TUMOR TISSUE REGISTRY

LOS ANGELES COUNTY HOSPITAL

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PROTOCOL

FOR

MONTHLY SLIDES

JULY 1963

LYMPHOMAS

NAME: P. S. JULY 1963 - CASE NO. 1

AGE: 80 SEX: Male RACE: Caucasian ACCESSION NO. 11075

CONTRIBUTOR: S. M. Rabson, M. D. Outside No. 89254
Mission Hospital
Huntington Park, California

TISSUE FROM: Axillary mass

CLINICAL ABSTRACT:

History: On July 28, 1960, this patient first noted a lump in the left axilla.

Physical examination disclosed this mass to measure 12 x 14 cm.

Laboratory report: Hemoglobin: 12.1 gms%. WBC 7,900 (45% segmented, 50% lymphs).

SURGERY:

The tumor was resected on August 8, 1960.

GROSS PATHOLOGY:

The specimen consisted of an 8 x 10 x 6 cm. multilobated mass encased in fat. Cut surface disclosed the mass to be formed by the confluence of many smaller masses, varying from tan to gray in color and firm to fleshy in consistency.

COURSE:

After resection of the mass, the patient was treated with irradiation to the axilla. He expired two years later of a "heart attack." There was then said to be no evidence of lymphomatous disease.

NAME: T. R. JULY 1963 - CASE NO. 2

AGE: 2 SEX: Male RACE: Caucasian ACCESSION NO. 11077

CONTRIBUTOR: David B. Dickey, M. D. Outside No. R-1251-60
Torrance, California

TISSUE FROM: Axillary lymph nodes

CLINICAL ABSTRACT:

History: This child was noted to have a rapidly enlarging mass in the left axilla. No other adenopathy was present, and the spleen and liver were not palpable.

There were no remarkable peripheral blood findings, and a chest film was reported as negative.

SURGERY:

The axillary mass was removed on August 29, 1960.

GROSS PATHOLOGY:

The specimen consisted of a cluster of enlarged and matted lymph nodes surrounded in large part by lobulated fatty tissue. The overall measurements were 8 x 5 x 3 cm. and the largest lymph node was 4 cm. in greatest dimension. The cut surface varied from orange-brown to gray in color and displayed punctate red-brown areas.

FOLLOW-UP:

The patient expired 8 months after surgery with "involvement of epicardium, lungs, spleen, liver, kidney, and lymph nodes."

NAME: M. K. JULY 1963 - CASE NO. 3

AGE: 52 SEX: Female RACE: Caucasian ACCESSION NO. 9940

CONTRIBUTOR: Frank R. Dutra, M. D. Outside No. S-7502
Eden Hospital Laboratories
Castro Valley, California

TISSUE FROM: Lymph node, right inguinal region

CLINICAL ABSTRACT:

History: This patient was hospitalized in August 1956 because of bilateral axillary lymphadenopathy. At that time excision biopsy was performed on one of the nodes in the right axilla. Pathological diagnosis at that time was "marked hyperplasia, lymphocytic, and lymph node," but it was also noted that some of the changes were suggestive of a small cell lymphosarcoma. Her post-operative course was uneventful.

She was re-admitted to the hospital on May 5, 1957 with a tender lump in the right groin of 4 to 6 weeks' duration. This lump was more prominent when standing. The pain was dull and aching in nature with radiation down the inner aspect of the right thigh to the knee level. There was a weight loss of approximately five pounds in the previous month, but no history of chills, fever, night sweats or weakness.

SURGERY:

On May 13, 1957, a biopsy of a lymph node of the right inguinal region was performed.

GROSS PATHOLOGY:

The specimen consisted of a large ovoid mass of tissue containing an enlarged lymph node, and small amounts of adipose tissue adherent to a delicate gray translucent capsule. The mass was ovoid in shape and measured 5.5 x 3.2 x 2.0 cm. Sections revealed the cut surfaces to consist of homogeneous, pale pinkish-gray tissue with a center core of delicate blood vessels and collagenous tissue. This core was seen to communicate directly with the hilar portion of the node.

COURSE:

In October 1957, the patient noted increase in size of the nodes in the nuchal, right inguinal, and femoral areas. She was re-hospitalized for nitrogen mustard therapy.

From August 1956 to November 1957, the patient's blood counts were normal. A follow-up report stated that the patient expired with disseminated lymphosarcoma in June or July of 1961.

NAME: G. D.

JULY 1963 - CASE NO. 4

AGE: 64 SEX: Male RACE: Caucasian

ACCESSION NO. 12984

CONTRIBUTOR: S. K. Abul-Haj, M. D.
Walter Reed Hospital
Washington, D. C.

Outside No. S-369-60

TISSUE FROM: Inguinal mass

CLINICAL ABSTRACT:

History: In 1960, this patient sought medical attention because of a lump in the right inguinal region, which had enlarged progressively over a six month period. He was otherwise asymptomatic.

The physical examination was essentially negative except for a large, non-tender, right inguinal mass which measured 7.5 x 6 x 5 cm.

Laboratory reports: Routine hemogram and urinalysis were within normal limits.

SURGERY:

The mass was resected.

GROSS PATHOLOGY:

The specimen consisted of an ovoid mass which measured 8.0 x 7 x 6 cm. The lesion was firm and knobby with an ellipse of attached skin. The cut surface was gray-white in color and coarsely nodular in consistency.

COURSE:

The patient received a single dose of x-ray therapy. After transfer to Walter Reed Hospital, the radiation therapy was discontinued. A comprehensive work-up, including serum electrophoresis, bone marrow examination and lymphangiography were all interpreted as within normal limits. Two years later the patient was free of symptoms and without evidence of disease.

NAME: H. R. JULY 1963 - CASE NO. 5

AGE: 11 mos. SEX: Male RACE: Caucasian ACCESSION NO. 12985

CONTRIBUTOR: S. K. Abul-Haj, M. D.
Walter Reed Hospital
Washington, D. C. Outside No. S 266-59

TISSUE FROM: Cervical lymph nodes

CLINICAL ABSTRACT:

This infant was hospitalized at the age of four months because of a skin rash and upper respiratory infection. The skin eruption began on the feet but had become generalized within two weeks. The patient responded somewhat to antibiotic therapy. However, the rash re-exacerbated and the upper respiratory tract infection recurred during the ensuing six months.

The patient was re-hospitalized at the age of 11 months because of a sore throat and cervical mass. Physical examination at this time revealed an infant of average development. The skin was covered with a spotty, maculopapular, occasionally hemorrhagic, impetigenous type eruption. The ears were thought to show evidence of otitis media and the throat was fiery red. A 2 x 2 cm. mass was present in the left cervical area. Auscultation of the chest revealed noisy rales. The liver and spleen were enlarged. Axillary and inguinal lymph nodes were moderately enlarged, rubbery, firm, and discrete.

Laboratory report: Hemoglobin 7.5 gm%. WBC 22,500 (88% PMNs).

X-ray reports: Chest films revealed exaggerated bronchovascular markings and diffuse mottling of both lung fields.

SURGERY:

In November 1959, an attempt was made to drain the fluctuant neck mass, yielding a small amount of gray-white debris. Culture and smears of this material were unrevealing. The cervical mass was biopsied and resected.

FOLLOW-UP:

Subsequently, serum protein electrophoresis was done showing the gamma globulin to be .05 per cent. In spite of vigorous therapy with antibiotics and meticortin, the patient's condition steadily deteriorated and he expired at the age of 13 months. Pertinent autopsy findings were limited to lung, spleen, bone marrow, and lymph nodes. The spleen weighed 90 gms. It was slightly enlarged and had focal 2 to 5 mm. size orange-yellow nodules scattered in its pulp. Similar nodules were seen in the bone marrow. The lymph nodes were enlarged, discrete, and orange-yellow. The lungs showed diffuse gray hepatization. The immediate cause of death was giant cell pneumonia of Hecht.

NAME: D. O.

