Staff Meeting Bulletin
Hospitals of the » » »
University of Minnesota

Generalized Amyloidosis

Volume XIII | Friddoy, October 10, 1941 | Number 2

STAFF MEETING BULLETIN
HOSPITALS OF THE . . .
UNIVERSITY OF MINNESOTA

Volume XIII | Friday, October 10, 1941 | Number 2

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Published for the General Staff Meeting each week during the school year, October to June, inclusive.

Financed by the Citizens Aid Society, Alumni and Friends

William A. O'Brien, M.D.

ROBERT CHILCOTE SCHENCK
(1895-1941)

Bob Schenck died Friday, October 3, during the noon hour. Just before staff meeting he complained of not feeling very well. He was found in his bed with an open book in his hand. Bob had been sick since June 9. He left September 5, and returned September 16, to stay until the 28th. He was feeling fairly well when he went back to work. Death was due to coronary disease. The funeral was held in Minneapolis, October 6. Burial was in Deerwood.

He was born October 27, 1895, in Duluth, Minnesota. His earlier years were spent in Grand Rapids where he became well known to many former Grand Rapids residents, now in the Twin Cities. He attended Hamline University, but because of his musical ability he was employed by the Orpheum Theatre in the double-capacity of musician and public relations expert.

He joined the University of Minnesota staff in 1931 in the credit division. He has been steward of the University of Minnesota Hospitals since 1934. Bob was well liked by everyone. His untimely death removes from our ranks an efficient staff member and a good friend. He had many hobbies and interests which created for him a wide circle of acquaintances. He will be missed by all. We are a better organization because of the time he spent with us.

I. LAST WEEK

Date: October 3, 1941

Place: Recreation Room
Powell Hall

Time: 12:15 to 1:15 P.M.

Program: "Annual Report 1940-1941"
Ray M. Amberg

Discussion
Ray M. Amberg
L. G. Rigler
Milton Levine
K. W. Stenstrom
J. C. McKinley
Irvine McQuarrie

Present: 114

Gertrude Gunn,
Record Librarian

- - - -

II. CORRECTIONS

P. 5 Add - Surgery Staff:
Bernstein, Wm. C.
P. 6 Change Downing, Arthur H., University of Minnesota to University of Chicago.
P. 8 Add Residence - Pittsford, N. Y., Clarence Davis.
P. 9 Add Residence - Billings, Mont., Robert Movius.
P.26 Change Tamarack (tree) to Tamarisk.

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III. MEETINGS

1. SPECIAL LECTURE

Dr. Chester S. Keefer, Wade Professor of Medicine, Boston University School of Medicine.

"The Epidemiology and Treatment of Hemolytic Streptococcal Infections"

Saturday, Oct. 11 at 10:30 a.m.
Medical Science Amphitheatre.

Wesley W. Spink.

2. SURGICAL CLINIC

Roscoe R. Graham, University of Toronto. Todd Amphitheatre, 11:00 a.m., Tuesday, October 14, 1941.

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3. MINNESOTA MEDICAL ALUMNI ASSOCIATION

announces its

ANNUAL CLINICAL PROGRAM AND MEETING

Friday, October 31, 1941, (the day before Homecoming). To be held in the University Hospitals as in previous years

Wesley W. Spink: "Sulfonamide Therapy"
Larry Boies: "Hearing Loss in Childhood"
*Lloyd H. Ziegler, Milwaukee: "Reactions of Psychotic Individuals to Surgery"
*Harry Christianson: "Ano-rectal Diseases"
Miland Knapp: "Physical Therapy of Fractures"
*Erling Platou: "Human Serum Therapy"

A short business meeting will immediately follow the Clinical Program.

Luncheon will be served in the Coffman Memorial Union at 12:30 p.m. This luncheon meeting will be addressed by Dr. Wallace H. Cole on his "Recent Experiences in England."

All Alumni of the University of Minnesota Medical School and other interested physicians are invited to attend.

*Member of 1921 class.

