Transitory Pulmonary Infiltration
(Loeffler's Syndrome) *

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In 1932, Loeffler1 described a series of five cases in which pulmonary shadows appeared and disappeared rapidly. Extensive lung changes were accompanied by minimal clinical symptoms and by high grade blood eosinophilia. In 1936, he2 reported additional cases bringing the total number observed by him to 51. The symptom complex was recognized by its three chief features: (1) a roentgen shadow in the lung fields; (2) the transitory or migratory character of the infiltrations; and (3) the accompanying blood eosinophilia which ranged from 6 to 66 per cent. Fourteen of his 51 cases were discovered on routine fluoroscopic examinations of patients who had no symptoms directly referable to the chest. This surprising absence of constitutional disturbance was regarded by Loeffler as a fourth, and not unessential diagnostic feature. Acoustic signs usually were slight or might be completely lacking. In many cases, there was an irritative cough, often of considerable intensity, with or without stabbing chest pain. Expectoration was lacking or scant, yellowish, muco-albuminous, but poor in cells. Occasionally, but not always, a few eosinophile cells were found. When sputum was obtained, it was always negative for tubercle bacilli. In rare instances, he observed pleural participation in the process, and still more rarely, a very small circumscribed pleural effusion, distinguishable in the roentgenogram or on fluoroscopy from the infiltrations. These pleural phenomena were likewise fleeting in character. The infiltrations observed were compared by Loeffler to the transitory lesions of erythema nodosum. In both conditions, fleeting inflammatory reactions occur, and he believed them to be the result of an allergic tissue reaction to many different antigens.

Following Loeffler's description, a number of similar reports have appeared in the literature and the cumulative evidence has made it increasingly clear that the syndrome results from an allergic tissue reaction as was hypothesized by him. As was predicted, a great variety of noxious agents such as ascaris,3 trichinal,4 and amoebic infestation,5,6 brucellosis,7 the pollen of privet,8 pronitosil,9 and gold10 have been reported as causative agents.

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Because the condition is essentially benign, the histological characteristics remained conjectural until 1942 when H. von Meyenburg\(^1\) described eosinophilic pulmonary infiltrations found in four patients after accidental death. These infiltrations varied in form and shape, and they varied likewise in location. High grade eosinophilia in the exudative-inflammatory lung nodule was common to all. Eosinophilic bronchitis and bronchiolitis was present in two cases, but was lacking in the other two. Based on the lesions found, von Meyenburg expressed his belief that such infiltrations were not necessarily always transitory. Harkavy\(^2\) reported a series of asthmatic patients in whom polyvalent sensitization was present. Symptoms were precipitated synergistically in at least seven of these patients. In one patient, subcutaneous injection of an autogenous vaccine composed of various bacteria grown from sinus washings repeatedly was followed by asthma, pulmonary infiltrations and petechiae which were regarded as indicative of an hyperergic vascular response. He emphasized that symptoms in bacterial allergy may be reactivated not only by the specific bacteria responsible for the initial sensitization, but also by heterologous bacteria, nonbacterial antigens and possibly by viruses. Such heterologous reactivation of symptoms is comparable to the Sanarelli-Schwartzman phenomenon. Patients with multiple shock tissues reacted not only in the bronchi, but in vessels of the pulmonary parenchyma and other tissues as well and characteristic transitory pulmonary infiltrations of the Loeffler type were observed repeatedly. The milder vascular reactions were regarded as reversible, and in eight patients of the group reported by Harkavy, removal of offending agents was followed by arrest and reversal of the disease process. However, repeated attacks resulted in chronic allergic inflammatory changes in the interalveolar septa and progressive vascular changes.

Allergic infiltrations are not confined to pulmonary tissues alone. von Meyenburg described eosinophilic infiltration of an epididymis removed surgically from a patient in whom transitory pulmonary infiltrations had been observed previously. Likewise, brawny infiltrations were observed by Harkavy in the thigh of a patient who had fleeting pulmonary infiltrations accompanying asthmatic attacks. Biopsy showed these lesions to be eosinophilic infiltrations. Klopotock and Steinitz\(^3\) reported similar migratory reddened swellings which recurred in scattered areas of the body and were accompanied by increased leucocyte counts varying from 12,000 to 15,000 and high grade eosinophilia.

Marked perivascular collections of leucocytes were observed in the nodular infiltrates in two of von Meyenburg's cases. Harkavy emphasized the periarterial lesions in his fatal cases. Periarterial
lesions in association with transitory pulmonary infiltrations also were observed by Lindberg and Baggenstoss\textsuperscript{14} in a 59 year old woman whose death from asthma occurred seven years after her first attack. For several months transitory pulmonary shadows of varying size had been accompanied by eosinophilia as high as 35 per cent. Necrotizing arteritis and arteriolitis were found at necropsy.