JULY 1963 - CASE NO. 6

AGE: 48 SEX: Female RACE: Negro

ACCESSION NO. 12986

CONTRIBUTOR: S. K. Abul-Haj, M. D.
Walter Reed Hospital
Washington, D. C.

Outside No. S 59-1418

TISSUE FROM: Cervical lymph node

CLINICAL ABSTRACT:

This patient was hospitalized because of fever and malaise, associated with lymphadenopathy of four weeks' duration. Pertinent physical findings included generalized lymphadenopathy, hepatomegaly and splenomegaly. The lymph nodes were firm, discrete and "egg-sized."

Laboratory report: Hemograms: Hemoglobin 11.8; WBC 11,500 (57 PMN, 41 lymphs, 2 monos.). Urinalysis: No significant abnormal findings.

SURGERY:

In May 1959, a cervical lymph node was removed.

GROSS PATHOLOGY:

The node measured 6.0 x 5 cm. and was ovoid in shape. The capsule was smooth and shiny. Cut section revealed marked exaggeration of the normal markings. The surface was pale yellowish tan.

FOLLOW-UP:

Because of the peculiar and unusual lesion in the lymph node, it was advised that serum protein and lipid studies be performed. The serum protein was 9.7 gms.; globulin 7.2 gm.; and albumin 2.5 gm. The patient was lost to follow-up and no further information was received.

NAME: M. L. M. JULY 1963 - CASE NO. 7

AGE: 28 SEX: Male RACE: Caucasian ACCESSION NO. 10958

CONTRIBUTOR: D. Tatter, M. D. Outside No. 65177
Los Angeles County Hospital
Los Angeles, California

TISSUE FROM: Lymph nodes

CLINICAL ABSTRACT:

History: This patient was hospitalized on May 29, 1960 with a history of progressive weakness for approximately 10 months, associated with pedal edema and a weight loss of 35 lbs. A hematologic workup by his physician had revealed pancytopenia with a WBC of 1,000 associated with hepatomegaly and splenomegaly. A splenectomy was performed at an outside hospital. Post-operatively, the patient's course was one of progressive weakness, weight loss, fever, chills, jaundice, and bleeding tendency.

Pertinent physical findings included palpable, shotty nodes in the neck and axilla. The abdomen was distended by a huge liver.

Laboratory report: Hemoglobin 11.5 gm. WBC 9,000 with a grossly normal differenetial. (segs. 60%, bands 11%, lymphs 10%, monos 17%, myelocyte 1, metamyelocyte 1). Bone marrow aspirate: Increased numbers of reticulum cells.

COURSE:

The patient was treated with cytoxin, 50 mg. b.i.d. However, his course continued downhill and he developed left lower abdominal pain associated with a distended silent abdomen. He expired on May 29, 1960.

At autopsy, approximately 3000 cc. of turbid yellow, foul-smelling material was removed from the peritoneal cavity. The peritoneal surfaces were thickened, opaque, shaggy, and yellow in color. The ileum contained an oval, depressed ulceration, which measured 3 x 2 cm. in diameter. The base was granular and hemorrhagic with a 3 mm. perforation opening freely into the peritoneal cavity. Also noted in the wall of adjacent bowel were 5 to 10 ill-defined submucosal patches without induration or perforation. The liver edge extended to 10 cm. below the right costal margin. External and cut surfaces displayed numerous umbilicated nodules, measuring .8 to 1.5 cm. in diameter, dispersed throughout the parenchyma. Enlarged lymph nodes were present in the porta hepatis, along the lesser curvature of the stomach and para-aortic regions. These were white in color and soft in consistency. The bone marrow was extensively replaced with a whitish-yellow, soft, infiltrating tissue.

NAME: B. K.

JULY 1963 - CASE NO. 8

AGE: 35 SEX: Female RACE: Caucasian

ACCESSION NO. 12467

CONTRIBUTOR: James W. Decker, M. D.
Washoe Medical Center
Reno, Nevada

Outside No. S 2832-62

TISSUE FROM: Thorax

CLINICAL ABSTRACT:

History: In January 1962, during the third trimester of pregnancy, a routine chest film disclosed a mass in the right lower lung field of this 35 year old housewife. Her general health had been good with no significant previous illnesses.

Physical examination disclosed a single small nodule in the left lateral thyroid lobe.

Laboratory reports: Hemoglobin 13.6 gm%; hematocrit 41%; WBC 7,550 (62 polys, 37 lymphs, 1 eosinophil). Urinalysis negative.

SURGERY:

A thoracotomy was performed in August 1962.

GROSS PATHOLOGY:

The specimen consisted of a tissue mass, measuring 5 x 6 x 2 cm. A segment of vertebral body, measuring 6 x 2 x .3 cm., was present on the medial border, and a portion of rib, 3 cm. in length and 1 cm. in diameter, was present superiorly. The major portion of the mass consisted of an egg-shaped tumor, 3.5 x 3 x 2 cm. This was covered anteriorly by a segment of glistening intact pleura. On cut section, the mass displayed a homogeneous, moist, finely granular, yellow appearance with a central area resembling white fibrous tissue. The mass appeared encapsulated and superiorly was separated from the rib by approximately 8 mm. of fibroadipose tissue. Medially, the mass was closely adherent to the periosteum of the vertebral body. No definite cord-like structures were seen entering the mass, although at the superior medial border, several filamentous gray structures were present beneath the pleural surface.

FOLLOW-UP:

The patient was last seen by her physician on June 11, 1963. At that time she was doing well, had convalesced satisfactorily and was asymptomatic.

NAME: E. K. JULY 1963 - CASE NO. 9

AGE: 74 SEX: Male RACE: Caucasian ACCESSION NO. 12987

CONTRIBUTOR: J. R. McGrath, M. D. Outside No. C-3240-62
Centinela Valley Community Hospital
Inglewood, California

TISSUE FROM: Lymph nodes

CLINICAL ABSTRACT:

History: The patient developed progressive weakness requiring hospitalization. Physical examination revealed enlarged nodes in the groin and abdomen. After lymph node biopsy, the patient received 3 units of blood and x-ray therapy with improvement noted upon discharge from hospital.

SURGERY:

Lymph node biopsy was done during hospitalization in October and November 1962.

GROSS PATHOLOGY:

The specimen consisted of a circumscribed firm tumor mass, measuring 3.0 x 2.0 x 1.3 cm., which on section showed glistening granular pinkish-tan tissue.

FOLLOW-UP:

Following discharge from the hospital, the patient did fairly well for several months and then developed severe weakness and lethargy which progressed until his death.

NAME: A. B. JULY 1963 - CASE NO. 10

AGE: 66 SEX: Male RACE: Caucasian ACCESSION NO. 10692

CONTRIBUTOR: Ben Fishkin, M. D. Outside No. B255-59
Veterans Administration Hospital
Los Angeles, California

TISSUE FROM: Lymph node, right iliac area

CLINICAL ABSTRACT:

History: A 66 year old white male was admitted to the Veterans Administration Hospital because of progressive increase in claudication of the right leg. The patient had no constitutional symptoms. Past history revealed that the patient was a mild diabetic.

Physical examination revealed the liver to be palpable 6 cm. below the costal margin. No external lymph nodes were palpable.

Laboratory findings were as follows: Hemoglobin 16.4; hematocrit 49; WBC 6,250 (bands 2, neutrophils 58, lymphocytes 33, monocytes 3, eosinophils 3, basophils 1, platelets adequate).

SURGERY:

On January 20, 1959, bilateral aortic iliac endarterectomy was performed. During the course of surgery, three discrete enlarged lymph nodes were observed in the right iliac area, and a node was removed for study.

GROSS PATHOLOGY:

The specimen consisted of a lymph node that measured 2.5 x 1.5 x 1.3 cm. It was moderately soft in consistency and purple-tan in color. On cut section, the surface was partially homogeneous and gray-white.

