Platybasia Associated with an Unusual Case of Pulmonary Tuberculosis
Report of a Case with Necropsy*

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Chamberlain's paper in 1939 on "Basilar impression (platybasia)" has stimulated the interest of the medical profession in the study of malformation of the base of the skull and upper cervical spine, an anomaly up to that time receiving very little attention. In this paper he proposed a criterion for the diagnosis of platybasia based on the amount of projection of the odontoid process above a line drawn on a conventional lateral x-ray film of the skull connecting the posterior edge of the hard palate to the dorsal margin of the foramen magnum; the line now being referred to as "Chamberlain's line." Two cases in Chamberlain's series were treated by low occipital craniectomy and laminectomy of cervical segments with definite subjective and clinical improvement of their neurological symptoms.

According to Schueller, on the other hand, the diagnostic features of platybasia are the cephalad bulging of the floor of the posterior cranial fossa around the foramen magnum and the congenital variations of the upper cervical vertebrae.

The case described in this paper is a bizarre one, not only because of the presence of the developmental anomaly of the occipital bone and upper cervical portion of the spinal column, but also in the clinical course of the pulmonary tuberculous disease with its unusual concomitant complications.

Case Report with Necropsy Findings

J.C.B., No. 47,620, colored male, aged 25, was admitted to the Veterans Administration Hospital, Alexandria, Louisiana, on December 23, 1946, for treatment of pulmonary tuberculosis. In addition to the usual symptoms of pulmonary tuberculosis, the patient presented the following neurological findings: Excessive sweating limited to the entire left half of his body, fleeting joint pains, loss of the sense of differentiation between cold and heat, and dull and sharp, on the left side. The gait was staggering and unsteady but there was no history of trauma to the head or spine and at no time was there a loss of consciousness or convulsion.

The physical examination disclosed a well developed and well nourished colored male who appeared chronically ill. The temperature, pulse, and respirations were within normal limits and the blood pressure was 120/80.

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Examination of the chest revealed signs of a bilateral involvement with coarse post-tussive rales scattered throughout the middle portions and bases of both lungs. The above findings were confirmed by x-ray examination of the chest. Routine sputum examinations were consistently reported positive for acid fast organisms. Urine and Wassermann examinations were negative. Sedimentation index was 30 mm. in one hour. Complete blood count revealed: Hemoglobin 61.4 per cent, Red Blood Count 3,670,000, White Blood Count 8,200 Polymorphonuclears 60 per cent, Lymphocytes 38 per cent and Basophiles 2 per cent, Urea nitrogen 13, Cholesterol 120 and basal metabolism ranging from plus 1.9 to 19 per cent.

The Therapy Board recommended that patient be observed for a period of 30 to 60 days. During this period the author bronchosced the patient and aspirated a large amount of mucopurulent secretion from both main bronchi, finding no evidence of ulceration or stenosis of the bronchi. The patient's pulmonary condition, however, had not improved on routine management. Subsequent roentgenogram of the chest revealed a definite increase in the lesions in both lung fields and cavitation in the middle portions near the hilar region. Pneumoperitoneum was induced on March 25, 1947.

A transient improvement in patient's clinical and radiological condition was noted under this treatment, but the disease apparently was not controlled. In fact, the patient developed further cavities in the 2nd interspace on the left side and started to hemorrhage (Fig. 1). It was decided at this time to induce pneumothorax on the left side in addition to the pneumaobdomen. Patient's vital capacity (2,800 cc.), checked prior to inducing pneumothorax on the left side appeared very satisfactory. Electrocardiogram on September 22, 1947 showed sinus tachycardia but no definite cardiac pathology contraindicating combined pneumaobdomen with pneumothorax therapy. Initial treatment of 150 cc. of air,
under high negative pressure, was given on the left side on September 26, 1947 without any ill effect.

On October 8, 1947 without any obvious cause, the patient's temperature rose to 104.4°F, with a pulse rate up to 120 per minute and respirations of only 22 per minute. The patient was lying in bed quietly but complained of some fleeting pain in his shoulder joint and his left arm. There was no evidence of respiratory distress, nor any orthopnea.

Examination of the chest, including fluoroscopy, revealed collapse of both lungs with marked elevation of the domes of the diaphragm due to pneumoperitoneum. In other words, this patient had developed a spontaneous collapse of his right lung without noticeable dyspnea (Fig. 2). Continuous deflation was started immediately on the right side and patient was placed in an oxygen tent. The following day deflation was stopped because the air was not bubbling through the water trap. The possibility of continuing bilateral pneumothorax was then contemplated, provided that we were not dealing with a congenital mediastinal defect. It was noteworthy that while this patient's temperature was markedly elevated, up to 105°F, with a pulse of 136, his respiratory rate, nevertheless, was of usual frequency, only 18 to 24 per minute, a marked discrepancy between his respirations and his total clinical condition. The patient expired unexpectedly on October 12, 1947.