Three cases of transitory and migratory pulmonary infiltration which fulfill the criteria established by Loeffler are presented. In each instance there is a background of extrinsic asthma. The first two patients exemplify the early, completely reversible stage generally regarded as characteristic of Loeffler's syndrome. The third patient demonstrates a later stage in which roentgenograms now indicate increasing residual fibrosis of pulmonary tissues.

CASE REPORTS

\textit{Case 1:} W.A.M., Male, age 27. Since his first year there have been recurrent attacks of asthma some of which were caused by contact with danders and dust. Symptoms were somewhat more severe during summer months, although attacks occurred throughout the entire year. Nasal polyps were removed at age twelve. At seventeen, he was given injections of dust, grass, and ragweed antigen for three years without appreciable improvement. For the past six years, Tucker's asthma spray has been used with moderate symptomatic relief. Infantile eczema which appeared during his first year cleared when he was two. At puberty, eczema recurred and has persisted in mild form since. Slight dermatitis was present over both malar regions, and on the neck when he was first seen. The nasal mucous membranes were pale, boggy, and appeared typically allergic. The sinuses transilluminated well. Inconstant asthmatic whistles were heard over the chest. Heart sounds were normal with blood pressure 118/74, and vital capacity 3.5 liters. Mantoux test with 0.1 mg. old tuberculin was positive. Sputum was negative for tubercle bacilli.

He was found by skin testing and clinical observation to be sensitive to house dust, feathers, dog, cat, and horse dander, grass and fall pollens, and to Alternaria and Hormodendron spores. He has since been given perennial treatment with dust, pollen, and mold antigen, with improvement in his asthma, although occasional attacks have continued. In April 1943, he was seen after mild asthma of two days' duration. He had worked all day and although he did not appear ill, his face was somewhat flushed and his temperature was found to be 103°. No abnormal physical signs were discovered on chest examination. The next day his temperature was normal. Similar febrile episodes occurred in May, October, and December of that year, usually preceded by a day or two of increased asthma. On January 2, 1945, he reported that seven days previously he felt flushed and chilly when he reached home from work. That evening, his temperature was 103° and at 4:00 A.M. he had a drenching sweat, followed by normal temperature thereafter. After one day at home, he returned to work feeling quite normal. No unusual chest findings were present on January 2, and on January 4, apart from some diffuse emphysema, the lungs were roentgenologically negative.

June 25, 1945, he reported that there had been considerable coughing...
throughout the preceding day and some irritation remained in his throat. No abnormal chest findings were present. There was slight redness of the posterior pharyngeal walls, and his temperature was 99.4°. He was given 15,000 units of penicillin aerosol nebulized by oxygen at 3 liters per minute. One hour later, he reported a chill and temperature of 103.6°. When admitted to the hospital four hours later, his temperature was 103.2° and the leucocyte count was 14,800 with 88 per cent neutrophiles and 2 per cent eosinophiles. Chest roentgenogram at this time showed a nutoid size infiltration in the anterior portion of the right lung at the level of the fifth rib. No abnormal physical signs were detected over this area or elsewhere, and in spite of the fever he did not appear ill. The next day his temperature was 99.0° and the leucocyte count was 17,550 with 72 per cent neutrophiles and 1 per cent eosinophiles. Because this attack resembled previous ones in all respects and because he did not feel ill, he returned home. The following morning his temperature was 99° and was normal thereafter. Roentgenogram on July 2 showed the area of involvement to be almost clear. No blood count was made on this date, but on July 25 the leucocyte count was 7,300 with 5.5 per cent eosinophiles.

**Case II:** T.G., a boy who was seen first in 1937 at the age of four, had perennial rhinitis and recurrent attacks of asthma. Infantile eczema began at six months and persisted until he was about a year and a half old. Characteristic asthmatic breath sounds were present and the nasal mucosa was boggy and typically allergic in appearance. Skin tests showed sensitivity to house dust, alternaria, and the pollens of grass and ragweed. Dust precautions were instituted and perennial hyposensitization has been carried out with the antigens named with considerable general improvement. A bronchoscopic examination in 1940 showed only findings compatible with asthma. Occasional episodes of asthma usually have been associated with upper respiratory infections. Sputum examinations were repeatedly negative. Chest roentgenograms were normal apart from emphysematous appearing lung fields.