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4. CENTER FOR CONTINUATION STUDY

Medicine

Radiology of Chest - November 3-5
Sulfonamide Therapy - November 10-12
Urology - - - November 10-12
Diseases of Infancy and Childhood - December 15-20

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Hospital

Medical Record Library Service - -	October 6-8
Medical Technology -	October 20-22
Public Health Nursing -	November 6-8
Occupational Therapy -	November 17-19
Medical Social Service-	November 24-26

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5. NINTH ANNUAL ASSEMBLY

Omaha, Mid-West Clinical Society, October 27-31, 1941.

Representatives:

Medical School
John Leyland McKelvey
Leo G. Rigler

Mayo Foundation
Albert M. Snell
Byrl Raymond Kirklin

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6. INTERNATIONAL MEDICAL ASSEMBLY

Minneapolis, October 13-17, 1941.

Representatives:

Medical School	Mayo Foundation
Horace Newhart	Paul A. O'Leary
Robert G. Green	Charles W. Mayo
C. Donald Creevy	William F. Braasch
J. C. McKinley	Alfred W. Adson
John L. McKelvey	Howard K. Gray
E. T. Bell	Claude F. Dixon
O. H. Wangensteen	Walter C. Alvarez
Leo G. Rigler	Waltman Walters
N. Logan Leven	
Irvine McQuarrie	
Hobart A. Reimann	
Maurice B. Visscher	

Senior Medical Students and Nurses admitted free Friday (only), Oct. 17; Interns and Graduate Nurses $1.00 (all sessions). All others $5.00, except guests and life members.

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IV. OFFICES

THE DULUTH CLINIC
announces the affiliation of

ROBERT H. LaBREE, M.D.

in the
Department of General Surgery

Office: 204 W. 2nd St., Melrose 1126 — Residence Hemlock 2709

- - - -

ALBERT F. HAYES, M.D.

Announces the opening of Offices at
519 Lowry Medical Arts Building
St. Paul, Minn.

Practice limited to Obstetrics and Gynecology — Telephone: CE. 3414

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V. MORE PROMOTIONS

Charles E. McLennan, assistant professor of obstetrics and gynecology.

John M. Adams, clinical assistant professor of pediatrics

Frank C. Andrus and George K. Higgins, clinical assistant professor of pathology

Ragnvald S. Ylvisaker, Joseph F. Borg and Johannes K. Moen, clinical assistant professor of medicine

John J. Hochfilzer, clinical assistant professor of otolaryngology

Ernest L. Meland, clinical assistant professor of urology

Russell W. Morse and Harold O. Peterson, clinical assistant professor of radiology

Wallace F. Ritchie, clinical assistant professor of surgery.

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VI. GENERALIZED AMYLOIDOSIS

J. B. Arey and F. W. Hoffbauer

Secondary amyloidosis, associated with an etiologic suppurative focus, is not uncommon. In such cases the deposits of amyloid are usually situated in specific locations, notably the liver, spleen, kidneys and adrenals. Primary amyloidosis, not associated with a chronic suppurative process, is of rather rare occurrence, and in such instances the amyloid deposits are said to involve mesodermal tissue, the cardiovascular system, gastro-intestinal tract, smooth and striated muscle and lymph nodes (Reimann, Koucky and Eklund). The present case of secondary amyloidosis is presented because of the atypical location of the amyloid deposits with a resultant similarity to primary amyloidosis.

A 35 year old white woman was first seen in the Outpatient Department of the University of Minnesota Hospitals in May, 1936. At that time she gave complaints referable to osteomyelitis present since 1920. She had been in Gillette Hospital for $5\frac{1}{2}$ years, (1921-1926), because of infection of the femur. Since her discharge she had been getting along quite well until 1933, when she began to suffer from pain across the sacrum, especially on the left side. At that time (1936) urinalysis was negative on repeated occasions. Hemoglobin 90%, leucocyte count 3,920, normal differential. The clinical diagnoses were old pyogenic arthritis of left hip with bony ankylosis and extreme deformity; old osteomyelitis of right fifth rib with deformity but no evidence of an active process; there was likewise no evidence of an active process in the left hip. An osteotomy was performed in an attempt to correct the deformity, and following a long convalescence she was sent home.

She was readmitted in November, 1937, complaining of swelling, pain and tenderness in the right groin and pain in right lumbar region. Previous history of osteomyelitis of 17 years duration which had at times involved the left hip, right leg, right ileum and right femur. She had apparently obtained good results from the osteotomy (1936), her present complaint beginning about 10 weeks prior to this admission.