During the period of hospitalization he continued to have bizarre neurological complaints. The neurological examination disclosed the following positive findings: Pupils central and unequal, the right pupil being smaller; a rotary nystagmus bilaterally which became more pronounced on gazing toward the right temporal side; cremasteric reflex absent on the left side; knee and ankle jerks hyperactive on the right side; deep reflexes on the left side markedly diminished; definite sensory changes from the level of the left nipple down and marked sweating of the entire left half of the body.

FIGURE 3: Roentgenogram of the skull shows projection of the odontoid process above the line drawn from the posterior margin of the hard palate to the posterior lip of the foramen magnum.
These neurological symptoms were suggestive of syringomyelia. The x-ray of the skull (Fig. 3), however, showed a flattened and stenosed foramen magnum and projection of the odontoid process well above the "Chamberlain's line," consequently the diagnosis of platybasia was made.

The pertinent postmortem findings were as follows: There was narrowing of the foramen magnum in the antero-posterior diameter and there was elevation of its edge at the anterior rim. The exact anatomy of the atlas and second cervical vertebra was not explored to avoid mutilating the body. There was no protruberance of the cerebellar tonsils through the foramen magnum. The lung findings were in accordance with those demonstrated by x-ray. No demonstrable interpleural communication was present. Histologically nothing of significance was found except for chromatolytic changes in occasional anterior horn cells.

**Comment**

No other case of pulmonary tuberculosis associated with basilar invagination is to be found in the medical literature. The relationship is coincidental but the neurological changes appear to have masked many of the patient's pulmonary symptoms. The discrepancy between the slow respiratory rate and the febrile clinical course was striking during the entire period of hospitalization.

There is a possibility that we are dealing here with more than one congenital anomaly, both a mediastinal defect and platybasia. This is evidenced by the fact that this patient after the fourth injection of air into his left side developed a spontaneous pneumothorax on the right side without its usual symptoms. The question arose: was there a very slow leakage of air through the mediastinum? This question is impossible to answer positively, because the patient did not survive long enough to permit proof of the defect in the mediastinum. The fact that a mediastinal opening could not be found at autopsy does not rule out its presence because others, such as Smith and Willis, also could not demonstrate the mediastinal defect at autopsy in a proven case of interpleural communication during patient's life.

Our case had a bilateral pneumothorax with collapse of both lungs up to 60 per cent in addition to an extensive pneumoperitoneum, a combination rarely observed in tuberculous patients. There was no noticeable dyspnea at any time in this case which could be attributed to pressure on the respiratory center by some pathological process depressing the respirations. At necropsy there was no demonstrable impression on the medulla or the upper cervical cord due to pressure; nevertheless, histologically there was occasional anterior horn cell chromatolysis signifying a possible degenerative process.

Finally it would not be amiss to stress the fact that thorough search of the skull and cervical spine, especially roentgenolog-
ically, in all suspected cases of spinal cord degeneration showing symptoms of syringomyelia, multiple sclerosis, or other unusual patterns, might disclose many more cases of platybasia.

**SUMMARY**

1) Platybasia associated with an unusual case of pulmonary tuberculosis is reported and the possibility of a congenital mediastinal defect is also discussed.

2) The basilar invagination in this case apparently influenced the respiratory center by a depressing effect on the response of the respiratory rate to stimulation.

3) It is advisable that careful search for platybasia be made in every case with degenerative changes of the spinal cord.

4) The necropsy in this case confirmed our clinical diagnoses of platybasia and pulmonary tuberculosis with bilateral pneumothorax and pneumoperitoneum.

**RESUMEN**

1) Se informa sobre un caso de platibasia asociada con tuberculosis pulmonar inusitada y se discute también la posibilidad de que existiera un defecto congénito del mediastino.

2) En este caso la invaginación basilar aparentemente influenció el centro respiratorio y causó un efecto depresivo sobre la respuesta a la estimulación de la velocidad respiratoria.

3) Es prudente que se investigue cuidadosamente la existencia de platibasia en todo caso que presente alteraciones degenerativas en la médula espinal.

4) Es este caso la autopsia confirmó nuestros diagnósticos clínicos de platibasia y tuberculosis pulmonar con neumotórax bilateral y neumoperitoneo.

**REFERENCES**


2 Epstein, I.: "Mediastinal Defect with Intereplural Communication, Observed During Pneumothorax Therapy," Accepted for publication in *Diseases of the Chest*.
