CARCINOMATOSIS OF THE MENINGES OF THE SPINAL CORD AND BASE OF THE BRAIN, WITHOUT INVOLVEMENT OF THE PARENCHYMA, SECONDARY TO CARCINOMA OF THE LUNG

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Metastatic carcinoma of the nervous system limited to the meninges is rare, but it has been frequently described. Carcinomatosis of this sort is usually confined to the meninges over the cerebral hemispheres and only occasionally spreads to the spinal membranes. Meningeal carcinomatosis confined to the spinal cord and to a portion of the base of the brain without involvement of the cerebrum itself is so unusual that the following case is presented as an addition to our knowledge concerning metastatic carcinoma of the central nervous system.

REPORT OF CASE

CASE No. 36-23924: Weakness five months; lumbar pain one month; mass in left upper lobe demonstrable roentgenographically; severe pain in legs; wasting and progressive weakness suggestive of spinal cord lesion; flaccid paralysis; fibrillary tremors in trunk and limb muscles; diminished lower abdominal and leg reflexes; inconclusive Queckenstedt test; pleocytosis in spinal fluid. Necropsy findings: mass in left lung, probably primary carcinoma; meningeal carcinomatosis of spinal cord membranes and membranes at base of brain; no tumor in cord or brain.

History: J. R., a fifty-six-year-old white farmer, recently employed as a church sexton, was admitted to the medical service of Dr. Alfred Stengel on Sept. 12, 1936, complaining of lower back pain which had confined him to bed for a month. In April 1936, the patient's wife had noticed clubbing of his fingers, but he continued in his usual good health until mid-July, when weakness and fatigability caused him to seek medical aid. He suffered also from soreness of both knees and a recurrent sense of pressure over the precordium. In August 1936, he complained of a dull lumbar ache, which soon rendered him bedfast. This pain was intensified by motion of the spine, particularly by flexion, and the seated posture became unbearable. There had been a loss in weight of 15 pounds during the six weeks before hospitalization.

Examination: The patient was well oriented, cooperative, moderately emaciated, and did not appear acutely ill; his blood pressure, temperature, pulse and respiratory rates were normal. He exhibited the deliberateness of movement associated with severe pain. Examination disclosed moderate sclerosis of the peripheral vessels, distant heart sounds without murmurs, lungs normal to auscultation and percussion, a flat but resistant abdominal wall, a barely palpable liver, clubbing of the finger-tips, and obvious lumbosacral pain upon flexion of the spine. The abdominal and deep tendon reflexes were exaggerated; touch, pain and vibratory senses were preserved. Roentgen study disclosed large emphysematous sacs in each lung, and an ill-defined solid parenchymal lesion in the left upper lobe behind the hilum, about 1 1/2 inches in diameter. No osseous defects in the spine or pelvic bones could be demonstrated. The urinalyses, blood count, urea nitrogen, blood sugar, sedimentation rate and blood serology were normal. The patient was discharged on Oct. 5, 1936, without improvement to the Orthopedic Out-Patient Clinic. The diagnosis was recorded as “arthritis of the spine, pulmonary tuberculosis.”
**Course of Illness:** After a week at home without relief, the patient developed shooting "rheumatic" pains which radiated downward into the hips, legs, and feet. After eleven days at home, the pain became so excruciating that morphine was administered and he was readmitted to the hospital. He failed gradually but steadily over a period of ten weeks. The slow progression and the diffusely destructive nature of the spinal cord lesion, accompanied by increasing weakness and emaciation, led by a process of exclusion to a tentative diagnosis of metastatic malignancy, despite the absence of a clinically demonstrable primary tumor.

