The Prevention of Cerebral Abscess Secondary to Pulmonary Suppuration

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Experience with two veterans hospitals at the Veterans Administration Hospital, Oteen, N. C., in recent years for pulmonary suppuration and secondary brain abscess prompted this report, the purpose of which is to suggest a possible way of preventing the serious cerebral complication. Both patients had been cured of their pulmonary suppurations by drugs and thoracotomy, and both were readmitted following discharge from the surgical service, one after 46 days, the other after 21 days, with paralysis of an arm and leg due to brain abscess. Both patients died after spontaneous rupture of brain abscess. In one patient, the abscess ruptured into a lateral ventricle; in the other, rupture was into the subarachnoid space. Neither patient had received vigorous drug therapy during the period of hospitalization for the pulmonary infection and neither patient had been advised to continue drug therapy following discharge after surgery. The purpose of this report is to recommend continuous administration of antibiotics or chemotherapeutic agents for the pulmonary infection from the day the diagnosis is made to the day of discharge and for about six weeks after discharge.

Brain abscess apparently never follows an acute pulmonary infection. Collis has stated that in his series of 44 cases, no instance of brain abscess was seen following an acute pulmonary infection. The pulmonary suppuration must be chronic, lasting at least three months, to give rise to metastatic brain abscess. Collis also found that brain abscess occurs more readily when thoracotomy is performed for the pulmonary suppuration. The chronicity of the suppurrative process and the surgical procedure create the pathologic conditions necessary for the metastatic brain abscess. All available information, experimental and clinical, indicates that an infected embolus from an intercostal thrombophlebitis in the chest wall is able to enter the spinal veins and thus enter the cerebral circulation. The same mechanism is responsible for metastatic brain tumor from cancer of the breast and lung. The spinal veins communicate above with the venous sinuses of the skull and below with the intercostal and lumbar veins. These veins are valveless, and, as a result, blood can readily flow in either direction, although gravity and differential pressure will usually direct this flow downward. Collis has shown how injections of radiopaque material into the intercostal veins readily reaches the spinal veins and the base of the skull.

Only chronic pulmonary suppuration can cause secondary brain abscess because it is necessary for vascularized adhesions to form between the lung and chest wall. If an infected embolus from a venous thrombus breaks loose, it will be carried to the azygos veins from where, in most cases, it will be transported to the superior vena cava and caught in the pulmonary bed without ill effect. If, however, the embolus becomes detached when conditions favor a reversed flow in the spinal veins, then it may enter this system and be carried to the brain. These conditions will be present during coughing, straining at stool, or when the patient is lying flat on his back.

Operative interference increases the danger of brain abscess because the infected sinus is in close proximity to the intercostal vein and its tributaries. If surgical drainage involves more than one rib, as is often the case in lung abscess, the danger of cerebral complication is increased. Collis has even suggested that when rib resection is performed for drainage, the intercostal veins...
should be occluded so that there will be no chance of a thrombotic process spreading to
them.

The interval between the onset of the lung infection and the appearance of neurologic signs due to brain abscess varies considerably. Cohen states that lung abscess occurred four months to four years prior to the onset of brain abscesses. In Collis' series, the average interval was seven months. In the two cases which are the subjects of this report, the intervals were two and one-half months and five months.

CASE 1

W.W.M., a 41-year-old white veteran was admitted to the Veterans Hospital, Oteen, N. C., on November 17, 1959, complaining of weakness, dyspnea, cough productive of yellowish sputum, loss of weight, night sweats, fever, and pain in the right lower chest on deep inspiration which began on November 13. He had been a fairly heavy drinker of whiskey for several years. In March, 1956, he was hospitalized for broncho-pneumonia involving the right lung and was discharged as cured after 11 days.

In May, 1959, he was admitted to City Hospital, Winston-Salem, N. C., for seven days for treatment of a respiratory infection. He returned to his job as hotel bell-hop in June, 1959, but had to quit after a few weeks because of weakness, anorexia, and weight loss. He went to live on his brother's farm, quit drinking, took it easy, but did not improve. On November 13, 1959, he had sharp pain in the lower right chest, night sweats, began to cough considerably, and expectorated yellowish sputum. His private physician recommended admission to Oteen Veterans Hospital.

Physical examination—He was poorly nourished, thin, and appeared to be chronically ill. The essential findings were flatness on percussion over the lower third of the right lung with distant breath sounds. The temperature was 103°F, the heart rate was 100, and the blood pressure was 100/70. All upper teeth were missing, and the eight lower teeth were in poor condition. He weighed 110 pounds, down from his average weight of 145 pounds.