November 17, 1945, his mother reported much coughing and a temperature of 99.4° because of which sulfonamides had been given. Mild fever continued and on November 23, he entered the hospital. Chest roentgenogram on admission showed considerable infiltration about both hilar areas, extending on the left side for a considerable distance toward the periphery of the lungs. The temperature was 102°. There was normal resonance everywhere over the chest, with scattered asthmatic whistles present. Medium-sized inconstant sticky rales were heard in the left front chest at the level of the fifth interspace. Leucocyte count in the morning was 28,000 with 2 per cent eosinophiles. Sputum cultures on admission showed no unusual organisms. He was given 10,000 units of penicillin intramuscularly every three hours and in addition, he was given penicillin aerosol. His temperature continued to rise to 101 or 102° each afternoon. Roentgenogram on November 28 showed bilateral hilar infiltration which was now marked on the right side, and had somewhat subsided on the left. Intramuscular penicillin was discontinued November 30, when it was apparent that its use had resulted in no appreciable change. Throughout this time, his general condition had been good with no evidence of toxemia consistent with the pulmonary involvement present. On December 3, chest roentgenogram showed that the intrapulmonary infiltration had largely disappeared. The transitory and migra-
tory character of the roentgen shadows now made the diagnosis of Loeffler's syndrome quite certain. Mantoux test on December 3 was negative with 0.1 mg. tuberculin. Stool examination was negative for ova and parasites, and agglutination test for B. abortus was negative.

In early February, he had a frank upper respiratory infection and raised yellow, purulent sputum. Again March 30, he was seen with a reddened throat, a temperature of 101.6° and had scattered sibilant asthmatic rales in the chest. Fever continued for about a week and subsided after oral penicillin.

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**TABLE I**

Blood counts in Case II during two episodes of pulmonary infiltration.

<table>
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<tr>
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<td>13</td>
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<td>2.0</td>
<td>11</td>
<td>0</td>
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<td>25</td>
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<td>48.0</td>
<td>1.0</td>
<td>16</td>
<td>2</td>
<td>33</td>
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</tbody>
</table>

August 4, he had severe asthma which lasted three days during which he stayed in his room equipped with a pollen filter. On August 17, chest roentgenogram showed a new infiltration in the first and second interspace on the left side which remained without appreciable change in roentgenograms of August 23 and September 3.

Leucocytes on August 21 numbered 7,200 with 11 per cent eosinophiles, and 12,300 with 21.5 per cent eosinophiles on August 23. A sputum specimen on August 21 contained masses of purulent yellow material, with many eosinophiles and pus cells present, but no Curschmann's spirals or Charcot-Leyden crystals. No acid-fast bacilli were found and there was no growth of fungi on Sabouraud's medium. Beta hemolytic streptococci, S. viridans, and N. catarrhalls were present in blood agar sputum cultures. Albumen (grade 2) was found in the urine August 23 with 1-2 R.B.C. and 4-6 W.B.C. per high power field. No casts were present. (Previous urine specimens in June and July were negative). Urine culture on August 23 showed a fairly heavy growth of hemolytic staphy-
lococcus aureus and colonies of alpha hemolytic streptococci and S. viridans.
Throughout this period he did not feel especially ill, although his general appearance was not good. The presence of hemolytic cocci in the sputum and urine during the course of this last episode are felt to indicate that bacterial allergy was responsible in part, at least, for the pulmonary infiltration observed. Unequivocal proof of causal relationship is, however, lacking.

Case III: J.A.B., developed typical ragweed hay fever in 1928 at the age of seventeen. Symptoms recurred each fall thereafter with increasing severity. During the winter of 1935, while installing a heating system in a burlap bag factory, asthma occurred for the first time. Wheezing stopped at once after he left the building and there was no recurrence of asthma until 1940 at the peak of the ragweed season. Nasal symptoms which at first were confined to the fall season, gradually became perennial, although maximum rhinitis continued to occur in the spring and fall. In mid-May 1942, he observed dyspnea and some wheezing while climbing stairs and a diagnosis of virus pneumonia was made by his physician. Chest roentgenogram a little later showed infiltration of the left apex. A Mantoux tuberculin test at this time was positive. Repeated sputum examinations including cultures were negative for tubercle bacilli. He remained at home for seven weeks with bed rest during the first three. Roentgenogram June 3, 1942 showed the lung fields clear.

