Bulletin of

Staff Meeting

University of Minnesota Hospitals

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Case Reports

INDEX

I. MEETING OF STAFF

Date: April 19, 1934

Place: Recreation Room, Nurses' Hall

Time: 12:16 to 1:16

Attendance: 118

Program: Breast Tumors

Discussion: W. K. Stenstrom
L. G. Rigler
W. T. Peyton
O. J. Campbell

Theme: W.K.S.: Explained report of results of treatment of breast carcinoma here.

L.G.R.: This patient represents a remarkable example of what a patient can have and still live. Skull shows striking, extensive osteoclastic and osteoblastic metastasis. Note numerous irregular mushy looking areas of lessened density. Femora shows similar areas throughout both shafts. Irregularity is striking feature. Chest film shows involvement of clavicle, scapula, humerus, and almost all the ribs. Thoracic spine shows areas of increased and decreased density with almost half vertebra deformed and crushed. Striking feature, of course, is extensive character of metastasis. I believe these areas of increased density represent transformation in the presence of metastasis to bone producing process. Skull shows much the same type of process, i.e., multiple areas of rarefaction alternating with increased density. No evidence of involvement in the feet but note this tumor in the hands.

W.T.P.: There are so many things to discuss it is difficult to know just what to take up. If we would arrive at an ideal condition all patients would come when a lump appears and we could not distinguish between benign and malignant change. First symptom is usually lump in breast. Question of prognosis depends upon lymph node involvement. 75% of all cases with carcinoma of breast have involved nodes. Question is what to do with breast in most cases. In patients under 25 years of age carcinoma is a very rare disease; in fact, only one carcinoma in approximately 1000 will occur under 25 years of age. When you get in the next group, 25 to 30, it jumps up 1%, then about 5% in next 5-year period. Lump in breast becomes more dangerous lesion when patient gets older. Under 25 usually fibroadenoma of breast. After 40 there is so much chance of carcinoma developing in breast we should investigate all definite lumps.

O.J.C.: Anything I can say to you will be trite and will have been said many times. Am sorry I did not hear whole presentation. It is difficult problem no matter how much we see them. Three main problems. First, diagnosis. Dr. Peyton just outlined how we feel about dealing with lesions in breast. We have certain definite principles to follow. In case of doubt remove tissue and find out what you are dealing with. Never make an exception to this rule.

In spite of fact that in my mind we have found no relationship between cystic disease and carcinoma of the breast, all persons having cystic disease, as they get older might have affected breast removed because of fear of carcinoma developing without detection. Case in dispensary showed numerous nodules in breast. Woman carried lumps 20 years, thought nothing of it. Thought nothing when new one developed, but it was carcinoma. Second consideration is question of borderline lesion under microscope. Is it cancer or some form of hyperplasia. I think borderline tumors should be considered apart from malignancy. The case which cannot actually be called benign should be considered as separate group even though we might do radical procedure to avoid undue optimism. Third consideration: Having made up mind that it is cancer, the best treatment is radical surgery. No material improvement in surgery of breast since Halsted operation in 1892. In using x-ray and radium (particularly radium) I have seen

impressed with the fact that radium in sufficient quantities can destroy cancer even though most breast tumors are considered radioresistant. Prognosis can be improved to certain extent. We are limited by the fact that we do not get cases until disseminated throughout body. Few axillary metastasis cases ought to be treated by surgery. X-ray treatment is used in the hope that they are radiosensitive tumors. We will get just as long survivals as though we treated them by surgery. We are getting more and more information about radium. In England they are treating with radium. Memorial Hospital is treating some. I believe we are going to make some progress by the use of interstitial radiation associated with surgery. I deplore habit of doing radical amputation of breast with backache and other evidence of remote metastasis.

Dr. Campbell then gave illustrated talk on various points in diagnosis and treatment of carcinoma of breast.

Gertrude Gunn,
Record Librarian.

II. PROGRAM TODAY

We vary our method of presentation for today. The histories will be presented by the clinicians, the comments by those who had charge of the cases. Copies have been submitted in advance. This is an attempt to learn which method is the most effective for the group; other types will be tried.

III. CASE REPORTS

1. ACUTE INFECTIOUS JAUNDICE. TERMINAL SEPTICEMIA.

Case is white, female infant, 3 months old, admitted to University of Minnesota Hospitals 2-16-34, expired on same day.