Examination revealed fluctuant areas in right groin with surrounding erythema and induration. Tenderness in right lumbar region but no other findings. Left hip was ankylosed. X-ray examination of involved areas was negative. Hemoglobin 73%, leucocyte count 14,000, normal differential. Urinalysis showed an occasional white cell. Smear and culture of the drainage from right groin failed to reveal tubercle bacilli. In December, 1937, 10 cc. of purulent material was aspirated from the area in right groin. Sinus tract in this region eventually developed granulation tissue about it. Patient was discharged April, 1938.

Readmitted August, 1938, again complaining of pain, swelling and tenderness over left hip. Onset of this illness was about 2 weeks prior to admission. Physical examination now revealed extreme limitation of motion of pelvis, any movement of pelvis producing excruciating pain. No definite evidence of abscess formation. X-ray examination no definite evidence of osteomyelitis, in general the findings were as previously described. In an attempt at immobilization a hip spica cast was applied At time of discharge it was noted that an abscess had ruptured over the previous site of pain, swelling and increased temperature about left hip. There was no evidence of fecal contamination of drainage, but the patient stated that gas passed via sinus rather than through rectum.

Readmitted shortly afterward because of drainage of gas and fecal material through the sinus. X-ray at that time showed communication of many sinuses with rectum.

January, 1939, the patient was sent to Urology Clinic because of pain in left flank. She stated that since January 2, 1939 she had had almost constant pain in left kidney region. Pain radiated into left lower quadrant, and nocturia (3-4) as well as frequency had been noted during this period. Physical examination now revealed a palpable but

non-tender left kidney. There was some tenderness on deep pressure in left lower quadrant. Urine showed 3+ albumin (2-3+ albumin had been first noted in urine in May, 1938 and had been present on repeated occasions at that time). A few red and many white cells were present in urine. Hemoglobin 80%, leucocyte count 9,100. Indigo carmine returned in normal time and in normal concentration from each kidney. In view of the findings, the patient was considered to have pyelonephritis.

Readmitted April, 1939, at which time a colostomy was performed because of fecal fistula in left groin. Hemoglobin 72%, the urine contained 2+ albumin as well as numerous white blood cells. E. Coli were cultured from the urine, but no tubercle bacilli could be found.

Readmitted August, 1939, as colostomy had now retracted and fecal material escaped from rectum, colostomy and sinus tract. Colostomy was repaired and she was given intensive course of sulphanilamide therapy in an attempt to clear up the pyelonephritis. Following this the urine was almost entirely free of pus cells and cultures were sterile. 2-3+ albumin persisted in urine. Showed a total return of 20% phthalein.

Readmitted November, 1939 because of poor kidney function, the latter having been first noted in Outpatient Department. Except for colostomy and old osteomyelitis there were no subjective complaints. Specific gravity of urine varied from 1006 to 1009, 1-2+ albumin was present and white and red cells were found in the sediment. Hemoglobin 40 to 50%, but by means of a transfusion was raised to 85% just prior to discharge. Blood urea nitrogen varied from 24 to 35 mgs. %, creatinine from 3.4 to 4 mgs.%. The total proteins of the blood were somewhat reduced. Phthalein test gave a value of 6% return in 2 hours. She was unable to concentrate urine higher than a specific gravity of 1002. 56% of Congo Red was retained in the blood 1 hour after injection. Blood pressure was normal. Except for transfusions no active therapy was instituted and patient was discharged with a diagnosis of renal amyloidosis following chronic osteomyelitis.

October 1940 hemoglobin 43%, red blood count 2,000,000, blood urea nitrogen 45 mgs.%, creatinine 5.8 mgs.% November, 1940 hemoglobin 65%, blood urea nitrogen had now risen to 74 mgs.% and creatinine to 6.7 mgs.%. Urinalysis still showed albumin, although on several occasions only 1+ albumin was present.

April, 1941 the patient's blood urea nitrogen was 65.4 mgs.%, creatinine 7.7 mgs. %. By June, 1941 only 1 plus albumin was present in urine. On repeated occasions after this either no albumin or only a trace was present.

