A fatal complication of bronchiectasis

Brain abscess with intraventricular rupture

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ABSTRACT

The classic clinical manifestations of bronchiectasis are cough and daily production of purulent sputum for months to years. The most common complications are hemoptysis and respiratory failure. Brain abscess has become rare in the recent antibiotic era. In this report, we present a case of bronchiectasis complicated by brain abscesses. Despite the early diagnosis and appropriate management, and while the condition of the patient was improving, an intraventricular abscess rupture led to rapid coma then death. Presentation and management of this potentially fatal complication of bronchiectasis are discussed.

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Bronchiectasis is pathologically defined as an abnormal and permanent dilatation of one or several bronchi. The clinical picture is variable and affected individuals may be asymptomatic or suffer from severe respiratory failure. Daily sputum production is the most common, though unspecific symptom of bronchiectasis. Occasionally, major life threatening complications occur, such as severe hemoptysis, lung abscess or empyema or brain abscesses.[1] A fatal case with 34 discrete brain abscesses was previously reported.[2] We report another fatal case where death occurred after an intraventricular brain abscess rupture, while the patient seemed responsive to antibiotic treatment.

Case Report. A 37-year-old Saudi man presented to the hospital with new onset focal motor seizures of the right arm. He also noticed unusual headache and progressive weakness of the right side of his body over the 2 weeks prior to presentation. He was known to have bronchiectasis for 18 years on antibiotic prophylaxis when needed and had also old pulmonary tuberculosis that was treated successfully. He had a cough and yellowish sputum regularly with no changes over the last weeks prior to presentation. On examination, the patient looked thin, under weight, not pale or jaundiced. He had obvious clubbed fingers and toes. There were no lymphadenopathy and no neck stiffness, temperature was 36ºC; pulse 86/min and respiratory rate 20/min. His general examination was unremarkable except for bilateral coarse crackles up to the middle of the lungs with bronchial breathing over the left lower lobe. On neurological examination, he was fully conscious, oriented, with no speech disturbances. There was no papilledema. Power was slightly decreased (4- to 4+/5) in the right side of the body with astereognosia of the right hand. Reflexes were brisker on the right side and there was a right Babinski sign. Results of brain computed tomographies (CT) then magnetic resonance imaging

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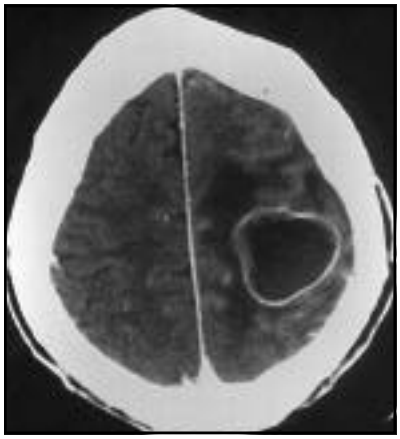

Figure 1 - T2W brain MRI showing an image suggestive of abscess in the left parietal lobe with severe peripheral edema.

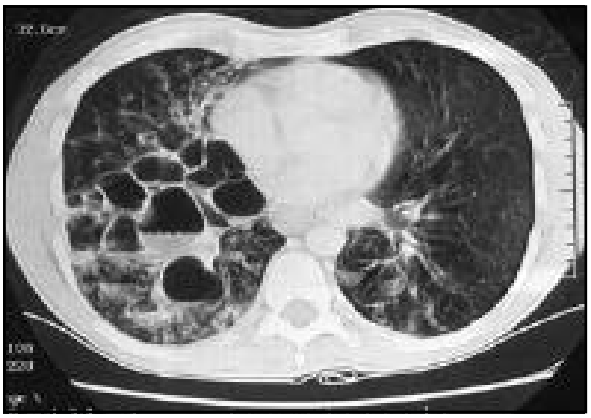

Figure 2 - Chest CT showing bronchiectasis of both lower lobes of the lungs, more severe on the right side.

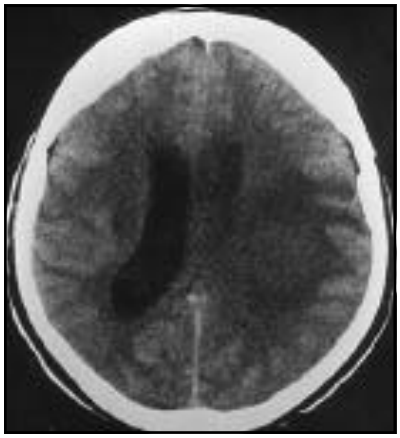

Figure 3 - Brain CT without contrast showing edema of the left hemisphere with isodense material in the left lateral ventricle that proved to be pus.