2-13-34 - Perfectly well up until this time. On this day, the child had a slight elevation of temperature and vomited several times.

2-14-34 - Had 2 clat-colored stools. Marked jaundice. Fever receded and vomiting stopped.

2-15-34 - Jaundice increased. Several clay-colored stools.

2-16-34 - 7 A.M. - Severe generalized convulsion. Temperature 99, pulse 100, respirations faint and shallow. Retraction of head. Positive Kernig's sign. Reflexes hyperactive. Convulsions controlled with warm packs. Had residual spasticity, some hyper-irritability and a positive Chvostek sign. Enema given, normal yellow stool obtained. 11:30 A.M. - Convulsion recurred in more severe form. Had an emesis of several ounces of dark brown material. Temperature 104, pulse 120, respirations very slow and shallow (sighing character). Unable to swallow. Admitted at 3 P.M. Moribund. Marked jaundice present. Temperature 105.8. Having slight convulsive movements of limbs. Respirations have practically creased but the heart tones can still be heard. 3 minims of adrenalin administered intramuscularly. Few moments later, heart tones ceased. 5 minims of adrenalin given intracardially. Artificial respiration for several minutes without result. Expired at 3:10 P.M.

Family History

In neighborhood from which patient comes, several cases of jaundice are reported at the time the patient became ill. Two relatives of the patient were jaundiced, one of these (the grandmother) showed a residual paralysis following the illness.

Autopsy

Body is well-developed and well-nourished, white, female infant, 3 months old. No rigor or edema. Slight hypostasis present. No cyanosis. Marked jaundice present. Pupils are equal. Marked dependent lividity present.

Peritoneal Cavity is normal. Appendix is free and anterior, showing no disease.

Pleural Cavities and Pericardial Sac are normal.

Heart shows numerous petechiae of muscle, both on epicardial and endocardial surfaces.

Lungs show numerous petechial hemorrhages on surface and in parenchyma.

Spleen shows evidence of slight hyperplasia.

Liver weighs 200 grams and is very hard and fibrotic on section. Normal liver markings are obliterated. There are small yellow areas through the substance. Liver has a marked jaundiced color. Bile is quite mucoid, containing no bile pigment. Bile ducts show no obstruction, edema or inflammatory reaction.

Gastro-Intestinal Tract, Pancreas and Adrenals are normal.

Kidneys show extensive fatty change throughout. There are several petechial hemorrhages in the pelves.

Bladder is normal.

Genital Organs show bilateral hemorrhagic cysts in ovaries.

Thymus shows several small petechial hemorrhages.

Head shows a peculiar fatty deposit on osseous surface of dura. External surface of brain is grossly negative. Section of brain shows diffuse petechiae throughout cerebrum and cerebellum, most marked in cerebrum around ventricles.

Diagnoses:

1. Acute infectious jaundice.
2. Terminal septicemia.

Bacteriology (B.J.O.):

Cultures taken of liver, spleen and brain - all negative on brain broth, blood agar and eosin-methylene blue plates. If any infectious agent was present, it may have been in a virus form.

Microscopic:

With exception of liver, none of the organs show any significant change.

There are hemorrhagic cysts in the ovary; small patches of atelectatic lung parenchyma; congestion of blood vessels of brain without hemorrhage.

Liver - can only be recognized by the presence of bile ducts within the tissue. These are proliferating and forming many new biliary vessels. The liver substance has no resemblance whatsoever to liver cords. The lobule is replaced by a mixture of cells having no particular architecture. The chief cells are mononuclear cells, some of which are lymphocytes, plasma cells and other forms of mononuclears. A number of cells appear to be proliferating from the reticulo-endothelium of the sinuses. An occasional cellular body appears to be a remnant of the liver cell. No intact liver cords are seen. Most of the monocytic infiltration is about the periphery of the lobule. The medium-sized ducts show no change. The main bile duct shows no edema and no leucocytic infiltration.

Comment:

This type of case is difficult to interpret. A more intensive study by gross dissection and serial microscopic section might demonstrate some focus of inflammatory reaction or obstruction in the larger bile ducts or main duct. While no extensive search was made in this case, study of the slides and the gross specimen gives one the impression that the essential change in the liver is located in the periphery of the liver lobules. Monocytic infiltration suggests some form of chronic or subacute inflammatory reaction. Clinically, the entire course extends over a period of only three days but the change appears to be much older than this because of the extensive proliferation of the small biliary capillaries. In Buffalo, New York, Roman studied an epidemic of so-called infectious catarrhal jaundice and performed several autopsies on this type. He felt that this represents another form of acute yellow atrophy of

the liver. The cause of the illness was **not determined.**

R.W.K.