Admitted for last time July 6, 1941. At that time she was still complaining of frequent pains in back and down the left leg and there was still a draining sinus in left buttock. She further stated that she had frequent crampy abdominal pain, usually occurring after meals, and at times associated with vomiting of undigested food. Colostomy had not been functioning as well as previously, so that there was some discharge from rectum. She also noted considerable weakness and rather marked swelling of ankles.

Physical Examination

Revealed soft systolic murmur over entire precordium. Heart was not enlarged. Blood pressure was 150/88 (no previous record of hypertension had been noted). Liver and spleen not palpable.

July 8, 1941 her phthalein was again 6% in 2 hours, blood urea nitrogen had fallen from 74% (Jan. 1941) to 42%. Creatinine 6.5 mgs.% and uric acid 3.1. Urinary concentration test again showed maximum concentration of 1012. Congo Red test showed about 10% more of dye removed from blood than in 1939, only 47% of the dye now remaining in blood at end of one hour, as contrasted with 56% in 1939. Plasma proteins continued at low level, reaching 4.7 on July 25, 1941, with 2.6 albumin and 1.9 globulin. August 15, 1941 the colostomy was revised under local anesthesia. In spite of repeated transfusions her course was stadily downhill, blood urea nitrogen rose terminally to 124 mgs.%, creatinine to 12.8 mgs.%. August 2[illegible], 1941, CO_2 combining power was 15. Blood phosphorus

14 mgs.%. Temperature remained within normal limits, she became irrational for a short time prior to death and died on August 26, 1941, approximately 21 years after the onset of osteomyelitis.

SUMMARY

	Female	Age 35	Expired Aug. 26, 1941
1920		Osteomyelitis left femur	
1921-1926		Gillette Hospital Multiple foci of osteomyelitis, with draining sinuses.	
1929		University of Minnesota Hospitals Typhoid fever	
1936		University of Minnesota Hospitals Out-Patient Department Ankylosis left hip	Urine - negative Blood pressure - normal
1937	Feb.	Osteotomy	
1938	Jan.	Abscess right groin	Urine - trace albumin Specific gravity - 1012 Blood pressure - 116/78
	May		Urine - 3+ albumin Granular casts White cells
	Aug.	Fistula into sigmoid	Urine - Albuminuria Pyuria Congo Red - 81%
1939	April	Colostomy	Urine - 2+ albumin Pus 2+, clumps Blood pressure - 110/78 Intravenous pyelogram - Good function
	Aug.	Pyelonephritis (?)	Urine - 3+ albumin Pus - occasional Phthalein - 19% Blood - Hemoglobin 57% Erythrocyte - 2,700,000
	Nov.	Urology	Urine - as above P.S.P. - 3% Congo Red - 56% Blood urea nitrogen - 42mgs.

Summary (Cont.)

1940	Feb.	Hemoglobin - 50% Blood urea nitrogen - 32 mgs.% Blood pressure - 112/72
		32 mgs.% Blood pressure - 112/72
	Oct.	Hemoglobin - 43% Blood urea nitrogen - 74 mgs.% Creatinine - 6.7 mgs.%
1941	Jan.	Blood pressure - 140/86
	July	Blood pressure - 150/88 Phthalein - 2% Congo Red - 47% Urine - C+O trace albumin
	Aug.	Blood urea nitrogen - 124 mgs.% CO_2 - 13

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Autopsy

The body is that of a poorly nourished white woman, numerous scars of previous surgical procedures are present over pelvis, lower extremities and right thorax. No foci of suppuration visible and no sinus tracts are present. Colostomy in left lower quadrant.

Peritoneal cavity contains no excess of fluid; there is no leakage about the edges of the colostomy. Each pleural cavity contains 200 cc. of slightly cloudy fluid. The pericardial sac has a small amount of slightly blood tinged fluid, its surfaces are smooth. There are a few petechial hemorrhages in epicardium.

