiency. By X-ray it was possible to observe diffuse cellular pneumoschlerosis of different prevalence, sometimes with the presence of bronchiectasis. In period of pneumonic flareup in lungs there were again determined spotty darkening, sometimes taking on mixed character.

For illustration we will give a case history.

Female patient M., 22 years old, entered clinic November 16, 1949 complaining of shortness of breath, chest pain coughing bringing up a small amount of phlegm, bad appetite, weakness, and weight loss. These phenomena began to be observed already on November 1, but the patient continued to work; on December 12 in connection with deterioration of her health, she went to a dispensary, whence she was sent to clinical therapy.

The patient is pale, her lips are cyanotic and her nutrition is considerably lowered. In inferolateral pulmonary areas, breathing is rigid, with bronchial shade, there are heard crepetant roles, and the breathing rate is 25 per minute. Limits of the heart are normal, tones are deaf. Arterial pressure is 100/60 mm., pulse of average filling, rhythmical, 88 beats per minute. Liver on 3 cm lower than costal arc, average density, sickly.

X-ray photograph of pectoral cell on November 21: roots of lungs are expanded, insufficiently structural, illegibly outlined; during the period of average and lower belts against the background of diffuse lowering of pneumatization, there is strengthened the vascular-interstitial figure and its architectonics is disturbed. Furthermore, against the background of these changes there are seen numerous, dispersed average-focus changes, more expressed on the right. The diaphragm is badly differentiated.

Analysis of blood: Hb 60 g%, erythrocyte 4,000,000, leucocyte 5700, eosinophil 0.5%, protein 9%, serum 68%, lymph 19%, monocyte 3.5%; erythrocyte sedementation rate 23 mm per hour. In urine there are traces of protein, in precipitate there are single lixiviated erythrocytes.

Clinical X-ray data allowed treating the condition of the patient as beryllium compound poisoning complicated by bilateral infra-lobar pneumonia.

The patient received a combined treatment with sulfanilamide preparations, antibiotics, cardiovascular agents and oxygen therapy, which brought about some improvement; however, on November 26, the condition of the patient grew worse, there appeared severe tachypnea, breathing rate increased (44 per minute), pulse was 92-100 beats per minute, for 3 days body temperature stayed at the level of 38.2-38.5°. In lungs, in the upper left part, there was determined a new pneumonic area. By November 30, the condition of the patient had improved somewhat, breathing rate decreased (30 per minute), pulse slowed to 88-90 beats per minute, body temperature was subfebrile, but physical data in lower parts of both lungs and in upper part of left one remained as before: breathing was bronchial; there were heard crepitant rales. All of this made it necessary to continue the former combined treatment. On December 6, in connection with the appearance of a pneumonic area in the middle of the right lung, the condition of the patient became extremely serious: breathing rate climbed to 44-55 per minute, pulse rate rose to 120-140 beats per minute, severe hypoxemia developed, and the patient could not live without inhaling oxygen. Assimilation on December 15 of a new pneumonic area in the upper right part led to development of total pneumonia in all 5 parts of both lungs. On December 16 the patient died during phenomena of marked pulmonary and cardiovascular deficiency.

Clinical diagnosis: poisoning by beryllium compounds, toxic bronchiolitis complicated by bilateral total pneumonia; pulmonary edema; dystrophia of myocardium; toxic hepatopathy and nephropathy.

Pathoanatomical diagnosis: bronchiolitis; confluent macro-focal pneumonia; fibrous interlobitis; pulmonary edema; degeneration of the myocardium; fatty degeneration of liver and kidneys; hyperplasia of the pulp of the spleen; atrophic gastritis.

Microscopic examination of the lungs following poisoning by beryllium compounds reveals a multitude of pathological changes. Attention is attracted by areas with presence of serous exudate in cavity of alveolae and in interstitial tissue. In alveolae there is serous exudate or minimum quantity of big mononuclear cells during preserved parietal lining. Group of such alveolae surrounded by granulation tissue is somewhat similar to atypical structure of adenic tissue. There are rarely encountered gigantic multinuclear cells and inclusions of different configuration colored dark blue by hemotoxylin, which are phagocytic or freely lying among cells. Inclusions are similar to conchoidal corpuscles.

In other group of alveolae among serous exudate along with big mononuclear cells there are histicytes, plasma cells, fibroblasts and desquamated, cubiform cells covering alveolae as manifestation of efficent inflammation. In considerable group of alveolae, intraalveolar exudate is carried out by fibrous masses with phenomena of its organization. In each alveola there is papillary growth. In group of such alveolae this simulates picture of intracanalicular papillary adenoma. In most of cavity of alveolae, tissue is subjected to fibrosis. These sections constitute picture of granulation tissue with presence in it of plasma and epithelial cells, monocytes, lymphocytes, fibroblasts and connective-tissue fibers. On maturity granulation tissue is different. In places it is mature and fibrous; in places it has many small blood vessels. In it there are little nodular foci of histiocytes and lymphocytes. In certain sections fibrosis is expressed by a picture of carnification. Amid the cellular composition of connecting tissue there are seen slots covered by cubiform epithelium (accommodation of alveolar epithelium).

There is also rathery marked catarrhal-desquamative reaction in alveolae with subsequent organization of intraalveolar exudate. In certain sections the contours of modified alveolae are preserved, and in larger part are absent due to development of connecting tissue. There is noted clearly expressed unspecific inflammatory efficent reaction from the side of interstitial tissue. Essential changes are detected in bronchi. Not infrequently the bronchi (more frequently the small ones) are destroyed. The opening left by them is filled with slime and various cells, including epithelium which has peeled. Sometimes these masses undergo fibrosis and cause obliteration of bronchi. Emphysematous sections have considerable cavities with remains of torn alveolar partitions.

All these changes characterize bronchopneumonia with early development of fibrous changes and absence of polymorphous-nuclear leucocytes. Along with bronchopneumonic process, it is possible to detect development of the infiltration-proliferation process in interstitial tissue with presence in it of lymphocytes, histiocytes and cells of the epithelial type, and also cells of connective-tissue series with subsequent development of powerful connective-tissue fibers. Similar changes can be treated as chronic interstitial pneumonia.

Changes in kidneys are expressed by picture of nephrosis-nephritis, and changes in liver are expressed by edema of Disse's spaces, granular degeneration of liver epithelium and even intralobular necrosis. In heart along with hypertrophy of myocardium there is detected ederna of intermediate intumescence of endocardum.

Conclusions

1. Pneumonia, which is the most serious and rather frequent complication of poisoning by beryllium compounds, develops in interstitial tissue against the background of toxic lesion of alveolae and bronchioles, which leads to partial atelectasis and swelling of separate pulmonary areas with considerable increase in the

permeability of the pulmonary vessels. Development of pneumonia usually involves association of a secondary pneumotropic infection or activation of the primary one, which then acquires virulent properties.

2. In view of essential changes of reactance of the organism, most beryllium pneumonias run the course of a hypoergic type without appreciable rise in body temperature, with moderate leucocytosis or normal leucocyte count, and without special shifts in the rods and nuclei. There is observed arterial hypoxemia, hyperventilation, and proctivity towards marked tachypnea, tachycardia, hypotonia and collapse-like conditions. Due to the protracted course of the pneumonic process the functional capacity of the respiratory and hemodynamic systems is slow to recover its normal vigor.

3. Treatment with antibiotics even of a broad spectrum is not sufficiently effective. Only prolonged combined sulfanilamide and antibiotic mediation used together anti-allergic bronchodilating and cardiovascular agents proves beneficial.

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