Bronchopneumonia Due to Hemolytic Streptococci Following Influenza

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ERNEST W. GOODPASTURE, M.D.
(BOSTON)
Lieutenant (Junior Grade), M. C., U. S. N. R. F.
CHELSEA, MASS.

BRONCHOPNEUMONIA DUE TO HEMOLYTIC STREPTOCOCCI FOLLOWING INFLUENZA *

ERNEST W. GOODPASTURE, M.D. (Boston)
Lieutenant (Junior Grade), M. C., U. S. N. R. F.
CHELSEA, MASS.

The bacteriology and pathology of bronchopneumonia during the last two months (December and January) have become quite different, at the Chelsea Naval Hospital, from that found during the preceding three months, including the most severe period of the first local outbreak of influenza. In the earlier period, cultures of Pfeiffer's bacillus were obtained from the lungs in 86 per cent. of cases postmortem, the organism often being found in pure culture, pneumococci in 50 per cent., and hemolytic streptococci in 13 per cent.; while during the last two months, cultures of hemolytic streptococci have been obtained from the lungs in 100 per cent. of those examined at necropsy, and in the majority of instances pure cultures were obtained. Pfeiffer's bacillus was demonstrated in only two cases of a total of sixteen, and these two were examined the first week of December.

This marked variation in the type of invading organism in bronchopneumonia is of particular interest since it represents the experience of a single station during a relatively short period of time; and we are now familiar with wide differences in bacteriologic observations from various sections of the country since the epidemic of influenza began.

From the twenty-six cases studied during the height of the epidemic in the early fall, we became more or less familiar with a type of pneumonia which was fairly typical of that series, and were able to recognize many modifications obviously due to secondary invasion by pathogenic organisms. An acute bronchiolitis

* From the U. S. Naval Hospital.

characterized by the presence within the bronchioles of a dry exudate consisting of a few leukocytes, polymorphonuclear and mononuclear, mucus and fibrin, usually with partial or complete necrosis of the mucous membrane, and the presence of a characteristic irregular hyaline membrane on the infundibular and alveolar walls, were regarded as typical early lesions. Bronchopneumonia following this was fulminating in type and consisted in a widespread toxic injury to alveolar tissues, so that they became filled with fluid exudate, fibrin, and hemorrhage with extensive injury to alveolar walls and capillaries. From a study of these cases we stated that "the pulmonary injury and reaction being so acute and often widespread, and the fact that in certain very early cases bacteria of any kind are so scarce or not found at all, make us feel, notwithstanding the demonstration of influenza bacilli in pure culture in the lungs in all but one instance, that at this stage organisms are as yet comparatively few within alveoli, and their primary injury is due to a very potent toxic substance elaborated in and disseminated through larger air passages." Following this initial injury, pathogenic bacteria readily invaded the pulmonary tissue, producing injury and reaction more or less typical for the organism concerned. In striking contrast to the group of cases that constitutes the material for the present report, hemolytic streptococci at that time played a relatively small rôle as a secondary invader, complicating only five, or 13 per cent., of the cases. In three of these, multiple abscesses were present in the lungs, and in the other two streptococci and influenza bacilli were associated in the production of widespread bronchiectatic abscesses and ulcerative bronchitis.

The present report is based on the postmortem examination of sixteen cases, constituting all the cases of bronchopneumonia examined at necropsy during the past two months. Eleven of the patients developed empyema, and the pleural exudate yielded pure cultures of hemolytic streptococci.

The accompanying table presents these cases. Although some of the patients entered the hospital with well-developed bronchopneumonia, in several instances the clinical diagnosis of influenza or post-influenzal pneumonia was made.

In five cases the empyema was bilateral, in four on the left, and once on the right side. In each instance bronchopneumonia was bilateral. One case showed an extensive streptococcal pneumonia in each lung