Throughout the summer of 1942, he had considerable rhinitis and during the ragweed season there were ten or twelve sharp attacks of asthma. In November 1942, a preemployment roentgenogram showed infiltration of the right middle lobe. Sputum and gastric specimens were negative for tubercle bacilli. November 30, 1942, he entered Muirdale

![Figure 1](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21161/)

**Figure 1**

*Figure 1, Case 3: May 25, 1942. Extensive infiltration, left upper lobe.—Figure 2, Case 3: June 3, 1942. Essentially clear. Questionable haziness in left sub-apical region.*

![Figure 2](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21161/)
Sanatorium where he remained until January 23, 1943. During this period, repeated sputum examinations showed many eosinophiles but were negative for tubercle bacilli; stool examinations were negative for ovae, amoebae, and parasites; muscle biopsy and skin test were negative for trichiniasis. A bronchoscopic examination showed nothing unusual and direct smear made from the secretions showed no fungi. During this period, there was a mild leucocytosis at times and eosinophiles fluctuated between 5 and 14 per cent. At no time did he appear ill and a tentative diagnosis of Loeffler’s syndrome was made on discharge.

He later gave the additional story of occasional hives in childhood, and reported two injections of antitetanic serum in the past without reaction. His mother had been subject to headaches, a maternal uncle had allergic rhinitis, and his five-year-old son has hay fever. Cutaneous reactions were present to tree, grass, and fall pollens, to alternaria spores, and to house dust. With the exception of tree pollens for which no passive transfer tests were made, circulating reagins were demonstrated for these antigens and for wheat and rye. He has had perennial hypo- sensitization with the indicated inhalant allergens for the past three years with definite clinical improvement. However, there have been occasional mild asthmatic attacks and he has had several episodes of pulmonary infiltration sometimes preceded by malaise, cough, and increased asthma, but at other times infiltrations have been discovered on a routine roentgen recheck of the chest in the absence of any accompanying symptoms.

During the height of the 1943 ragweed season, there was slight asthma which continued into October. On November 26, after spreading marsh hay over his garden, he had a sharp attack of asthma which lasted for several days. On December 9, he had generalized grippe-like aching and some pain under the left shoulder. Medium-sized moist rales were present at the end of inspiration in the left infraclavicular region. His tem-
perature was 100.4°, and the leucocyte count was 8,550 with neutrophils 77.5 per cent and eosinophiles 3.5 per cent. Roentgenogram at Muirdale Sanatorium December 11 showed dense infiltration in the left hilar region at the level of the second and third ribs anteriorly. Roentgenogram on December 18 showed marked clearing, and another December 24 showed still further improvement. During this episode, a single sputum specimen was reported positive for tubercle bacilli after culture and guinea pig inoculation. Since that time, repeated sputum cultures and guinea pig inoculations have all been negative for tubercle bacilli.

Roentgenogram April 18, 1944, showed a new and rather extensive infiltration in the right upper lung field with some residual fibrosis still visible in the left infraclavicular region. Leucocyte count was 8,950 with 22 per cent eosinophiles. Sputum culture was again negative for tubercle bacilli. Except for very slight wheezing which occurred on the night of April 25, there were no clinical symptoms during this period of pulmonary infiltration. No rales or physical signs were disclosed on examination other than those demonstrated in the roentgenogram, and he continued to work throughout this period without subjective feeling of illness. In the fall of 1944, considerable asthma, September 16 to 18, coincided with high atmospheric concentrations of alternaria spores, and subsequently, occasional mild wheezing continued throughout October. On November 20, he felt congestion in the chest and coughing increased. November 22, his temperature was 100.4°, but no abnormal chest findings were detected. Roentgenogram November 24 showed a wedge-shaped density in the left lower lung field with the apex directed laterally from the left heart border. Leucocyte count was 8,800 with 68 per cent neutrophiles and 10 per cent eosinophiles.

In the spring of 1945, occasional mild wheezing occurred during the
grass season. On May 16, excessive dust exposure while unpacking dusty materials caused coughing and increased wheezing which required ephedrine for relief. No subsequent symptoms were observed but on June 8, roentgenogram showed a new infiltration in the mid portion of the right lung, with considerable fibrosis still present in the upper left hilar region. Repeated sputum cultures on July 9 and August 3 were negative for tubercle bacilli and guinea pigs inoculated at this time were likewise negative when killed on September 14. Leucocyte counts on March 7, July 7, and August 22, 1945 were 12,500, 7,800, and 8,750 respectively with corresponding eosinophile count of 10, 13, and 12.5 per cent. Sedimentation rates at one hour on these dates were 15 mm., 16 mm., and 2 mm., respectively.