2. ASCITES. CARCINOMA OF OVARY.

Case is white female, 56 years old, admitted to University of Minnesota Hospitals 10-12-33 and discharged 12-22-33 (71 days); readmitted 1-9-34 and discharged 1-27-34 (18 days); readmitted 3-28-34 and expired 3-30-24 (2 days). Total stay - 91 days.

1931 - Nervousness. Soon thereafter, observed weight loss, insomnia, weakness and tachycardia.

4- -33 - Developed severe cold, following which there was extreme exacerbation of symptoms. In addition, had belching, epigastric pain and distress after eating, and poor appetite.

9-13-33 - O.P.D. B.M.R. +60%.
9-16-33 - O.P.D. B.M.R. +61%. Put on Lugol's solution.

10-12-33 - O.P.D. B.M.R. +33%.

Admitted:

Physical examination: shows emaciation, nervousness, apprehension, widening of palpebral fissues, exophthalmos, marked pulsation of vessels of neck; negative lungs, abdomen and extremities (other than noted above). Numerous examinations of heart are recorded. Two examiners observed the presence of a murmur; one stated it to be present over the apex; other, over heart. Numerous other observers do not record the presence of any significant heart changes. Blood pressure (several readings) recorded within normal limit. One rectal examination done and said to be negative; no masses felt. Laboratory: Urine - negative. Blood - Hb. 80%, wbc's 7,150, normal differential. Gastric analysis - free Hcl - maximum 45°, total - 51°. Stool - negative for blood (3 examinations). X-ray of chest - slight lower respiratory infection.

10-14-33 - Sudden onset of marked tachycardia. Medical consultant did not record findings in heart. Medical opinion - tachycardia and tendency toward diarrhea (of recent origin) highly suggestive of thyroid crisis.

10-19-33 - Persistent high pulse rate ranging up to 150. Placed on absolute bed rest, sedatives, high caloric feeding and paraoral fluids.

10-26-33 - Tachycardia persisting, ranged between 130 and 160.

11-4-33 - Condition about same. X-ray treatment over thyroid begun. 130% S.E.D. in 5 treatments given. Electrocardiogram (4) - shows presence of auricular flutter with 2 : 1 block.

11-15-33 - Operation: Total ablation of thyroid gland done in hope that this would relieve the auricular flutter. Dissection of gland done, preserving both laryngeal nerves and parathyroid gland.

12-3-33 - Tachycardia persists. Medical consultation: Patient has an apparent pulsus bigeminus with an apical rate around 150. Electrocardiogram - shows ventricular rate of 150, auricular rate 300, regular rhythm; conclusion - auricular flutter, 2 : 1 block.

12-19-33 - Less nervous. Gaining weight. B.M.R. +13%.

12-22-33 - Discharged. Apical rate 140, regular.

Readmitted

1-9-34 - Interval history: Has gained some strength and weight since discharge. Has been seen in O.P.D. and a persistent auricular flutter has been present. X-ray of gastro-intestinal tract - showed negative stomach and duodenal cap with a diverticulum in the 2nd part of the duodenum, and rays of the gall-bladder showed a pathological gall-bladder. Observed enlargement of abdomen. She states that this began about at the time of discharge and has been definitely increasing since. Gained 12 lbs. Physical examination: Eyes, lungs - negative. Heart - tones clear and regular, systolic murmur at

apex transmitted to base of heart; slight irregularity present; somewhat enlarged to left; blood pressure 160/110; pulse 88; respirations 20. Abdomen - protruding, moderate fullness at flanks, tympany over abdomen with dulness in flanks shifting on movement, no tenderness, palpable inguinal nodes, liver and spleen not palpable, fluid wave present. Rectal and pelvic - not done. Laboratory: Urine - trace of albumen (once). Blood - Hb. 83%, wbc's 7,300. B.M.R. +1%, later +4%. Electrocardiogram - rate 80, regular rhythm; P split in all leads, T_{ii} diphasic, T_{iii} inverted and toxic. Medical consultation: There has been an increase in the size of the abdomen with some anorexia and tendency toward diarrhea. Mild ascites present. Liver and spleen not palpable. This could well be a cirrhosis of the liver as the result of prolonged hyperthyroidism. Surgical note: Patient very much better as far as the heart is concerned; no flutter.