Laboratory data—The urinalysis was normal. The tuberculin and fungus skin tests were negative. The white blood cells were 12,600 per mm³ with 76 per cent neutrophils, 20 per cent lymphocytes, 3 per cent monocytes, and 1 per cent basophils. The red blood cells were 2.8 million per mm³, hemoglobin 7.7 grams, and the hematocrit 28. The sedimentation rate was 66 mm³ in one hour. On November 18, roentgenograms showed an oval-shaped opacity in the right middle lobe, 6.5 x 11 cm., with evidence of fluid at the right base. A lung abscess with effusion was suspected. On November 21 thora-centesis yielded purulent fluid which, on smear, showed gram-positive cocci and gram-negative rods. The culture showed anaerobic streptococci.

Clinical course—On November 23, a thoracotomy was performed and the abscess space was drained. A drainage tube was inserted. Drainage was satisfactory and the wound was healed by January 27, 1960. During his hospitalization, he received the following antibiotics:

1. November 17 to 19: tetracycline 250 mg. q.i.d.
2. November 19 to 23: chloromycetin 1 gram intramuscularly daily.
3. November 20 to 25: chloromycetin 250 mg. q. 6 h.
4. January 14 to 19: tetracycline 250 mg. q. 6 h.
5. January 23 to 26: tetracycline 250 mg. q. 6 h.

He was discharged, cured, on January 28, with no instructions to continue antibiotics.

He was readmitted on February 18, 1960, complaining of severe persistent occipital headache, dimness of vision, and weakness of the left arm and leg. He said that headache began a few days after discharge on January 28 and had been persistent. On February 13, he felt numbness of the left hand. On February 14, he noted weakness of the left arm. On February 15, he had complete paralysis of the left arm and beginning weakness and stiffness of the lower extremity. Physical examination on February 18 revealed the left hemiplegia and bilateral papilledema which was more marked in the right eye. On February 19, he had a generalized convulsion in bed. A clinical diagnosis of metastatic brain abscess involving the right cerebral hemisphere was made and he was transferred to the Veterans Hospital in Durham on February 19 for further care. At 10:30 p.m. on February 19, a right carotid arteriogram revealed no shift or displacement of the vessels from the normal. On February 20, lumbar puncture revealed a clear spinal fluid under normal pressure. There were 4 lymphocytes, the chlorides were 105; and the total protein was 64. The fluid was negative on smear and culture. The patient continued to lose ground clinically. On February 26, he developed extensor rigidity on the left side. Lumbar puncture on February 26 showed an opening fluid pressure of 300 mm. of water. On February 26, the right carotid arteriogram showed displacement of the anterior cerebral artery to the left of the midline. The mid-portion of the middle cerebral artery was displaced medially, indicating a space-occupying lesion in the right tem-
peroparietal area. He was immediately taken to surgery. An exploratory craniotomy was performed in the right frontal region with passage of a ventricular needle into the brain. A burr hole was made over the right postero-parietal area with passage of a ventricular needle in several directions. No abscess was encountered. Following surgery, his condition stabilized. He became more alert, but the left hemiplegia was unchanged. On March 15, a spinal air study showed an initial fluid pressure of 90 mm. of water with clear colorless fluid. Roentgenograms of the skull showed no shift in the ventricular system. His alertness, cooperation and ability to talk and converse with the people on the ward continued to improve remarkably following surgery and he was transferred back to Oteen, on March 18 with a diagnosis of cerebritis due to septicemia following the lung abscess. On March 24, he had a generalized convulsion and became comatose. His temperature rose to 103°F. On March 25, he was still comatose and nuchal rigidity developed. Lumbar puncture showed an opening pressure of 220 mm. of water, cloudy fluid with 3600 white blood cells per mm$^2$ of which 99 per cent were polymorphonuclears. No organisms were seen on smear. He died in coma on March 26.

Necropsy—The significant findings were confined to the brain. The convolutions were flattened throughout. Frontal pus was present over the right fronto-parietal area, in the cisterna, about the meninges of the cerebellum, and the base of the brain including the pons. The ventricles contained pus. On section, near the anterior end of the left temporal lobe, was an abscess which communicated with the temporal horn of the ventricle. In addition, there were two unruptured, well-circumscribed abscesses in the pre-frontal area, the largest measuring 2.5 cm. in diameter, lying in an area which could give paralysis of the left arm. The other cavity was 1 cm. in diameter lying near the cortex in the anterior portion of the superior temporal gyrus. At the junction of the right parietal and occipital lobes was an area of degeneration 2 cm. in diameter which did not appear to be suppurrative. The right lung showed marked thickening of the pleura over the middle lobe with a 1 cm. cavity in the lobe. On microscopic examination the lung cavity showed infiltration with inflammatory cells and signs of healing. Culture of pus from the brain abscess showed anaerobic beta streptococi similar to the anaerobic streptococcus obtained on culture of the chest fluid before thoracotomy.