COURSE:

The patient was given nitrogen mustard in 1959. This therapy was repeated in 1961, because of right paratracheal and left cervical lymphadenopathy and some constitutional symptoms such as fatigue, weight loss, and generalized weakness. On this admission, the white count was 44,000 which subsided to 15,000 on discharge. The patient was lost to follow-up after October 1961.

NAME: G. M. JULY 1963 - CASE NO. 11

AGE: 17 SEX: Female RACE: Caucasian ACCESSION NO. 11221

CONTRIBUTOR: Herbert I. Harder, M. D. Outside No. 60-3943
Glendale Sanitarium and Hospital
Glendale, California

TISSUE FROM: Lymph nodes, right side of neck

CLINICAL ABSTRACT:

History: This patient entered the hospital with a chief complaint of nodular swelling in the right neck, which had been present for about 9 months. The mass was slow growing, painless, and unaccompanied by any other symptoms.

Laboratory report: Hemoglobin 12.6 gms; WBC 11,850; lymphocytes 22; monocytes 5; basophil 1; eosinophils 2; neutrophils 70; stabs 1. The morphology of both white and red cells is normal. PCV 39%. Bone marrow was not done.

SURGERY:

At surgery, a group of nodes, measuring 4.5 x 2.5 x 2.5 cm., were removed from the right side of the neck.

GROSS PATHOLOGY:

The specimen consisted of a 4.5 x 2.5 x 2.5 cm. mass of matted lymph nodes, which were rubbery - firm, and had bulging, slightly translucent, pink-tan-gray, homogeneous cut surfaces.

FOLLOW-UP:

After leaving the hospital, the patient was treated with x-ray radiation. She improved, and when last seen by the physician in January of 1962, was in good health. The physician has not seen her since, but he has seen others of her family who have reported nothing about her.

NAME: H. E.

JULY 1963 - CASE NO. 12

AGE: 7 SEX: Male RACE: Caucasian

ACCESSION NO. 12860

CONTRIBUTOR: W. K. Bullock, M. D.
Orthopedic Hospital
Los Angeles, California

Outside No. OH-63-300

TISSUE FROM: Mass in left shoulder

CLINICAL ABSTRACT:

History: This patient had been under the care of a physician in Mexicalli for a tumor mass in the left shoulder that had gradually increased in size over a period of 18 months. The patient also complained of abdominal pain and nausea and was a poor eater. There were no previous illnesses, operations, or therapy. Family history, birth history and development were unremarkable.

SURGERY:

The patient was taken to surgery and a tumor mass removed from the left shoulder region.

GROSS PATHOLOGY:

The specimen consisted of a 115 gram tumor mass, consisting primarily of yellowish, lobulated cartilaginous material, which was very firm. On cut section, the specimen was gritty.

STUDY GROUP CASES
FOR
JULY 1963

LYMPHOMAS

CASE NO. 1, ACCESSION NO. 11075, S. M. Rabson, M. D., Contributor

SAN FRANCISCO:

Reticulum cell sarcoma, 5; lymphoblastic lymphosarcoma, 3; metastatic carcinoma, 2.

OAKLAND:

Undifferentiated metastatic carcinoma (specifically bronchogenic, 1; seminoma, 1), 11.

CENTRAL VALLEY:

Lymphocytic or lymphoblastic lymphosarcoma, 6; histiocytic or reticulum cell sarcoma, 5; reactive hyperplasia, 1.

SAN DIEGO:

Lymphocytic lymphosarcoma, 7; reactive hyperplasia in lymph node, 3; solitary lymphocytoma, 1.

WEST LOS ANGELES:

Malignant lymphoma, reticulum cell type, 9; malignant lymphoma, lymphosarcoma type, 5.

VENTURA:

Malignant lymphoma, probable reticulum cell sarcoma, 2.

ORANGE:

Lymphosarcoma, 2; lymphoblastic sarcoma, 1; reticulum cell sarcoma, 2.

WALTER REED HOSPITAL:

Reactive hyperplasia, 1; lymphocytic lymphoma, focal, 1.

LOS ANGELES:

Reticulum cell sarcoma, 9; no diagnosis, 2.

FILE DIAGNOSIS: Lymphosarcoma 550-830 F

Cross-file: Reticulum cell sarcoma 550-831 F

CASE NO. 2, ACCESSION NO. 11077, David B. Dickey, M. D., Contributor

SAN FRANCISCO:

Undifferentiated malignant lymphoma, 1; pleomorphic reticulum cell sarcoma, 8; Hodgkin's sarcoma, 3.

OAKLAND:

Malignant lymphoma, 14 (Hodgkin's, 7, vs. pleomorphic reticulum cell sarcoma, 7).

CENTRAL VALLEY:

Malignant reticuloendotheliosis, 5; Letterer-Siwe's disease, 2; Hodgkin's disease, 4; stem cell lymphoma, 1.

SAN DIEGO:

Malignant reticuloendotheliosis, 2; undifferentiated reticulum cell sarcoma, 2; Letterer-Siwe's disease, 1; Hodgkin's sarcoma, 1; reactive hyperplasia in lymph node, 1.

WEST LOS ANGELES:

Malignant reticuloendotheliosis, 10; malignant lymphoma, reticulum cell type, 4.

VENTURA:

Malignant lymphoma, reticulum cell sarcoma, 1; Hodgkin's sarcoma, 1.

ORANGE:

Reticulum cell sarcoma, 5; Hodgkin's sarcoma, 1.

WALTER REED HOSPITAL:

Hodgkin's disease, 2.

LOS ANGELES:

Reticuloendotheliosis, 7; 4 considered it as probable reticulum cell sarcoma and Hodgkin's sarcoma.

FILE DIAGNOSIS:	Malignant lymphoma	550-839 F
Cross-file:	Hodgkin's disease	550-832 F
	Reticulum cell sarcoma	550-831 F

CASE NO. 3, ACCESSION NO. 9940, Frank R. Dutra, M. D., Contributor

SAN FRANCISCO:

Lymphosarcoma with pseudofollicular pattern, 12.

OAKLAND:

Giant follicular lymphoma in transition to lymphosarcoma, 15.

CENTRAL VALLEY:

Giant follicular lymphoma, 8; small cell lymphosarcoma with follicular pattern, 4.

SAN DIEGO:

Follicular lymphocytic lymphosarcoma, 8.

WEST LOS ANGELES:

Malignant lymphoma, nodular, with diffuse changes, lymphocytic type, 8; malignant lymphoma, lymphosarcoma type, 7.

VENTURA:

Lymphocytic lymphosarcoma, 1; Hodgkin's paragranuloma, 1.

ORANGE:

Lymphosarcoma, 7.

WALTER REED HOSPITAL:

Follicular lymphoma, lymphocytic type, 2.

LOS ANGELES:

Malignant lymphoma, but differed in classification, 11 (8 favored lymphocytic lymphosarcoma, others voted for polymorphous lymphosarcoma, giant follicular lymphoblastoma. All agreed that it had remnants of giant follicular lymphoblastoma).

FILE DIAGNOSIS: Malignant lymphoma	550-839 F
Cross-file: Lymphosarcoma	550-830 F

CASE NO. 4, ACCESSION NO. 12984, S. K. Abul-Haj, M. D., Contributor

SAN FRANCISCO:

Reactive hyperplasia, 2; pathologic hyperplasia, possibly Hodgkin's disease, 4; Hodgkin's disease, 1; reticulum cell sarcoma, 3.

OAKLAND:

Malignant lymphoma, 10 (Pleomorphic reticulum cell sarcoma, 7; Hodgkin's disease, 2; poorly differentiated lymphoblastic lymphosarcoma, 1); reactive node (viral?), 1.

CENTRAL VALLEY:

Hodgkin's disease, 5; reactive hyperplasia, 5; histiocytic or reticulum cell sarcoma, 2.

SAN DIEGO:

Reticulum cell sarcoma, 6; Hodgkin's sarcoma, 2.

