Wilms Tumor: Diagnosis by Thoracentesis

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A diagnosis of Wilms tumor was made by thoracentesis in a critically ill child in whom immediate surgery was contraindicated. This confirmed the clinical impression and supported a definitive program of radiotherapy and chemotherapy preoperatively. The tumor was removed surgically after her clinical condition had improved and after reduction in the size of the mass had been obtained. Postoperatively, daunomycin was administered at two monthly intervals for two years and the child is alive and well without evidence of tumor four and a half years after diagnosis.

Pleural effusion may result from invasion of the pleura by neoplastic disease. The effusion is usually hemorhagic and examination of the fluid by the pathologist may reveal tumor cells. In the absence of a known primary, the cells may be recognized only as malignant, and further investigation is necessary to determine their origin. Pulmonary metastases of Wilms tumor usually involve the parenchyma of the lung; pleural metastases are less common but if invasion occurs, the material obtained by thoracentesis may reveal characteristics of the primary tumor. This report concerns a patient in whom Wilms tumor was confirmed by thoracentesis.

CASE REPORT

A four-year-old Caucasian girl was admitted to the Children's Hospital Medical Center on Sept. 30, 1967, with a history of abdominal pain of two weeks' duration. One week prior to admission, she experienced fever, sweating, lethargy and anorexia. Physical examination revealed a cachectic and acutely ill child with a distended abdomen and moderate ileus. She was pale and dehydrated. The right side of the abdomen was firm, with engorged vessels on the surface. The blood pressure was 110/80 mm Hg, pulse rate 140, respiratory rate 30 and temperature 100° F. Radiographic examination of the chest demonstrated multiple patchy densities in the right lung consistent with focal areas of infection or metastases.

Laboratory data (on admission) were the following: sodium, 124 mEq/liter; potassium, 6.9 mEq/liter; chloride 90 mEq/liter; carbon dioxide, 20 mEq/liter; blood urea nitrogen, 39.5 mg per cu mm; pH, 7.28; hematocrit 25 percent;

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FIGURE 1. Pleural effusion in right hemithorax. Left hemithorax shows no abnormality.

white blood count, 31,200 (64 percent polymorphonuclear leukocytes and 21 percent lymphocytes); urine, 3+ albumin and microscopic hematuria.

She was treated with blood transfusions and appropriate intravenously administered fluids. An intravenous pyelogram revealed a nonfunctioning kidney on the right and a normal calyceal pattern on the left. Although Wilms tumor of the right kidney was the more likely diagnosis, other possibilities included: hepatoma, intrarenal or suprarenal neuroblastoma, retroperitoneal lymphoma, and hydronephrosis, with pulmonary infection. An inferior vena cavagram demonstrated some obstruction of the vessel at, or above, the level of L-3 and slight anterior displacement by a large right-sided abdominal mass. The nonfunctioning kidney on the right side was also confirmed by delayed films.

Because of her poor clinical condition, and the extensive disease, radiotherapy to the right side of the abdomen was begun preoperatively. The plan was to administer less than 700 rad over the next several days to decrease the size of the abdominal mass, so that surgery could be safely and more effectively performed. During this period, her condition deteriorated, she became semiconscious and developed tachypnea and dyspnea. Radiographic examination of the chest revealed some opacification of the right hemithorax (Fig 1). The left lung was clear. An immediate diagnostic thoracentesis was performed, with removal of 150 ml of sanguineous fluid. The fluid was centrifuged, and a paraffin cell block was prepared from the sediment after fixation in Zenker's fixative. Microscopic examination of slides stained with toluidine blue and eosin revealed fragments of tumor and clumps of tumor cells formed by small cells with indis-
administered weekly for three months postoperatively, and dactinomycin in repeated courses at two monthly intervals over the ensuing two years.

At the time of discharge, five weeks after admission, the chest radiograph was normal, except for slight elevation of the right dome of the diaphragm. Eight months later, a "second look" operation demonstrated an organized thrombus in the inferior vena cava, with no evidence of tumor within the abdomen. At the time of writing, four and one-half years after diagnosis and treatment, she is alive and well.