(MRI) (**Figure 1**) were highly suggestive of pyogenic brain abscess in the left parietal region. Usual blood investigations were normal apart from mild leukocytosis (white blood cell count 13.100/mm^3 with normal deferential), thrombocytosis (platelets 711.000/mm^3) and slightly elevated erythrocyte sedimentation rate (31mm/h). The total gamma globulin level was normal. Immunoglobulins A (IgA) and G (IgG) were slightly elevated: IgA 5.15 g/l (normal: 0.68 –3.78 g/L), IgG 19.60g/l (normal: 6.50 –16.20 g/L), IgM had a normal level. Blood cultures were negative. Chest x-ray then chest CT (**Figure 2**) showed bilateral cystic changes filled with fluids but no significant changes since previous chest x-ray 6 months before presentation. Sinus CT revealed mucosal thickening of frontal, maxillary and ethmoid sinuses but no fluid level. The patient was immediately started on intravenous (IV) antibiotics in the form of ceftriaxone 2g ever 12 hours, vancomycin 1gm ever 12 hours, and metronidazole 500 mg ever 8 hours. He received also carbamazepine and dexamethasone. The next day, a burr hole of the left parietal area was performed and 15cc of thick yellowish foul smelling pus was aspirated under local anesthesia. Culture proved positive for *Streptococcus Milleri*, which was sensitive to the antibiotics he was receiving and negative for acid-fast bacillus, anaerobes and fungus. The same antibiotic treatment was continued for 2 weeks with some improvement of the clinical status; headache was less and the plantar reflex became equivocal. Repeat brain CT results showed minimal reduction in edema but no change in the size of the lesion. On the 17th day of hospital stay the patient suddenly developed severe headache and nausea and started to have frequent seizures, then became unconscious within one hour. He was transferred to the intensive care unit (ICU) and put on assisted ventilation. An urgent third brain CT without contrast was obtained, and results showed rupture of the abscess into the left lateral ventricle (**Figure 3**). He was immediately taken to the operative theater, and a second left coronal burr hole was carried out with insertion of an external ventricular drain. Ten cc of thick foul smelling pus was removed. The patient stayed in ICU for 3 days under the same IV antibiotic therapy. He developed diabetes insipidus that was treated with Deamino-8-D-Arginine Vaspopressin (DDAVP). On the third day, his condition deteriorated to deep unresponsive coma. Brainstem reflexes became absent. A perfusion scan showed no cerebral perfusion, and the patient was declared brain-dead.

Discussion. Brain abscess may arise following hematogenous dissemination, by extension along emissary veins from infected cranial structures, or as a consequence of trauma or neurosurgical procedures. Half of hematogenous abscesses (10-15% of the total) are due to chronic pulmonary infections including bronchiectasis.[3] Most of these abscesses originating

from bloodborne infection lie within the territory of the middle cerebral artery and are often located near the junction between the cortex and the subcortical white matter and their capsule is usually poorly formed. Mortality from brain abscesses has decreased from around 40% in the 1950's to less than 10% in the era of modern imaging, advanced neurosurgical techniques and potent antibiotics. The use of diffusion-weighted magnetic resonance imaging has made the diagnosis relatively easy.[4] In our case, the diagnosis was made properly and timely, the patient was started on wide spectrum potent antibiotics, the abscess was drained, he began to improve and good prognosis was expected. Unfortunately, an intraventricular rupture occurred and he died within hours.

Despite all the new advances in diagnosis and treatment, intraventricular rupture of brain abscess (IVROBA) remains a catastrophic fatal complication. In a review of all cases published from 1950 to 1993, the mortality rate of IVROBA approached 80%.[5] In many cases, IVROBA was not detected until the patient deteriorates into a semicoma or coma state.[6] Brain abscesses that are related to hematogenous spread, such as in our case, are the ones the most prone to intraventricular rupture because of their usually deep location and poorly formed capsule. In a series of 33 cases of IVROBA published recently, those abscesses secondary to hematogenous spread were 31 (94%).[6] In this same series, the mortality rate was 40% instead of the 80% reported in the literature because of the aggressive management, including repeated drainage and intrathecal antibiotherapy. Our patient was re-operated upon immediately after the discovery of the rupture but continued to deteriorate quickly. He received IV but not intrathecal antibiotics.

In conclusion, this case illustrates that bronchiectasis can have rapidly fatal complications. Even in the era of advanced diagnosis and effective treatments of brain abscesses, potential intraventricular rupture still carries very high mortality rate. Becoming familiar with this complication and its early symptoms and signs, and rapid initiation of aggressive treatment are probably the most effective factors of better prognosis.

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