WEST LOS ANGELES:

Reactive lymphoid pseudotumor, 2; sclerosing Hodgkin's disease, granuloma type, 2; malignant lymphoma, mixed lymphocytic and reticulum cell type, 11.

VENTURA:

Hodgkin's paragranuloma, 1; Hodgkin's sarcoma, 1.

ORANGE:

Chronic lymphadenitis, 4; reticulum cell sarcoma, 2; lymphoblastic lymphosarcoma, 1; Hodgkin's disease, 1.

WALTER REED HOSPITAL:

Reticulum cell sarcoma, 1; reactive hyperplasia, 1.

LOS ANGELES:

Malignant lymphoma, 11 (reticulum cell sarcoma, 3; malignant lymphoma, unclassified but favored Hodgkin's or reticulum cell sarcoma but could not make a definite diagnosis as to type,8)

FILE DIAGNOSIS: Malignant lymphoma 550-839 F

Cross-file: Reticulum cell sarcoma 550-831 F

JULY 1963

CASE NO. 5, ACCESSION NO. 12985, S. K. Abul-Haj, M. D., Contributor

SAN FRANCISCO:

Dermatopathic lymphadenitis, 5; Letterer-Siwe's disease, 8.

OAKLAND:

Letterer-Siwe's disease, 13; dermatopathic lymphadenopathy (lipomelanotic alteration), 2.

CENTRAL VALLEY:

Letterer-Siwe's disease, 6; reactive lymphadenitis, 5; plasmacytoma, 1.

SAN DIEGO:

Letterer-Siwe's disease, 6; reactive reticuloendotheliosis, 2.

WEST LOS ANGELES:

Letterer-Siwe's disease, 15.

VENTURA:

Chronic granulomatous lymphadenitis with melanotic reticulosis, 1; malignant melanoma, 1.

ORANGE:

Letterer-Siwe's disease, 4; reactive hyperplasia, 4 (poor "reactor" type, 1).

WALTER REED HOSPITAL:

Follicular lymphoma, lymphocytic type, 2.

LOS ANGELES:

Letterer-Siwe's disease, 11.

FILE DIAGNOSIS: Letterer-Siwe's disease 010-759
550-759

CASE NO. 6, ACCESSION NO. 12986, S. K. Abul-Haj, M. D., Contributor

SAN FRANCISCO:

Lipid-histiocytosis, type unknown, 8; histiocytic medullary reticulosis, 1; no diagnosis, 2.

OAKLAND:

Storage disease, further unclassified, 8; histiocytosis, further unclassified, 1; toxoplasmosis, 1.

CENTRAL VALLEY:

Confused, 12; myeloma, 4; histiocytosis, 3; reactive atypical hyperplasia, 1; don't know, 3; no vote, 1.

SAN DIEGO:

Multiple myeloma, 1; lipid histiocytosis, 1; reactive reticuloendotheliosis, 1; Waldenstrum's macroglobulinemia, 1; Boeck's sarcoid, 1; kala-azar, 1; abnormal lymph node, 1; deferred, 1.

WEST LOS ANGELES:

Thesaurosis, 15 (a. Dysproteinosis with abnormal storage of lipoglycoprotein, 13; b. Lipid storage disease, 2).

VENTURA:

Histiocytosis, 1; amyloidosis, 1.

ORANGE:

Reactive histiocytosis, type ?, 8.

WALTER REED HOSPITAL:

Megakaryocytic myelosis, 1; macroglobulinemia, 1.

LOS ANGELES:

Tentative diagnosis of that given by Drs. Sam Abul-Haj and Robert Lukes of dysproteinosis with abnormal storage of lipoglycoprotein was accepted by the group. This (if it is an entity) has not found its way into the literature and little is known about the patients that Drs. Abul-Haj and Lukes have had lymph nodes from, to study. Histochemically, PAS positive material (glycogen) is present in many of the histiocytic cells and fat can be demonstrated in many of the same type cells. Hyperglobulinemia has been found in some of their cases (total 4 seen) and is presumed to be a part of the picture.

FILE DIAGNOSIS:	Lymphadenopathy due to undetermined cause	550-Y10
	Hyperplasia, lymph node, due to unknown cause	550-943

DIAGNOSIS ON PENDING CASES

JULY 1963

CASE NO. 7, ACCESSION NO. 10958, D. Tatter, M. D., Contributor

FILE DIAGNOSIS: Lymphosarcoma 502-830

Cross-file: Malignant lymphoma, unclassified 502-834 F

The PAS stain did not reveal any torula, but there was a good deal of PAS in the cytoplasm of many of the cells

7-1-64

CASE NO. 7, ACCESSION NO. 10953, D. Tatter, M. D., Contributor

SAN FRANCISCO:

Reticulum cell sarcoma, 9; no diagnosis, 4.

OAKLAND:

Malignant lymphoma, further unclassified, with cytoxan effect (?), 15.

CENTRAL VALLEY:

Hodgkin's disease, 7; erythremic myelosis, 3; reactive hyperplasia, 1; no vote, 1.

SAN DIEGO:

Malignant reticuloendotheliosis, 3.

WEST LOS ANGELES:

Malignant lymphoma, lymphosarcoma type, 6; granulocytic leukemia, 8; agnogenic myeloid metaplasia, 1.

VENTURA:

Lymphosarcoma, 1; reticulum cell sarcoma, 1.

ORANGE:

Myeloid metaplasia, 4; lymph node with myeloid metaplasia secondary to cytotoxin treatment, 1; lymph node showing post-treatment lymphoma with myeloid metaplasia, 3.

WALTER REED HOSPITAL:

Reticulum cell sarcoma, 2.

LOS ANGELES:

Question was raised of the possibility of torulosis in leukemic node. Diagnosis deferred until PAS could be studied.

FILE DIAGNOSIS: Deferred, pending special stain

CASE NO. 8, ACCESSION NO. 12467, James W. Decker, M. D., Contributor

SAN FRANCISCO:

Benign hyperplasia, posterior mediastinal lymph node, 13.

OAKLAND:

Benign mediastinal lymph node hyperplasia (as described by Castleman and Iverson), 15.

CENTRAL VALLEY:

Reactive hyperplasia, 10; aberrant thymic tissue, 1; no vote, 1.

SAN DIEGO:

Lymphadenitis, nonspecific (reactive hyperplasia), 8.

WEST LOS ANGELES:

Atypical lymphoid hyperplasia (choristoma of Stout), 15.

VENTURA:

Lymphoid hamartoma, 1; mediastinal lymph node hyperplasia, 1.

ORANGE:

Hyperplasia, lymph node, 8.

WALTER REED HOSPITAL:

Reactive hyperplasia, 2.

LOS ANGELES:

Benign lymphoid tissue of the type described by Castleman, Iverson, Lattes, and Pachter, 11.

FILE DIAGNOSIS: Hyperplasia, lymph node, due to unknown cause 550-943

Reference:

Lattes, Raffaele and Pachter, M. Richard: Benign lymphoid masses of probable hamartomatous nature. Cancer 15:197, Jan. - Feb. 1962.

Castleman, B., Iverson, L, and Menendez, V. P.: Localized mediastinal lymph node hyperplasia resembling thymoma. Cancer 9:822-830, 1956.

JULY 1963

CASE NO. 9, ACCESSION NO. 12987, J. R. McGrath, M. D., Contributor

SAN FRANCISCO:

Lymphosarcoma, 7; reactive hyperplasia, 1; no diagnosis, 4.

OAKLAND:

Hyperplasia, lymph node (with varying degree of suspicion of malignancy), 7; malignant lymphoma, 8 (reticulum cell sarcoma, 5).

CENTRAL VALLEY:

Lymphosarcoma, 9; malignant lymphoma, not specified, 1; giant follicular lymphoma, 1; no vote, 1.

SAN DIEGO:

Lymphosarcoma, 3; lymphadenitis, 2.

WEST LOS ANGELES:

Malignant lymphoma, diffuse lymphosarcoma type, 10; malignant lymphoma, diffuse reticulum cell type, 5.