CASE 2

F.C.C., a 41-year-old white veteran was admitted to the Veterans Hospital, Oteen, N. C., on February 2, 1962, complaining of pain in the right chest, productive cough, weakness, and recent loss of 15 pounds which began on January 10. He had been working steadily in a furniture factory and had to quit a few weeks prior to admission because of weakness.

Past history—Pulmonary tuberculosis was discovered in 1945 when he was separated from the Army. Right pneumothorax was instituted in September, 1945 and was discontinued in February, 1950. His tuberculosis had been considered inactive in 1950 and he worked steadily until he was admitted to Oteen in February, 1952 because of reactivation. He was treated with streptomycin and PAS and was discharged as inactive in June 1954. He returned to his former work and got along well until September, 1961. A routine follow-up chest roentgenogram in September, 1961 showed no evidence of reactivation of the tuberculosis.

Present illness—In January, 1962, he had gradual onset of weakness, cough, and pain in the right side of the chest. Physical examination on admission on February 2, 1962 showed a well developed man who appeared to be chronically ill. He was 68 inches tall and weighed 115 pounds, down from his average weight of 130 pounds. The positive physical findings were confined to the chest. There was dullness on percussion over the right lower chest with lag on inspiration. The breath sounds over the lower right lung were distant; scattered expiratory rhonchi were heard over the upper lung field. His temperature was 101°F., the pulse 110, and blood pressure 110/70.

Laboratory data—Urinalysis showed a trace of albumin. The white blood cell count was 15,700 per mm$^3$ with 70 per cent neutrophils, 14 per cent lymphocytes, 5 per cent monocytes, and 11 per cent eosinophils. The red blood cell count was 4.7 million per mm$^3$, hemoglobin 11.3 grams, and the hematocrit 40. The sedimentation rate was 49 mm. There was slight anisocytosis and hypochromia. All sputum tests were negative for acid-fast bacilli on smear and culture. The chest roentgenogram showed a loculated effusion lying posteriorly in the lower right chest with a small pocket of air.

Clinical course—Because of the previous history of tuberculosis, he was given streptomycin, 1 gram daily, and ferrous sulphate for his anemia. He had a septic temperature with daily spikes to 101°F. The chest roentgenogram on February 19, 1962 showed a slight increase in the amount of fluid. On February 19, chest aspiration yielded 220 ml. of thick, foul-smelling, brownish purulent fluid which was negative on culture for aerobes, but grew Bacteroides on anaerobic culture. On February 19, penicillin was injected. On February 21, closed thoracotomy was done and a catheter drainage tube
was left in situ. On February 24, he received 500 ml. of whole blood. Penicillin and streptomycin were continued. There was drainage all during the month of March. Injection of contrast medium into the sinus tract showed persistent empyema space. On April 3, a modified Schede thoraecoplasty was performed with partial resection of the seventh to tenth ribs inclusive. Following this operation, the drainage stopped and the wound healed. He was discharged on June 1, 1962, having received maximum hospital benefits. He had received the following antibiotics during his hospitalization:

1. Streptomycin: 1 gram daily from February 3 to March 3.
2. Penicillin: 600,000 units daily from February 19 to March 12.
3. INH: 100 mg. t.i.d. from February 27 to June 1, 1962.
4. Pyrazinamide: 1.0 gram t.i.d. from February 27 to June 1, 1962.
5. Chloromycetin: 500 mg. q.i.d. from March 12 to 19.

He was readmitted 46 days later on July 18, 1962. On June 29, 1962, he noted gradual onset of weakness, night sweats, fever and headache. On July 11, he noted weakness of the left arm followed by weakness of the left lower extremity on July 14. On July 18, he began to vomit, became drowsy and mentally confused. The physical examination showed paralysis of the left arm and leg with no sensory change. There was left facial weakness, signs of meningeal irritation, and early papilledema. The patient was somnolent, confused, and was unable to give a coherent history.

**Laboratory data**—The white blood cell count was 19,000 per mm³ with 86 per cent neutrophils, 12 per cent lymphocytes, and 2 per cent monocytes. The hemoglobin was 13.1 gram and the hematocrit was 42. A lumbar puncture on July 18 showed a cloudy fluid with 300 white blood cells per cu. mm., 98 per cent of which were polymorphonuclears. The glucose was 54 mg., total protein 265, and the globulin was increased. On July 23, the spinal fluid showed 891 white blood cells per mm³ with 75 per cent polymorphonuclears, 21 per cent lymphocytes, and 4 per cent monocytes. The glucose was 24 mg. and the globulin was increased. Smears and cultures of both spinal fluids were negative for pyogens.