1-24-34 - Paracentesis, 3,350 cc. fluid removed; specific gravity 1.016, albumen 15 gms. per 1000 cc., culture negative, no study of cells.

1-27-34 - Discharged. Clinical diagnosis: Ascites. Possible beginning cirrhosis of liver.

Readmitted

3-28-34 - Ascites has rapidly increased. Patient feels well except for fullness of abdomen and shortness of breath. Physical examination: general condition poor. Heart - not enlarged; soft, loud, blowing systolic murmur, maximum over apex, and heard most over precordium; blood pressure 75/52. Abdomen - shows extreme ascites; 11,700 cc. fluid withdrawn; no masses felt. No rectal or pelvic examinations recorded.

3-30-34 - Had been feeling well. Previous evening, temperature 98.3, pulse 100. 6 A.M. - Having dyspnea; restless; cyanotic. Stimulants given. 7 A.M. - Expired.

Autopsy

Body is 56 year old woman measuring 165 cm. in length and weighing about 150 lbs. Development is of average type and nourishment fair. No demonstrable jaundice. Pupils equal, each measuring 4 mm. in diameter. Collar incision over neck. Small paracentesis wounds in abdomen.

Peritoneal Cavity contains about 2 liters of slightly reddish-amber fluid. After this is evacuated, several tumor implants are seen on the peritoneal surface and 3 or 4 polypoid implants over the sigmoid. There are numerous hard, cystic masses in the omentum and several over the spleen. Spleen is adherent to parietal peritoneum. Peritoneal surface of liver and diaphragm markedly thickened by tumor infiltration.

Pleural Cavities are free of adhesions and contain no excess fluid. Pericardial Sac is completely adherent over entire heart. Adhesions are fairly light and can be broken down with fingers. Adhesions are generalized and not of the web or stringy type.

Heart shows very definite hypertrophy, weighs 540 grams. Musculature, particularly on left side, is thickened. Mitral and aortic valves show old rheumatic endocarditis. Cords of mitral valve are thick and short. Valve itself is thick and there still can be seen small roughenings along valve edge which appear to be sclerotic rheumatic nodules. Aortic valve is involved to a lesser degree. Leaflets are thickened and same type of roughening is seen at valve edge. Musculature shows no infarction, softening or intramural thrombi. Root of Aorta shows no syphilis. There is slight atheromatous change. Coronaries are open on both sides. No thrombi and no very significant degree of sclerosis. Pulmonary arteries are packed solid with an antemortem clot which extends deeply into branches of lung.

Right Lung weighs 475 grams and shows slight atelectasis along margin and some discoloration of base, probably hypostasis. Left Lung weighs 400 grams and a similar process is present in base. No bronchopneumonia, bronchitis or metastatic tumors.

Spleen weighs 200 grams. Capsule is adherent to surrounding tissue. Moderate amount of trabeculation persists.

Liver weighs 1400 grams. Capsule is thickened by tumor infiltration and right lobe is partially adherent to diaphragm. Liver appears somewhat small, cuts with usual amount of resistance and does not show an increase of fibrous tissue. Markings are fairly well retained. Liver substance is somewhat dark. No tumor nodules seen in liver. Grossly, although liver appears small, it does not appear to be cirrhotic.

Gastro-Intestinal Tract: Esophagus and stomach show only postmortem change. No ulcers, tumors or polyps. Duodenum is free of ulcers and polyps. Diverticulae seen on x-ray not found. Small bowel shows occasional small peritoneal implants from tumor. Larger implants, measuring up to 1 cm. in size, are present on sigmoid. No diverticulae or areas of ulceration found.

Pancreas is soft and shows no fibrosis or tumor masses.

Adrenals are imbedded in metastatic tumor tissue. No hemorrhage or tumors.

Capsules of Kidneys strip fairly easily. No infarcts, either old or recent. No evidence of hypertensive type of change on surface. There are a few irregular markings which possibly may be arteriosclerotic. Left ureter is dilated to a diameter of about 1.5 cm. and dilatation extends into left kidney pelvis. There does not appear to be any infection. Mucosa is smooth. Blood vessels are not engorged. Ureter is constricted in left broad ligament which is infiltrated with tumor tissue.