VENTURA:

Malignant lymphoma, Hodgkin's, 1; Hodgkin's sarcoma, 1.

ORANGE:

Lymphosarcoma, 8.

WALTER REED HOSPITAL:

Malignant lymphoma, probably associated with chronic lymphocytic leukemia, 2.

LOS ANGELES:

Malignant lymphoma, 11 (lymphosarcoma, 6; unclassified, 5).

FILE DIAGNOSIS: Malignant lymphoma 550-839 F

Cross-file: Lymphosarcoma 550-830 F

JULY 1963

CASE NO. 10, ACCESSION NO. 10692, Ben Fishkin, M. D., Contributor

SAN FRANCISCO:

Lymphosarcoma, 12.

OAKLAND:

Lymphosarcoma, 14; Hodgkin's paragranuloma, 1.

CENTRAL VALLEY:

Lymphosarcoma, 8; malignant lymphoma with chronic lymphocytic leukemia, 3; no vote, 1.

SAN DIEGO:

Lymphocytic lymphosarcoma, 3; Hodgkin's paragranuloma, 3.

WEST LOS ANGELES:

Hodgkin's paragranuloma, 7; malignant lymphoma, lymphosarcoma type, 7.

VENTURA:

Lymphocytic lymphosarcoma, 2.

ORANGE:

Lymphocytic leukemia or circulating cell sarcoma, 8.

WALTER REED HOSPITAL:

Hodgkin's paragranuloma, 1; lymphosarcoma, 1.

LOS ANGELES:

Malignant lymphoma, 11 (Hodgkin's paragranuloma type, 9; lymphocytic lymphosarcoma type, 2).

FILE DIAGNOSIS: Lymphosarcoma 550-830 F

Cross-file: Hodgkin's paragranuloma 550-832 F

JULY 1963

CASE NO. 11, ACCESSION NO. 11221, Herbert I. Harder, M. D., Contributor

SAN FRANCISCO:

Hodgkin's disease, 12.

OAKLAND:

Hodgkin's disease, 15.

CENTRAL VALLEY:

Hodgkin's disease (paragranuloma), 11; no vote, 1.

SAN DIEGO:

Hodgkin's granuloma, 8.

WEST LOS ANGELES:

Hodgkin's granuloma, 15.

VENTURA:

Hodgkin's granuloma, 2.

ORANGE:

Hodgkin's granuloma, 6.

WALTER REED HOSPITAL:

Hodgkin's disease, 2.

LOS ANGELES:

Hodgkin's disease, 11.

FILE DIAGNOSIS: Hodgkin's disease 550-832 F

JULY 1963

CASE NO. 12, ACCESSION NO. 12860, W. K. Bullock, M. D., Contributor

SAN FRANCISCO:

Hodgkin's disease, 12.

OAKLAND:

Hodgkin's disease, 14; sarcoma (lipo?), 1.

CENTRAL VALLEY:

Hodgkin's disease (granuloma), 11; no vote, 1.

SAN DIEGO:

Sclerosing Hodgkin's granuloma, 8.

WEST LOS ANGELES:

Hodgkin's granuloma, 13; Hodgkin's sarcoma, 2.

VENTURA:

Hodgkin's granuloma, 1; sclerosing Hodgkin's disease, 1.

ORANGE:

Hodgkin's granuloma, 7.

WALTER REED HOSPITAL:

Hodgkin's disease, 1; sclerosing Hodgkin's disease, 1.

LOS ANGELES:

Hodgkin's disease, 11.

FILE DIAGNOSIS: Hodgkin's disease 550-832 F

DIAGNOSIS ON PENDING CASES

JULY 1963

CASE NO. 7, ACCESSION NO. 10958, D. Tatter, M. D., Contributor

FILE DIAGNOSIS: Lymphosarcoma 502-830

Cross-file: Malignant lymphoma, unclassified 502-834 F

The PAS stain did not reveal any torula, but there was a good deal of PAS in the cytoplasm of many of the cells

7-1-64

JANUARY 1964

CASE NO. 8, ACCESSION NO. 11887, E. F. Ducey, M. D., Contributor

FILE DIAGNOSIS: Myxoid liposarcoma 083-872 F

7-1-64

JR. STUDY GROUP July 1963

TUMORS OF THE FEMALE REPRODUCTIVE ORGANS
(From cases presented September 1961)

No.	Diagnosis	Accession & Case No.
1.	Reticulum cell sarcoma of ovary Cross file: Dysgerminoma; lymphocytic lymphoma; anaplastic carcinoma)	Acc. 11429 (Case No.2)
2.	Mucinous cystadenocarcinoma of ovary Cross file: Mesometanephric rest tumor (Hertig) Mesonephroma	Acc. 11256 (Case No.3)
3.	Mucinous cystadenocarcinoma of ovary Cross file: Mesometanephric rest tumor (Hertig) Mesonephroma	Acc. 11031 (Case No.4)
4.	Brenner tumor of ovary	Acc. 11403 (Case No.5)
5.	Primary adenocarcinoma of fallopian tube of alveolar medullary type	Acc. 10992 (Case No.6)
6.	Infarcted benign adnexal cyst	Acc. 10900 (Case No.7)
7.	Stromal sarcoma Cross file: Endolymphatic stromal myosis	Acc. 11497 (Case No.8)
8.	Malignant mixed mesodermal tumor (Malignant mixed mullerian tumor)	Acc. 11538 (Case No.9)
9.	Clear-cell adenoacanthoma involving both ovary and fallopian tube (cannot tell which is primary)	Acc. 10628 (Case No.11)
10.	Adenoacanthoma of ovary, primary in endometriosis	Acc. 11527 (Case No.12)

Reticulum cell sarcoma of ovary

Acc. 11429
(Case No. 2)

General data

These lesions are rarely discussed in the literature, as probably only two cases of primary lymphosarcoma of the ovary have been reported to 1962.[2] [3] By "primary" is meant that there is one site of origin and that all secondary sites are the result of metastases.

Clinical data

The lesions occur at almost any age (young childhood cases have been reported by Burkitt and O'Conor [1]). They have presented abdominal masses with few signs and symptoms, abdominal pain and bladder symptoms being most common. Laboratory findings are normal in cases considered primary. In secondary cases, findings consistent with usual lymphosarcomas are present. X-ray may be helpful in localizing the mass.

Etiology and pathogenesis

A difference of opinion exists as to whether any lymphomas are unicentric or multicentric in origin. Nelson and his associates, as well as Zeigerman and his, contend that these cases of lymphosarcoma of the ovary favor the theory of multicentric origin. However, Willis and Stout believe that certain lymphomas are unicentric in origin and may originate in extranodal sites.[2]

Gross and microscopic pathology

Grossly the ovaries were replaced by lobulated white, rubbery homogeneous tumors up to 15 cm. Our case has omental implants but no lymphadenopathy.

Treatment and prognosis

Treatment consists of surgical removal followed by irradiation. The prognosis is probably the same as any lymphosarcoma. Collins and Piper's case survived almost five years before dissemination.

Discussion

This case of unilateral ovarian tumor in a 45-year old woman has been followed since 1960. W.K. Bullock thinks it is primary in the ovary but prefers the diagnosis of small-cell dysgerminoma. P. Melnick leaned toward a granular-cell tumor. M.B. Dockerty's diagnosis is a lymphoblastoma of the reticulum-cell type. Time will give the answer. Collins and Piper's case has survived five years and only recently manifested lymphnode involvement, while Duggan's case survived two years at the time of the report.

Gonadal involvement (ovarian and testicular; secondary) was a prominent feature in the cases of African children reported by Burkitt and O'Conor. Their cases seem to form a readily recognized clinical syndrome with the initial presentation being a jaw or orbital tumor or as an abdominal swelling. Thus their 106 cases had predominantly extra-nodal genesis and a wide anatomical distribution.