**Neurological examinations** by Dr. W. B. Cadwalader and Dr. A. M. Ornsteen disclosed the fact that the lower abdominal reflexes and the tendon reflexes of the legs were diminished and within a month these were entirely lost. On readmission both legs were weak and this weakness progressed to a complete flaccid paralysis. Fibrillary tremors were first noticed in the left foot, then in the left gluteus maximus, later in many of the muscles of the trunk and extremities, and finally in the face. Vibratory sensation was diminished below the knees and later was completely lost in the legs. An inconstant zone of hypesthesia was present about the pelvis, sparing the perineum. During the final fortnight of life, paresthesias of both hands and the left arm, progressive facial weakness, and difficulty in speech became apparent. Urinary retention necessitated an indwelling catheter one month before demise, and for the first time the patient became febrile, with clinical evidence of low-grade cystitis. During this hospitalization he required narcotics constantly, and steadily lost strength and weight until, on Dec. 27, he succumbed to respiratory failure with marked cyanosis.

The spinal pressure on admission was 90 mm. of water; the spinal fluid was quite xanthochromic, and contained 42 mononuclear cells which were considered to be lymphocytic. The total proteins were 350 mg. per cent; the Queckenstedt test was normal; the Wassermann reaction of the spinal fluid and the colloidal gold curve were negative. A fortnight later, the pressure and xanthochromia were unchanged; the proteins were further elevated to 470 mg., the pleocytosis somewhat less marked. The Queckenstedt test upon repeated observations was regarded as atypical. During jugular compression there was no rise in the spinal fluid pressure, but after release of compression the water manometric readings temporarily rose from 90 to 200-220 mm. Campiodol studies showed no spinal subarachnoid block.

**Autopsy:** At autopsy (No. 35–1432), twelve hours after death, emaciation and decubitus ulcers were noted. The viscera occupied their normal sites and there were no serosal exudates. The heart was not enlarged despite pericardial fibrous adhesions, and moderate sclerotic changes were present in the coronary arteries and in the valvular leaflets. The lungs were anthracotic, and presented an extraordinary degree of bullous emphysema, particularly in the apices. A small chronic tuberculous cavity was found in the right apex. The parenchymal lesion visible in the roentgenogram was situated behind the hilum in the left upper lobe, beneath a deep retraction of the visceral pleura on the mediastinal aspect. The mass was ovoid in shape, and consisted of intensely anthracotic, hard but not calcified fibrous tissue. The cut surface exhibited accumulations of white amorphous material, suggestive of caseation. The transition from the mass to the adjacent lung tissue was ill-defined, without gross evidence of encapsulation. Absence of any appreciable lymphadenopathy in the mediastinum was noted. The urinary bladder was hyperemic and edematous, and contained cloudy urine. The other viscera presented only such pathological changes as were compatible with the chronic wasting illness. The testes and mammas were not examined.

**Histological examination** confirmed the presence of a chronic tuberculous cavity in the right apex, the emphysematous changes, the extreme anthracosis, the purely fibrous character of the pericardial adhesions, and the subacute cystitis. The mass in the left upper lobe consisted of fibrous tissue, abundant interstitial amorphous debris and anthracotic pigment, much more suggestive of an old inflammatory lesion than of a neoplastic process. Viable cells were rarely discernible within the mass. About the periphery, however, a narrow zone of epithelial hyperplasia showed slight evidence of interstitial invasion and questionable lymphatic permeation. The small cuboidal cells were for the most part arranged in single layers, tightly packed, and lying in orderly fashion about empty lumina. The large nuclei had dense nuclear membranes and a moderate amount of chromatin material. The
FIG. 1. PHOTOMICROGRAPHS FROM PARENCHYMAL LESION IN THE LEFT UPPER LUNG

The dense band of fibrous tissue in the lower section is the thickened pleura overlying the lesion without being invaded. × 132
cytoplasm was pale and granular, and occasionally contained large, clear vacuoles. Cilia could not be demonstrated. These cells closely resembled the cuboidal cells found in the meninges. They also suggested the embryonal type of pulmonary epithelium not infrequently encountered in chronic inflammatory lesions of the lungs.

The brain showed nothing very striking except for a mild degree of cortical atrophy involving both frontal lobes, and a moderate thickening of the pia-arachnoid along the longitudinal fissure. The spinal cord, grossly, showed a severe degree of thickening of the pia-arachnoid extending over the entire length of the cord, involving the dorsal surface more than the ventral. The thickened meninges had a fibrinous, white appearance. Long strands of fibrinous tissue could be seen extending over the dorsal part of the cord. The meninges were firmly adherent to the cord.