Heart weighs 290 grams. The chambers are of normal size. Scattered throughout the ventricular and auricular mural endocardium, as well as on the valvular endocardium are innumerable, very minute, pinpoint pink spots. These are readily visible after fixation, barely discernible in the fresh state. On palpation they are barely discernible, the surfaces involved by them being very finely granular to the touch. These punctate areas are far more numerous on the right side of the heart, being much less extensive on the aortic and mitral valves and mural endocardium of the left heart than on the endocardium of the right side, pulmonary and tricuspid valves. On the valves the punctate areas are not limited in distribution but are scattered diffusely throughout leaflets. The valves themselves do not appear thickened, there is no separation of any of the commissures, no fusion of the commissures or chorda tendinae. A few similar punctate pink areas are present in the mural endocardium covering the papillary muscles of the right and left ventricles. On section of the myocardium there is a suggestion of very fine, web-like pink lines running in the direction of the muscle bundles. The coronary ostia are patent, the coronary arteries thin walled and patent throughout. The arch of the aorta is smooth.

Right lung weighs 350 grams, the left 480. A small amount of fibrin is present on the surface of both lungs. Both lungs are nodular to palpation, the nodules being up to 1-2 cm. in diameter. On section these nodules, scattered throughout both lungs, show congestion, edema and small areas of consolidation. and small amounts of pus could be expressed from them. The intervening parenchyma, which comprises the major part both lungs show nothing of note

except for slight edema on the left. The pulmonary arteries and bronchi are free of change.

Spleen weighs 120 grams. The capsule is wrinkled and studded with innumerable punctate, pink spots. An occasional whitish firm nodule about 1 mm. in diameter is present on the surface. On section the parenchyma is only moderately firm and has a mottled appearance, darker red areas up to 2 cm. in diameter alternating with more pale parenchyma. Scattered throughout the substance are innumerable punctate pink and white areas reaching as much as .5 mm. in diameter. A few white firm nodules resembling miliary tubercles are also visible.

Liver weighs 1300 grams. The capsule is smooth, but immediately beneath the capsule is a fine network of pink staining vessels. On section the organ cuts with normal resistance, there is no darkening of the centers of the lobules and no increase of portal connective tissue. Scattered throughout the parenchyma are occasional punctate pink areas and fine threadlike pink staining vessels, apparently distributed in the portal spaces. A few vessels up to 2 mm. in diameter have distinctly pink walls. The amount of pink staining material in the liver is slight and is apparently all in relation to vessels.

Esophagus, stomach, small and large intestine down to the colostomy are free from demonstrable change. The proximal limb of the colostomy is patent, the distal limb distinctly stenotic. There is abundant dense fibrous tissue along either wall of the pelvis, more marked on the left. The rectum is very firmly bound by this dense fibrous tissue to the posterior wall of the pelvis, this causing the rectum to make a very sharp angulation at the most posterior and superior aspect of the pelvis. Above this point the rectum is not adherent to the wall of pelvis. At the point of angulation the lumen of the rectum is distinctly narrowed and remains thus for a distance of about 5 cm.; however, a small amount of firm fecal material is present in the lumen of the rectum, immediately proximal to the anal canal. The mucosa of the entire large bowel is free of change, no fistulae are present.

Pancreas weighs 60 grams. Both the surface and deeper parts of the parenchyma are distinctly pink except for a few normal appearing lobules at the tail of the organ. Lobular markings of the organ are retained throughout. Adrenals show no gross evidence of disease.

Right kidney weighs 65 grams, the left 95 grams. The capsules strip easily revealing very finely granular surfaces studded with innumerable faintly discernible pink spots. A few of the fine granular markings are whitish, there is an abscess 2 mm. in diameter on surface of left kidney. On section there is no dilation of either pelvis or ureter. The cortices are markedly narrowed, averaging 1-2 mm. in thickness, and each kidney contains a few smooth walled cysts up to 15 mm. in diameter, involving both cortical and medullary regions. Scattered throughout cortices of both kidneys are innumerable punctate pink areas; only a very few similar points are visible in medullary regions.

Bladder is firmly adherent to pelvic wall by means of the aforementioned pelvic fibrous tissue. Its lumen contains a small amount of thick greenish purulent material, its mucosa is free from change. The uterus, tubes and ovaries are free from change except for slight pinkish discoloration of a portion of fundus of uterus.