Other case reports are associated with disseminated disease and lymphnode involvement. Testicular involvement seems to occur with about the same rarity; most cases were part of generalized lymphomas.

Reticulum cell sarcoma of ovary (cont'd) Acc. 11429 (Case No. 2)

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1. Burkitt, D. and O'Conor, G.T. Malignant Lymphoma in African Children, I and II. Cancer 14:258, 1961.
2. Collins, James and Piper, P.G. Lymphosarcoma of Ovary. Report of a case with review of the literature. Obst.& Gynec. 20:686-9, 1962.
3. Duggan, A.R.H. Lymphosarcoma of Ovary. M.J. Australia 44:792, 1957.
4. Durfee, H.A., Clark, B.F., and Peers, J.H. Primary Lymphosarcoma of Ovary: Report of a case. Am.J. Cancer 30:567, 1937.
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9. Willis, R.A. Pathology of Tumors. 2nd Ed. Mosby, St.Louis,1953.
10. Zeigerman, J.H., Imbriglia, J., Makler, P., and Smith, J.J. Ovarian Lymphosarcoma: Review of a case with review of the literature. Am.J.Obst. & Gynec. 72:1357, 1956.

Mucinous cystadenocarcinoma, nongravid

Acc. 11256
(Case No. 3)

Clinical data

Mucinous cystadenocarcinomas are most commonly found in women during the fourth to sixth decades.

Etiology and pathogenesis

This lesion is a malignant prototype of mucinous cystadenoma. It is estimated that five percent of benign cystadenomas undergo malignant change.

Prognosis

Mucinous cystadenocarcinomas have a predilection for metastasizing to the uterus.

Discussion of case

Patient, a 63-year old caucasion female, eight years into menopause, developed a pelvic mass on the right side; no other significant history; laboratory studies unrevealing. Physical examination revealed a nonfixed right adnexal pelvic mass. Past medical history, "ovarian cyst" removed 35 years earlier.

At surgery a tumor involving the right ovary was removed; tumor measured 15 x 12 x 10 cm. and weighed 520 gm. Surface of the tumor was smooth and glistening with deep fissures. The parenchyma consisted of dense stroma with mucoid cysts and a more cystic, softer, yellow stroma. No distinct papillary formation was noted. Three months after surgery, patient was feeling well.

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1. Hertig, A.T. and Gore, H. Fascicle of Tumors of the Female Sex Organs. Armed Forces Institute of Pathology, 1961.

2. Novak, E. and Jones, G.S. Gynecology Textbook. Williams and Wilkins Co., Baltimore, 1961.

Mesonephroma of ovary

Acc. 11256
(Case No. 4)

General data

Mesonephroma was first described by Peham in 1899 as a tumor formed by columnar cells and arranged in a pattern similar to the classic hypernephrones. In 1939 Schiller described this tumor as being of mesonephric origin because of a resemblance of the tumor cells to the endothelium of the glomerulus, a generalized tubular pattern histologically resembling the mesonephric tubules, sometimes with structures resembling glomeruli, and a tendency to involve not only the ovary but other areas compatible with a mesonephric origin. Schiller named this tumor mesonephroma ovarii and pointed out that owing to the close relationship of the mesonephros and the gonad in the developing embryo, such a tumor could arise as a result of inclusion of a portion of mesonephros in the ovary. Teilum in 1954 grouped all clear-cell carcinoma of ovary as mesonephric carcinoma. He believes there are two varieties of mesonephroma: 1) mesonephroma of Schiller, arising from a primordial germ cell and is a tumor intermediate between a dysgerminoma and chorion-epithelioma and 2) true mesonephroma which arises from mesonephric nests. More recent suggestions on histogenesis is that the ovarian clear-cell carcinomas originate in a tissue more primitive than mesonephros, arising in the premesonephric mesodermal tissue. (The metanephros and not the mesonephros gives rise to the definitive kidney.)

Novak supports the mesonephric origin of the tumor. He has described a series of tumor of the female genital tract situated variously in the ovary, broad ligament, cervix, vagina, each of which sites have been identified with the mesonephros. Novak and associates in 1959 contended that Schiller's tumor should be amalgamated with the clear-cell carcinoma group, that the entire group should be considered mesonephric, that too much was made of the glomeruloid structure, and that all these tumors are basically of similar histology.

Clinical data

The age distribution ranges from 8 months to 69 years, but the majority of the cases occur in the fifth and sixth decades. Nearly 50 percent of the patients were nulliparous.

This type of lesion accounts for less than five percent of ovarian carcinomas.

Symptoms were those of abdominal mass and pain observed in cases where the tumor had twisted on its pedicle. Bleeding was an uncommon complaint. Mesonephroma is apparently endocrinologically inert.

Gross and microscopic pathology

The tumors tend to range from a medium to large size. The right and left ovary are involved with equal frequency. The tumor may have a smooth surface, may be involved by inflammatory neoplastic adhesions. It may be cystic, multilocular or unilocular, or solid. The cystic tumor grossly cannot be distinguished from the common cystadenocarcinoma. Intracystic, intramural, and extramural papillary and solid growths are gross features of these clear-cell carcinomas. In the solid form, minute cysts embedded in a dense fibrous stroma are present.

Mesonephroma of ovary (cont'd.) Acc. 11256 (Case No. 4)

Pathology (cont'd.)

Microscopically the cells are clear, with nuclei uniform in size and shape, and are arranged lining tubular or cystic spaces, with an extremely delicate and highly vascular stroma. The stroma in certain cases appeared as primitive mesenchymal tissue, appearing to generate clear cells. The patterns found are: alveolar (sheets of clear cells) papillary, tubular, polylocular (widely separated, large tubular and cystic spaces surrounded by dense connective tissue).

The Schiller's feature of the tumor is that of tubules lined by low cuboidal epithelium heaped up over a connective tissue core on which capillaries are found. This bud projects into the lumen of the tubule and may create an imperfect impression of a glomerular unit. Considerable variation is often noted.

The two distinct patterns characterized by the large clear cells resembling, if not identical with, hypernephroma and the Schiller's "glomeruloid" pattern may occur independently; but, it is not uncommon to find them both in the same lesion and adjacent microscope fields. Fat and mucin stains are inconclusive.

Treatment and results

The mesonephroma must be considered as a malignant tumor. Therefore the treatment is bilateral salpingo-oophorectomy and hysterectomy followed by high-voltage roentgenotherapy.

Prognosis is guarded and is about the same for carcinoma of the ovary in general; 24 percent of the cases had a 5-year survival.

In the region of the ovary, mesonephroma seems to be a great deal more lethal than in other sites, although it is the same basic lesion derived from mesonephric remnants. In the ovary it is characteristically "silent" until it is too far advanced for beneficial therapeutic results, while in the more exposed portions, such as cervix or vagina, early diagnosis may lead to more favorable results.

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Brenner tumor of ovary

Acc. 11403
(Case No. 5)

General data

The Brenner tumor is a fibroepithelial tumor of nests of transitional-like epithelium infiltrating a dense fibrous stroma. It is not generally considered to be hormone-producing tumor. It was first described by Brenner in 1907 under the name "oophoroma folliculare."

Clinical data

The tumor has been reported in females from ages 6 through 81, but it is unusual below age 30; over 50 percent of the tumors occur in females over 50. There is no known racial predilection. Some cases present an abnormal uterine bleeding, but most symptoms are only those referrable to a nonspecific pelvic mass.

Etiology and pathogenesis

Several theories exist concerning the origin of the tumor:

1) The most popular is that this tumor arises from the Walthard cell rests (Wolfian urogenital epithelium found beneath the ovarian capsule at the hilus and in the subserosa of the Fallopian tube), which is probably a modified coelomic epithelium. This is thought to explain why the Brenner tumor occurs so frequently in conjunction with mucinous cystadenomata. However, this theory is inconsistent with the fact that the Brenner tumor does not occur in the Fallopian tubes where the Walthard cell rests are so common.