CASES OF BRONCHOPNEUMONIA WITH PATHOLOGIC DIAGNOSIS

Case No.	Range of Leukocyte Counts	Pathologic Diagnosis	Bacteriology of Lungs	Clinical Diagnosis on Admission
1	6,600-12,800	Bronchopneumonia; no empyema	Influenza bacilli; hemolytic streptococci; pneumococcus Type IV	Influenza
2	15,000-13,000	Bronchopneumonia; empyema (bilateral)	Hemolytic streptococci; nonhemolytic streptococci; influenza bacilli	Influenza; bronchopneumonia
3	3,200	Bronchopneumonia; no empyema	Hemolytic streptococci	Influenza
4	13,000-48,000	Empyema (bilateral); pericarditis; slight pneumonia	Hemolytic streptococci	Bronchopneumonia
5	25,000	Bronchopneumonia; peritonitis; empyema	Hemolytic streptococci	Bronchopneumonia
6	12,000	Bronchopneumonia; empyema (bilateral)	Hemolytic streptococci	Bronchopneumonia; empyema
7	4,700-3,400	Bronchopneumonia; empyema (bilateral)	Hemolytic streptococci	Influenza
8	6,900	Bronchopneumonia; empyema (left)	Hemolytic streptococci	Influenza; bronchopneumonia
9	4,000-15,000	Bronchopneumonia; empyema (bilateral); peritonitis	Hemolytic streptococci	Influenza
10	8,000-10,200	Bronchopneumonia; empyema (bilateral)	Hemolytic streptococci	Empyema; bronchopneumonia
11	7,000	Bronchopneumonia; empyema (left)	Hemolytic streptococci	Influenza
12	16,800	Bronchopneumonia; empyema (left)	Hemolytic streptococci	Empyema; bronchopneumonia
13	5,000-8,200	Bronchopneumonia; no empyema	Hemolytic streptococci	Influenza
14	9,000-19,200	Bronchopneumonia	Hemolytic streptococci	Influenza
15	3,000-6,000	Bronchopneumonia	Hemolytic streptococci	Influenza
16	5,000	Bronchopneumonia	Hemolytic streptococci	Influenza

with no pleural exudate, and is of interest because numerous fibrous adhesions were present over the lungs, suggesting the possibility of an increased resistance to the infection because of previous inflammation.

The lungs were moist and congested, often atelectatic where pleural exudate was voluminous. The

areas of consolidation were grayish purple, and cut surfaces exuded a fairly thick, purplish pus. In the early stages of pleural inflammation the exudate is serous, containing relatively few cells and organisms; later it becomes thick pus containing enormous numbers of streptococci. In four cases the infection was very prominent in the lymphatics and interlobular tissue of the lungs. The lobules in areas of considerable size were found outlined by wide (from 3 to 4 mm.) yellow bands. These bands followed the veins and bronchi to the hilum. The lymphatics of the pleura may also be filled with streptococci. Abscesses have occasionally been found. Microscopically the alveoli are filled with polymorphonuclear leukocytes and usually enormous numbers of streptococci, with little or no fibrin. Often there is widespread necrosis. In no instance have we observed the appearances of interstitial bronchopneumonia, described by MacCallum, which occurs so frequently as a sequel to measles. The distribution of the pneumonia is usually lobular and rather massive. In cases of short duration it may be lobar in distribution and involve the greater part of the lung tissue. Nor have we recognized the toxic lesions of early influenzal pneumonia as seen during the first weeks of the epidemic, and I regard all the cases in this report as instances of primary streptococcus pneumonia frequently following an attack of influenza.

The question naturally arises whether some of these cases were not primarily streptococcus infections without influenza. Eleven of the sixteen had attacks typical of influenza which terminated in pneumonia; the diagnoses of the other five were questionable as to an attack of influenza. In view of the experiences last year at various stations of epidemic streptococcal infections of the respiratory tract, it does not seem unlikely that similar outbreaks will occur or are occurring this winter, and with the impressions of influenza so recently in mind there will probably be considerable confusion of the two diseases, especially as there do not appear to be any very certain criteria which distinguish them, particularly when bronchopneumonia is present. Symptoms of onset, leukocyte count and the fever chart may be quite similar, although apparently there

is usually more complaint of sore throat with streptococcal infections.

At this hospital, during the period covered by the present report, hemolytic streptococci have been exceedingly prevalent in various forms of inflammation, and this unusual prevalence explains in part their high incidence as a cause of bronchopneumonia attacking persons whose resistance is lowered by influenza.

The absence of the pathologic evidences of a preceding or accompanying toxic inflammation of the lungs, which in the early part of the epidemic of influenza appeared fairly characteristic of the pulmonary lesions of this disease per se, makes me regard the cases of the present series as examples of primary streptococcal pneumonia.

Bronchopneumonia following influenza has at present absolutely no etiologic or pathologic definition, for the type of organism concerned and the type of lesion in the lungs are decidedly variable at different times even at the same station. It is essential to bear this fact in mind in any consideration of the pneumonic complications of influenza, especially from the standpoint of prospective treatment.

Reprinted from The Journal of the American Medical Association
March 8, 1919, Vol. 72, pp. 724 and 725