Genital Organs: Each ovary is replaced by a cystic mass. Mass on right side measures about 7 to 8 cm. in widest diameter and is composed of a gelatinous, somewhat brownish, lobulated tumor. On external surface of this, there are 3 or 4 small flat papillae. Tumor on left side is somewhat smaller, measures about 5 cm. and is composed of same gelatinous, brownish tumor structure. Tumors are too soft and gelatinous to be of solid ovarian carcinoma type and probably represent cystadenocarcinoma rather than solid carcinoma. Many cysts contain serous fluid. Tubes are not distorted. Uterus is of small size and endometrium is smooth. No myomas present.

Lymph Nodes: There is a dense plaque of hard tumor infiltration along greater portion of abdominal aorta. This is most marked behind pancreas around origin of mesenteric vessels. Some of iliac nodes appear to be enlarged although they may not be involved by tumor.

Femoral veins are milked upward. On right side, small piece of antemortem clot, measuring about 5 cm., is freed. No clot expressed from opposite side.

Diagnoses:

1. Bilateral cystadenocarcinoma of ovary.
2. Multiple peritoneal implants.
3. Ascites.
4. Old rheumatic endocarditis.
5. Pericarditis, old.
6. Bilateral pulmonary embolism.
7. Left hydronephrosis.
8. Thrombosis of right femoral vein.

Microscopic:

Heart muscle - shows no change. Small blood vessels show no thickening of intima.

Kidneys - Blood vessels show no hypertensive changes. Tubules are intact. There is proliferation of endothelium of glomerular capillaries of terminal type.

Liver - shows no evidence of cirrhosis. There is slight fatty change and cloudy swelling.

Adrenals - One is infiltrated with tumor.

Spleen - shows no significant change.

Pancreas - Lungs - Ovaries - Uterus - show no significant change.

Diaphragm - shows infiltration with tumor on serous surface. In many areas, small tumor cells

can be seen projecting into lymph spaces. There is a mild lymphocytic infiltration around these tumor nests. Muscle is not infiltrated.

Tumor is of adenoid cystic type with numerous papillae going into the cystic spaces. Cells of cylindrical type. Most of spaces within tumor are distended with an amorphous secretion.

omment:

This case illustrates a fairly haracteristic picture of this type of varian tumor. The presence of papillae n the external surface immediately ndicates the presence of malignancy. The resence of a bilateral tumor shows the endency of the serous type of cyst to pread or else develop in the opposite vary. The histological picture of the iaphragm is interesting. A large number f the lymph spaces are plugged with the umor cells (cause of ascites?). In some ases of ovarian tumors, particularly he pseudomucinous type, the plugging of he lymphatics may be due to the heavy ecretion within the lymph spaces.

There are two interesting problems rought up by this case. The first is he part played by pulmonary emboli as a actor in death in various cardiac cases. his patient died suddenly and on the asis of a marked disturbance of rhythm hich had been previously studied in the ther admissions the possibility of coroary occlusion or death due to disurbance of conduction could have been ntertained. In this case, there is no uestion that death was due to massive ulmonary embolism. Similar situations ave frequently come up in which there ave been various degrees of pulmonary mbolism in patients with heart disease. everal times the clinical diagnosis as to cause of death was coronary obstruction. ometimes the degree of pulmonary embolism has been only moderate. The impression from autopsies has been that a considerable number of sudden deaths in cardiac patients has been due to pulmonary embolism, the source of the emboli being from peripheral vessels. It appears legitimate to assume that in the presence of a severely damaged heart the degree of pulmonary embolism need not be as extensive as that seen in the classical postoperative types in order to produce a fatal outcome.

The second feature which is well illustrated by this case, is that unsuspected findings are rarely found at autopsy when all the important clinical examinations have been done. In our experience, errors in diagnosis practically always are due to failure of examination rather than interpretation of the findings made.

R.W.K.

IV. ANNOUNCEMENTS

1. SURGERY SEMINAR

Dr. Charles E. Rea will speak on "Recent Studies in Bowel Obstruction" at the Surgery Seminar today, at 4:30, in Todd Amphitheater. Anyone interested is cordially invited.

2. RADIOLOGY SEMINAR

"Newer Methods in the X-ray Examination of the Colon" by Dr. Harry Weber of the Mayo Clinic, University Hospital X-ray Department, Room M-515, Friday, April 27 at 5 P.M.

Dr. Weber will also talk at 3 P.M. Friday in the Eustis Amphitheater on "X-ray Diagnosis of Diseases of the Colon." Anyone interested is welcome.