2) Teoh contests the theory of origin from Walthard cells since these cells stain differently from the epithelial nests in the Brenner tumor. He feels the Brenner tumor is from the granuloma-Theca cell series which has undergone metaplasia to form the epithelial cells in the tumor. This would be consistent with the name "oophoroma folliculare" as originally given the tumor by Brenner.

3) Microscopic continuity between the tumor and the surface mesothelium as demonstrated by Greene has been presented as evidence for origin from this structure.

4) Greene further demonstrated continuity between the tumor and the uninvolved rete-ovarii and suggests this also as an origin for the tumor.

Gross and microscopic pathology

The tumor is usually solid with smooth subserosal surface and varies in size from microscopic dimensions to 30 cm. The largest reported is 6,800 gm. The tumor may be bilateral in up to 10 percent of the cases. The cut surface is grey-white and whorled.

Microscopically the tumor is composed of abundant fibrillary connective tissue with epithelial nests of compact, polyhedral squamous-like cells having oval nuclei showing longitudinal grooving and indistinct nucleoli. Histochemical staining characteristics show variation: PAS and alcian-blue positive material may be seen in the epithelial agtregates, or not, as evidenced by its presence in a right ovarian but not in a left ovarian tumor in the same patient.

Brenner tumor of ovary (cont'd.) Acc. 11403
(Case No. 5)

Pathology (cont'd.)

These may be fat-positive material in the stroma. Silver stains show delicate network in the connective tissue but not in the epithelial masses, whereas reticulum stains may show some fibrils between the epithelial cells also.

The tumor is to be distinguished from metastatic epidermoid carcinoma.

Treatment and results

Simple excision is curative; however, rare instances of malignancy have been recorded in which epithelial nests were present in the lymphatics.

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3. Teoh. J.Path.& Gynec. 64:441, 1953.

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Primary adenocarcinoma of oviduct

Acc. 10992
(Case No. 6)

This lesion is the rarest carcinoma of the female genital tract. The age of onset is predominantly middle-age, but has been found in the third through the eighth decades; the mean age is 52 years.

The commonest symptoms are vaginal discharge, history of menstrual irregularities, and pain. Many authors associate sterility with tubal adenocarcinoma. Rarely is this lesion diagnosed preoperatively.

The laboratory findings are not pathognomonic; the x-ray findings may be contributory with uterosalpingography.

Etiology and pathogenesis

(Pathogenesis is unknown.)

Tuberculous salpingitis induces a marked adenomatous response and has lead to false diagnosis of adenocarcinoma of the tube.

Gross and microscopic pathology

Usually the lesion is unilateral, but bilaterally occurs in approximately a third of the cases. If the fimbriated end is closed, a hydro or pyosalpinx will result. The external surface is roughened and adherent to the floor of the pelvis. The tumor is usually found in the distal third and may fungate through the open fimbriated end. Primary tumor of the tube is usually confined to the tube and grows as a papillary endosalpingeal tumor.

Microscopically the criteria for primary adenocarcinoma are:

1) Epithelium of the endosalpinx is replaced by adenocarcinoma.
2) Histologically the cells resemble those of the endosalpinx.
3) Endometrium and ovaries are normal, have a benign lesion or contain a malignant lesion which appears to be metastatic from the tube.
4) Prime involvement is in the endosalpinx and perisalpinx: the lymphatics of the muscularis and mesosalpinx are rarely involved.
5) Tuberculosis must carefully be excluded.

Other criteria:

Grade I papillary: Columnar nonciliated cells, confined to mucosa and mitoses are scanty.

Grade II papillary-alveolar: Gland formation is evident with early invation of tubal wall.

Grade III alveolar-medullary: Solid sheets of cells or gland-like spaces but lacking a papillary pattern; frequent mitoses with lymphatic invasion.

Treatment and prognosis

Total hysterectomy with bilateral salpingo-oophorectomy, followed with x-ray. Prognosis is fair, with a 40 percent, 5-year survival and better results in the well-differentiated tumors.

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Infarcted benign adnexal cyst

Acc. 10900
(Case No. 7)

General data

Rests, cysts, and tumors of the adnexae have been designated by Gardner as:

1) Mesonephric body--wolffian body; organ of Rosenmuller; epoophoron; parovarian; Kobelts-tubules; parovarial tubule.
2) Mesonephric duct--wolffian duct; longitudinal tubules of epoophoron.
3) Mesonephric tubules--wolffian tubules; epoophoral tubules.
4) Paramesonephric duct--mullerian duct.
5) Oviduct and uterine tube--fallopian tube.
6) Accessory oviducts--accessory fallopian tubes.

Etiology and pathogenesis

Cysts of the pelvic supporting structures can be differentiated as to wolffian or mullerian origin by their microscopic details.

In those of wolffian (metanephric) origin, a basement membrane is present. The epithelial cells are low cuboidal, nonciliated in type with prominent central moderately vesicular, deeply staining nuclei. Fifty percent of intraligamentous cysts are of mesonephric tubule origin, while another 5.7 percent come from mesonephric ducts.

Cysts of mullerian (paramesonephric) origin lack a basement membrane and have epithelium identical with the oviduct. There are pale vesicular ciliated cells, nonciliated secretory cells and peg cells. This epithelium responds to steroid hormones in the same manner as the oviduct. This type constitutes 38.5 percent of intraligamentous cysts.

Discussion

This lesion is felt to be of mesonephric origin because small tubules with a basement membrane and lined by typical epithelium are present. The large cystic spaces lack epithelium, but basement membranes are present. Because of the infarcted state and the large size of the spaces, the loss of epithelium is not surprising.

Many thrombosed vessels are present. Some of these are large and dilated, causing some observers to classify the lesion with the vascular tumors. However, elastic and reticulum stains fail to show blood vessel anatomy in the walls of the large cysts.

The prognosis for cysts of mullerian and wolffian origin is uniformly good.

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Endolymphatic stromal mycosis

Acc. 11497
(Case No. 8)

Also called: Stromal endometriosis, endolymphatic fibromyosis, adenomyoma without glands, stromatosis, etc.

General data

Described in detail by Hunter (1953, 1962) as a rare aberrant proliferation of endometrial stroma invading the vascular system (esp. the lymphatics) and dissecting between muscle bundles. Cases usually have good prognosis in spite of local (pelvic) recurrence.

Clinical data

Incidence: Females between 25 to 50 years. Patients usually have menstrual bleeding, with or without pelvic tumor. Diagnosis is not made preoperatively but can occasionally be made at operation. Less than 100 cases have been reported. There is no evidence of hereditary transmission.

Etiology and pathogenesis

Etiology is unknown but is occasionally associated with frank adenomyosis uteri and endometriosis. It is therefore possible that these have a common cause.

Gross and microscopic pathology

The uterus is enlarged and the myometrium appears to be replaced subserosally by soft yellow tissue. Similar tissue extends either in a nodular or vermiform manner into the broad ligament and often extends to the lateral pelvic wall. The uterine cavity may be enlarged and distended by fundal polypi having a wide firm base and composed of soft yellow tissue. They may be necrotic. Cut surface of the thickened uterus reveals multiple round, nonencapsulated, yellow, soft tumors. Radiating from these tumors are infiltrating cords of tissue which have a somewhat elastic rubbery consistency and can be pulled out of the vascular spaces they occupy.

Microscopically the tissue consists of cells resembling the endometrial stromal cells of the late proliferative phase. Many thick-walled blood vessels are seen. Occasional areas of anaplasia and mitotic activity are present. In polypi the overlying epithelium appears to play no part in the tumor formation. Many areas of apposition and invasion of lymphatics and occasionally small veins can be seen. Metastases are rare (but not necessarily fatal). Recurrence is not invariable, even though tumor cords are across the surgical resection margin.

Treatment and results

Treatment is by total hysterectomy with bilateral salpingo-oophorectomy; in addition, most patients receive postoperative pelvic x-ray.

Endolymphatic stromas myosis (cont'd.) Acc. 11497 (Case No. 8)

Treatment and results (cont'd.)