On September 15, 1945, chest examination was essentially negative. Roentgenogram at this time showed a rather extensive infiltration involving the upper lobes on the right side. Fibrosis in the left upper still remained, and there had been no change in appearance of the left hilus. A sputum specimen at this time was again negative for acid-fast bacilli by concentration test, and by culture. The leucocyte count on September 19 was 7,400 with 11.5 per cent eosinophiles. Roentgenogram on January 16, 1946, showed essential clearing of the lesion in the left upper chest with only a minor amount of scarring in this area represented by a few fibrotic strands extending into the first interspace. In the right lung, a fan-like infiltration extending from the hilus into the lower part of the right lobe still remained, but was less marked than in September. The leucocyte count January 22 was 9,000 with 16.5 per cent eosinophiles. On July 9, his general condition was good, the chest examination was normal, the leucocyte count was 6,050 with 0.5 per cent eosinophiles, and the sedimentation rate was 13 mm. at one hour. Sputum specimen at this time was negative for acid-fast organisms both by concentration test and cultures.

**FIGURE 7**

*Figure 7, Case 3: Nov. 24, 1944. Apices clear. Hilar densities increased. Wedge shaped shadow in the left lower lung field.*

**FIGURE 8**

*Figure 8, Case 3: June 8, 1945. Very marked clearing of right middle lobe. Increased hilar markings remain, especially on the left.*
COMMENT

Transitory pulmonary infiltrations conforming to Loeffler's criteria occurred in each of three patients reported. Multiple sensitivities to extrinsic inhalant allergens were demonstrated in each. In the first patient, repeated fulminating febrile episodes occurred without accompanying evidence of toxemia and with a recovery too prompt to be consistent with bacterial invasion. A transitory pulmonary infiltration was demonstrated by roentgenogram during one such episode. In both of the last two patients, bacterial sensitivities at times may have acted as the trigger mechanism for an allergic infiltration. The first recognized infiltration described in detail in Case II probably was initiated by such bacterial antigens, although no specific organisms could be incriminated. The later infiltration was accompanied by evidence of respiratory and urinary tract infection which may well have been responsible for the infiltrate. In the present state of our knowledge, control of such intrinsic or idiopathic factors is exceedingly difficult. In the third patient, it is apparent that extrinsic factors contribute materially to the clinical picture, and infiltrations often were related directly to massive exposure to such extrinsic allergens as dust, mold spores, or pollens. However, in view of the positive Mantoux test and of the single positive sputum reported, it is entirely possible that tuberculin may be an intrinsic antigen in this patient although clinical evidence of active tuberculosis continues to be lacking. The persistent and increasing fibrosis seen in the roentgenograms is adequate evidence that irreversible changes are developing in this patient which may well be of the type associated with hyperergic vascular damage such as described by Harkavy. Hyposensitization and avoidance of recognized extrinsic offenders has resulted in very definite general improvement and it is suggested that the irreversible process might well proceed with greater rapidity were it not for control of the recognized extrinsic sensitivities.

SUMMARY

1. Three cases of Loeffler's syndrome are discussed. In each instance, atopic rhinitis and asthma were present and sensitivities to multiple inhalant allergens were demonstrated.

2. Presumably, polyvalent bacterial or intrinsic sensitivities were present in all three patients in addition to recognized extrinsic sensitivities. It is difficult and often impossible in the presence of multiple sensitivities to incriminate any single antigen as the cause of an episode of allergic pulmonary infiltration.

3. In two of the patients described, pulmonary infiltrations were
characteristically transitory. In the third patient, although early infiltrations were of fleeting character, repeated attacks have left increasing residual fibrosis and evidence of irreversible damage. It is believed that irreversible changes after allergic pulmonary infiltrations are especially apt to occur when so-called intrinsic or bacterial sensitivities are present.

RESUMEN

1. Se discuten tres casos del síndrome de Loeffler. Todos ellos presentaban rinitis atópica y asma, y en cada uno se demostró sensibilidad a múltiples alérgenos de inhalación.

2. Se supone que en todos los tres pacientes existían sensibilidades bacterianas polivalentes o intrínsecas, además de las sensibilidades extrínsecas reconocidas. Cuando se presenta sensibilidad múltiple es difícil, y a menudo imposible, incriminar a un solo antígeno como causante de un episodio de infiltración pulmonar alérgica.

3. En dos de los pacientes descritos las infiltraciones pulmonares fueron caracteristicamente transitorias. En el tercer paciente, aunque los infiltrados precoces fueron de un carácter efímero, repetidos ataques dejaron una creciente fibrosis residual y signos de daño irreversible. Se cree que alteraciones irreversibles consecutivas a infiltrados pulmonares alérgicos tienden a ocurrir especialmente cuando existen las llamadas sensibilidades intrínsecas o bacterianas.

REFERENCES