**DISCUSSION**

Although there were reasonable grounds to suspect a Wilms tumor on admission (abdominal mass, radiologic evidence of pulmonary metastases and microscopic hematuria), the presentation was uncommon, since on pyelography the calyceal system in Wilms tumor is usually visible. Vascular occlusion or ureteral obstruction by tumor may result in nonvisualization, but chronic hydronephrosis with ureteral obstruction, pneumonia, electrolytic imbalance and ileus could not be excluded. The inferior vena cavagram was performed in an attempt to elucidate the diagnosis by determining the patency of this vessel and the renal vein. The latter was nonpatent and the inferior vena cava partially obstructed, possibly increasing the tendency for hematogenous spread by tumor. Immediate treatment was considered mandatory. If the diagnosis can be established, immediate operative removal is the treatment of choice. However, if laparotomy for diagnosis of disease, or circumstances for operative removal are unfavorable (critically ill patient and/or massive primary tumor), reduction in the size of the primary tumor mass may be attempted with a combination of radiotherapy and chemotherapy. Since the operative risk was excessive, it was elected to administer local radiotherapy to contain the mass. In our experience, the cytologic examination of pleural fluid of patients with pulmonary metastatic Wilms tumor has been most often noncontributory. The stromal cell of the usual Wilms tumor has a small spindle shaped or oval pyknotic nucleus and does not exhibit the usual characteristics of a malignant cell, such as an increase in nuclear size and variability in shape. Although free floating isolated tumor cells in pleural fluid of a patient with metastatic Wilms tumor may be recognized to constitute an abnormal cell population, a diagnosis of tumor can be reached only by exclusion. In the present case, actual tissue fragments, which may have been dislodged as a result of the radiotherapy, could be identified in the pleural fluid. The spindle and oval shape of the stromal cell nuclei favored Wilms tumor over neuroblastoma, the cells of which have predominantly round nuclei. Although the rosettes seen in this tumor bear a superficial resemblance to those that are seen in neuroblastoma, there were certain differences. The cell nuclei of the rosettes in neuroblastoma are again usually round and the center is homogenous pink. In this tumor the cell nuclei of the rosettes were oval and the center was finely granular. The presence of a small lumen in some of these structures established them as imperfect tubules and tended to rule out a diagnosis of
neuroblastoma, since in this tumor the rosettes never have a lumen.

With a positive diagnosis of Wilms tumor, the treatment could be pursued with more confidence. Immediately after thoracentesis, chemotherapy was instituted, and the radiotherapy portal was extended. Both lungs now received irradiation, since hematogenous dissemination had been proved. There is ample documentation that dactinomycin is carcinolytic\(^\text{1-3}\) and has an additive effect to radiotherapy,\(^\text{5,6}\) thus allowing doses of irradiation to be delivered without causing radiation pneumonitis and lung fibrosis. It has also been shown that Wilms tumor including lung metastases may be affected by vincristine sulphate therapy,\(^\text{a}\) and Sullivan et al\(^\text{10}\) have indicated that it may be particularly useful in shrinking the tumor preoperatively.

**REFERENCES**


Coronary Aneurysms in a Young Woman: Angiographic Documentation of the Natural Course*

Hans van den Broek, M.D., and Bernard L. Segal, M.D., F.C.C.P.

A 19-year-old woman is reported with saccular aneurysms of the left anterior descending coronary artery and of diagonal branches. A scratchy systolic-diastolic murmur, heard on several occasions, gave the clinical impression that one of the aneurysms was in the process of rupturing. The patient's condition, however, remained stable until she developed an acute myocardial infarction. Postinfarction coronary arteriography showed complete occlusion of the anterior descending artery, most likely caused by thrombosis in one of the aneurysms which extended into the coronary artery.

In a young woman aneurysms of the left anterior descending coronary artery and spontaneous intraluminal thrombosis resulted in acute myocardial infarction. The abnormalities were documented angiographically.

**CASE REPORT**

A 19-year-old Negro woman was admitted to Hahnemann Medical College and Hospital because of chest pain.

The patient was well until December, 1970, when she developed substernal chest pain with radiation into the neck and left arm; the pain was constant, aggravated by deep breathing, and lasted for six days. She was then asymptomatic until the middle of April, 1971, when the pain recurred, this time aggravated by the recumbent position as well as deep breathing and relieved by sitting up. The patient was hospitalized elsewhere for four weeks after being told she had an abnormal electrocardiogram; a diagnosis of acute myocardial infarction was made. Following discharge she was asymptomatic.

On June 6, 1971, she was admitted to Hahnemann Hospital for further evaluation. There was no history of diabetes mellitus or hypertension; she smoked less than one half pack of cigarettes a day and denied taking oral contraceptives. The family history was not remarkable.

Physical examination showed a well-developed, well-nourished Negro woman in no acute distress. She was afebrile and remained so throughout her stay in the hospital.

**FIGURE 1A.** Electrocardiogram during first admission.

**FIGURE 1B.** Electrocardiogram during third admission.

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