The longest case, followed for 28 years, survived in spite of multiple recurrences for which she was reoperated. Fatalities due to the tumor are unknown.

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Mixed mesodermal tumor of the uterus

Acc. 11538
(Case No. 9)

General data

Mesodermal mixed tumors of the uterus are rare. The term "mixed" is doubly appropriate as it describes the histopathologic lesion and indicates the confusion and terminology that has existed for years and still exists. According to Perlstein[5] the first case was described by Wagner in 1854, even though he considered the neoplasm to be a chondrosarcoma.

A mixed mesodermal tumor is composed of a variety of types of mesodermal tissues, cartilage, striated muscle, myxomatous tissue, fat, and bone.[6] In order for a new growth to qualify as a mixed mesodermal tumor, the neoplasm should be composed of two or more heterologous elements.[3] Various terms used in recent years are: carcinosarcoma, mixed mullerian tumor, sarcoma botryoides, mesenchymal sarcoma, mixed mesenchymal mullerian sarcoma, and others.

Clinical data

It is believed that the incidence of these tumors is four to eight percent of all malignant neoplasms of the female genital tract.[6] Traditionally, sarcoma botryoides is a grapelike form of mixed mesodermal tumor occurring in the vagina of young children, the rare mixed cervical-type during active menstruation, and the fundal type after menopause. The average age of patients developing mixed tumors of the uterus has varied in different series from 47 to 58.9 years.[3],[4] In other words, usually after menopause, parity has no influence in the occurrence.[7]

Symptoms and signs of the lesion include vaginal bleeding preceded by vaginal discharge, either purulent or otherwise. The source of the bleeding is multiple friable polypoid masses attached to the anterior or posterior wall of the endometrium protruding in many instances from the external os of the cervix into the vagina, or protruding outside the vagina. The pelvis may become filled with tumor and these may be pressure symptoms of pain, urinary and rectal disturbances, and edema of the lower extremities. Ascites may occur. Pain and weight-loss rarely occur. Diagnosis can be made by biopsy.

Etiology and pathogenesis

The etiology of these tumors is unknown.

There has been a variety of opinions regarding the histogenesis of these tumors. They are now generally believed to be of mullerian duct origin[4] or derived from tissues which develop from the mesenchyme formed from and surrounding the mullerian duct in the urogenital ridge. The mesenchymal components may include a variety of connective tissue and epithelial elements.[2] The epithelial elements, when present, have a more restricted capacity for differentiation and are limited in histological type to those usually seen in endometrium, cervix, fallopian tubes, or vagina. Rhabdomyosarcomatous and chondrosarcomatous components are the most common heterogeneous elements.[2]

Mixed mesodermal tumor of the uterus (cont'd.) Acc. 11538 (Case No. 9)

Etiology and pathogenesis (cont'd.)

The presence of rhabdomyoblasts is no longer considered necessary for the diagnosis of a mixed mesodermal tumor of the uterus or the more preferred term "malignant mixed mullerian tumor." It is now believed that in the family of tumors the site of origin is the differentiated endometrial stromal cell. Endometrial stroma, though fully differentiated, is the least highly specialized cell type derived from mullerian mesenchyme.4/ In tissue culture, the character of the malignant cells is thought to indicate a stromal origin. A common histogenesis need not apply a common pathogenesis, as evidenced by sarcoma botryoides.

Gross and microscopic pathology

In the uterus, these tumors can arise from either the anterior or posterior wall of the fundus. The uterus may be markedly distended. The tumors are composed of multiple polypoid masses which are fragile and bleed easily. They are usually attached by a fairly broad base and often do not penetrate the deeper musculature. They are white or gray to red in color. The cut surfaces are usually homogeneous with small cysts and sometimes with necrosis and hemorrhage. The nonneoplastic endothelium is usually thin and pale.

On microscopic examination, the polypoid masses consist of intermingled sarcomatous or sarcomatous and carcinomatous elements with varying degrees of differentiation. It is no longer believed that the demonstration of rhabdomyoblasts is necessary for diagnosis. There may be histological evidence of myometrial invasion.

Metastases may be pure carcinoma or pure sarcoma.1/ Metastases to lymphnodes is infrequent. Metastases may occur to the peritoneum, mesentery, liver, lungs, and pleura. Skeletal and brain metastases are rare. Local recurrent and regional infiltration of the bladder, parometrium, vagina, and perineum are frequent. Metastases occur early in comparison to endometrial carcinoma.

Treatment and prognosis

Total hysterectomy with bilateral salpingo-oophorectomy is the treatment of choice. These tumors are radiation-resistent.1/ The average survival after diagnosis is 6 to 12 months, although there are reported cases of 9-year survival or more.3/

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Clear-cell adenocarcinoma involving both ovary and fallopian tube (cannot tell which is primary)

Acc. 10628
(Case No.11)

General data

Carcinoma of the fallopian tube was first described by Orthmann in 1886. It is the rarest of all gynecological carcinomas. The incidence varies from 0.16 percent to 0.5 percent of such carcinomas. They occur primarily unilaterally.

Clinical data

The average age is 52, but cases have been described ranging from 18 to 80 years. The most common symptom is vaginal discharge. Occasionally an abdominal mass can be palpated. Sterility appears to be associated with these lesions.

Etiology and pathogenesis

The etiology is unknown. Some believe that salpingitis predisposes to carcinoma, but this theory is not generally accepted, as the former is very common, and the carcinoma rare. Others believe that tuberculosis is an etiologic factor; however, it is thought that the marked adenomatous response to tuberculosis may occasionally lead to a false diagnosis of neoplasia.

Gross and microscopic pathology

The tumor is usually unilateral. If the fimbriated end of the tube is closed, then the tube has the gross appearance of a hydrosalpinx or pyosalpinx. The external surface of the tube is roughened and often adherent to the pelvic floor or intestine. Cut surface of the tumor is granular, gray to yellow, and friable. The tumor is most commonly found in the distal third of the tube.

Microscopically, there are several criteria used to determine whether it is a primary neoplasm of the fallopian tube, as follows:

1) The epithelium must be replaced by adenocarcinoma.
2) The histologic character of the cells must resemble epithelium of endosalpinx.
3) The endometrium and ovaries are normal, affected by a benign lesion, or contain a malignant lesion that by its small size, distribution, and histological characteristics appears to be metastatic from the tube.
4) The prime involvement is the endosalpinx.
5) Tuberculosis has been carefully excluded.

Clear-cell adenocarcinoma involving both ovary and fallopian tube (cont'd.) Acc. 10628 (Case No.11)

Pathology (cont'd.)

Classification of carcinoma of the fallopian tubes is as follows:

Grade I: Papillary type--tumor is papillary in character and confined to mucosa.

Grade II: Papillary-alveolar type--papillary projections and gland formation, with invasion of tubal wall.

Grade III: Alveolar-medullary--gland formation and areas of solid sheets of cells, with invasion of lymphatics.

Treatment and prognosis

Total hysterectomy and bilateral salpingo-oophorectomy followed by radiation. The prognosis is poor; 40 percent have a 5-year survival.

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Adenoacanthoma and adenoacanthoma arising in endometriosis of the ovary (cont'd.) Acc. 11527 (Case No. 12)

Treatment and results

As far as treatment is concerned, the usual complete pelvic operation (hysterectomy and bilateral salpingo-oophorectomy) is performed. Most authors feel, however, that the prognosis is far better for adenoacanthoma of the uterus and the ovary than the usual adenocarcinoma.

Novak feels that squamous metaplasia occurs only in his low-grade adenocarcinomas. In fact, Thompson lists his criteria for removing a unilateral ovarian adenoacanthoma, and preserving the opposite ovary, bute, and uterus.

(Of course, some authors report results that do not justify an optimistic prognosis.)

The problem of adenoacanthoma as to etiology, natural history, and prognosis is not settled. There is no single, comprehensive study with long-term follow-up and large series.

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