A clinical diagnosis of metastatic brain abscess secondary to the previous lung suppuration was made and the patient was transferred to the Veterans Hospital at Durham, N. C. for neurosurgery on July 27. The spinal fluid obtained at Oteen on July 18 was reported to show three acid-fast bacilli on smear. This was considered to be a laboratory error by the author. However, the laboratory report was incorporated in the clinical record. As a result, the physicians at Durham considered the patient to have tuberculous meningitis and he was given streptomycin, 1 gram daily, INH 200 mg. t.i.d., and PAS 4 grams t.i.d., from July 27 until August 17. On August 17 the streptomycin was discontinued; the INH and PAS were continued until his death. Because tuberculous meningitis was suspected, no carotid arteriograms were made. The patient got along well, seemed to improve clinically, and even grew a little stronger. However, the left hemiplegia was unchanged. On September 19, an electroencephalogram was described as "quite abnormal indicating a disturbance in the right fronto-temporal region." On September 22, the patient became confused, was incontinent of urine, and vomited several times. The right pupil was fixed in dilatation. There was bilateral papilledema. An emergency right carotid arteriogram showed massive displacement of the right anterior cerebral artery to the left side apparently due to a mass in the right frontal lobe. On September 22, he was operated upon immediately following the arteriogram. A right fronto-temporal craniotomy was performed. Subtotal resection of the right frontal lobe was done with excision of a huge fronto-parietal abscess. Smears and culture of pus from the abscess were negative for pyogens. The patient remained in coma following surgery and died on September 24. At necropsy, the brain showed a large area of tissue destruction in the right frontal lobe where the abscess had been excised. Hemorrhagic areas were present in the mid-brain and pons. The right occipital lobe was soft, mushy, and dark red due to a fresh infarct. There was purulent meningitis. There was no evidence of tuberculous meningitis. The lungs showed old healed apical fibrotic lesions. There was no evidence of active pulmonary suppuration or active tuberculosis.

**DISCUSSION**

In both patients during the initial admission for treatment of the pulmonary infection, drugs were given in interrupted courses. In Case 1, drugs were administered for only 24 days, although the patient was hospitalized for 72 days. The second patient was hospitalized for 188 days and received drugs for only 44 days. In view of the fact that thrombophlebitis develops during this period, it would be advisable to administer the most effective antibiotics or chemotherapeutic agents in adequate dosage during the entire period of hospitalization and for a period of several weeks thereafter.
Since cases of brain abscess have been reported in which there have been very long intervals, up to four years, between the onset of pulmonary suppuration and the appearance of cerebral symptoms, it is apparent that intercostal thrombophlebitis with virulent organisms may lie dormant for months and years. If an infected embolus breaks loose within the lumen of the vein at a time when conditions favor reversed flow into the spinal veins, metastatic brain abscess will develop.

Since the advent of chemotherapy and antibiotics, the incidence of brain abscess secondary to lung abscess has declined considerably. Therefore vigorous treatment with drugs during the stage of lung abscess should help prevent any cases of metastatic brain abscess.

Whether the route of the embolus is via the spinal veins or via the pulmonary bed into the carotid artery is academic. The available evidence indicates the former route. The reader is referred to Collis' excellent paper for fuller discussion.

**Summary**

Two cases of brain abscess secondary to lung abscess are reported. Both patients were discharged as cured of the pulmonary infection and were readmitted with fatal brain abscess. Chemotherapeutic and antibiotic therapy during the initial hospitalization was not continuous. It is suggested that drug therapy during the initial hospitalization should be vigorous. The most suitable drugs as determined by sensitivity tests should be given without interruption in adequate dosage. The drugs should be given for several weeks after discharge. Since surgical drainage (thoracotomy) greatly increases the risk of metastatic brain abscess, surgical intervention should be employed only if adequate medical treatment has failed. Since the reversed flow in the spinal veins is favored by coughing, straining at stool, and lying flat in bed, suitable measures for controlling cough and constipation should be taken along with early ambulation.

**Resumen**

Se relatan dos casos de absceso cerebral consecutivo a absceso pulmonar. Ambos enfermos se dieron de alta como curados de la infección pulmonar y fueron readmitidos por absceso cerebral que resultó mortal. La quimio y antibiótico terapia durante la internación inicial no fueron continuas. Se sugiere que la drogerapia inicial debe ser vigorosa. La droga más efectiva determinada por las pruebas de sensibilidad debe usarse sin interrupción a la dosis eficiente. Las drogas deben seguirse proporcionando por varias semanas después de la salida del hospital. Puesto que la canalización quirúrgica (toracotomía) aumenta mucho el riesgo de metástasis del absceso al cerebro la intervención debe emplearse sólo cuando el tratamiento médico bien llevado ha fracasado. Ya que el flujo retrógrado por las venas espinales es favorecido por la tos, el esfuerzo y la defecación, así como por el decúbito plano, deben tomarse medidas para controlar la tos, la constipación, combinando con ambulación temprana.

**Zusammenfassung**


**References**


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