Histologic examination revealed infiltration of the pia-arachnoid over the entire length of the spinal cord and the cauda equina. All levels of the cord were equally affected. In the meninges were sheets of cells, usually arranged in linear fashion, but in some places in poorly formed acini. These cells were confined entirely to the meninges. No infiltration could be seen in the cord except for a very slight penetration into the most peripheral portions of the dorsal column areas at the root entrance zone. This was present in only a few areas. Elsewhere the cord was completely free of infiltration both in the gray and white matter. The posterior and anterior roots were extensively invaded by the tumor cells, some of the roots almost completely so. The cells themselves were cuboidal or flat. The nuclei were round and contained dark, coarse chromatin granules. An external and internal membrane could be seen in all the cells, and in some definite cilia were visible.

A similar infiltration of the meninges was found over the base of the pons. This also was confined to the meninges, invading neither the pons nor its nerves. There was infiltration of the meninges over the medulla especially over the base but without invasion of the substance of the medulla. A similar infiltration was present over the midbrain, especially in the interpeduncular space.

**DISCUSSION**

The interest and importance of this case lie in the fact that the infiltration of the meninges was confined to the pia-arachnoid of the spinal cord and to the ventral surface of the pons. A careful search for similar cases reveals the rarity of this complication in carcinoma of the nervous system, regardless of the primary source of the carcinoma. When carcinoma from any of the
common sources metastasizes to the nervous system it usually spreads to the brain tissue in single or multiple nodules; it may be found rarely in the meninges alone or in the meninges and brain; or it may be confined, as in our case, to the spinal meninges. It almost never invades the spinal cord directly except in perivascular cuffings around the vessels of the spinal cord.

Only two cases have been found which simulated the one here reported in having the carcinomatosis confined to the meninges of the spinal cord and
base of the brain. There are many cases in which meningeal carcinomatosis was found with involvement of the brain also, but these were discarded since they did not conform strictly to the pattern of our case. Cases with infiltration of the cord were also discarded.

All three cases of metastatic carcinoma confined to the meninges of the cord have had their primary source in the lung. Rehn (1) reported a case of primary carcinoma of the right lung in a patient with crural paraplegia. The nervous system was invaded by tumor, the extension being confined entirely to the spinal cord meninges. The nerve sheaths of the cauda equina were especially affected. The posterior surface of the cord was much more involved than the anterior, and the posterior roots were invaded by carcinoma cells much more than the anterior roots. A similar case is recorded by Heyde and Curschmann (2). This was a carcinoma of the right lung with meningeal carcinomatosis of the spinal cord and brain involving more especially the base of the brain. Carcinoma cells were difficult to find in the meninges over the cerebral convexities. There was very slight penetration of the carcinoma cells into the brain.

How may the confinement of the metastatic lesion to the meninges of the spinal cord be explained? The probabilities are that a metastasis localized to the spinal meninges is not to be accounted for by blood stream dissemination. In our estimation the process probably occurred by direct extension along the intercostal nerves to the lymph spaces in the posterior roots and the posterior root ganglia, and from here probably spread directly into the meninges. The greater involvement of the posterior surface of the cord as compared with the anterior was probably the result of gravity, the cells gravitating to this surface of the cord with the patient in the dorsal decubitus position. A similar distribution of cells on the posterior surface of the spinal cord is seen in cases of extension of medulloblastoma from the cerebellum to the spinal subarachnoid space, and is explainable in the same way.

Conclusions

(1) A case is recorded of primary carcinoma of the lung with metastatic carcinomatosis limited to the meninges of the spinal cord and the base of the brain, particularly the pons.

(2) Only two similar cases were found in the literature.

(3) It is suggested that the carcinoma cells extended directly from the lung, along the lymphatic spaces in the intercostal nerves, to the lymphatic spaces of the posterior roots and posterior root ganglia to the spinal meninges.

References