Thyroid is normal in size, has a distinctly pink color and uniform appearance throughout. One definite parathyroid gland 12x7x2 mm. and two other questionable parathyroids 6x5x2 and 10x9x3 mm are present. The larynx and trachea show nothing of note.

There are several hyperplastic periaortic lymph nodes, a caseous hilar node, but no other lymphadenopathy. There is hard, irregular callus formation along the pleural surfaces of the right 4, 5, 6 and 7 ribs. Upon probing, portions of the left ileum are hard and irregular, both anteriorly beneath the anterior superior iliac spine and posteriorly in the region of posterior part of pelvis. No suppuration is demonstrable. Red mar-

row in the lumbar vertebrae.

The head is not examined.

The kidneys, pancreas and thyroid grossly give strongly positive reactions for amyloid with iodine and sulfuric acid; the spleen and myocardium react with slightly less intensity, the liver reacts only within the vessel walls, and portions of the esophagus and sterno-hyoid muscles fail to give any reaction.

Microscopic Examination

Liver: The hepatic arteries are markedly thickened, the normal arterial wall being completely replaced by hyaline, pink staining acellular material. The amyloid deposits appear to arise in the media, as shown by larger vessels in which the media is largely replaced by such deposits, yet the intima remains intact. Because of marked thickening of the arteries the portal vein radicles within the portal spaces are largely overshadowed, rendering their recognition difficult or impossible. No distinct involvement of either portal or hepatic veins could be made out.

Spleen:

Extensive deposits of pink, acellular hyaline material in media of the arteries. For the most part the deposits are in and about the Malphigian corpuscles, with complete replacement of the latter. A few small amyloid deposits are present between the sinuses.

Kidney:

No normal glomeruli remain, the majority being markedly enlarged and completely replaced by amyloid. In a few enlarged, only partially replaced glomeruli a capillary circulation is still present, and here the amyloid can be seen deposited immediately under the basement membrane. A large number of glomeruli appear to have completely disappeared.

In addition to the glomerular deposits, amyloid is deposited about a number of tubules beneath their basement membranes, with loss of the epithelium and resultant tubular amyloid deposits. For the most part, however, such deposits extend diffusely into the interstitial tissues, leaving no clue as to their original site of deposition.

Amyloid is also deposited in the media of the arterioles, small and medium sized arteries, from which point it extends inward to involve the intima and outward diffusely into the adjacent interstitial tissue of the kidney. Thus the major part of the amyloid distributes diffusely throughout the interstitial tissue of the medulla, without any longer bearing any demonstrable relation to the tubules or arteries. In the medulla the tubules are markedly dilated, while in the cortex, where the deposits are almost limited to the glomeruli and arteries, the tubules are very atrophic.

Adrenal:

Medial deposits of amyloid in the peri-adrenal arteries. Formation of "amyloid rings" in the peri-adrenal fat. Extensive amyloid deposits in the muscularis of the central vein, with complete replacement of the wall in some areas. A few deposits of amyloid are present in the zona glomerulosa and zona fasciculata, with resultant disappearance of cortical cells in these areas; however, only a very minimal part of the cortex was replaced by amyloid.

Thyroid:

Disappearance of majority of acini, their site being filled by dense deposits of amyloid involving primarily the interacinar connective tissue. The remaining acini are for the most part dilated, a few are partly filled with colloid. The remaining acinar epithelium is remarkably thin and atrophic.

Pancreas:

Extensive replacement of parenchyma by amyloid. Except for involvement of arteries and islands of Langerhans the amyloid appears to arise in interstitial tissue, so that small islands of acinar tissue are completely isolated from one another by dense deposits of amyloid. The lobular pattern of the pancreas is, however, retained, the deposits of amyloid apparently not extending from one lobule into another. Several relatively small islands

are still remaining, others are partly or completely replaced by amyloid deposited immediately outside the walls of the capillaries.

Stomach:

Localized accumulation of bacteria and polymorphonuclear leukocytes in the serosa. Sections from both the antrum and corpus show considerable deposits of amyloid within the mucosa between the glands. Some deposits appear to originate immediately outside the basement membrane of the gland; as the deposits increase the epithelium atrophies and finally disappears, leaving tubular amyloid deposits. These in turn may extend more diffusely in the interstitial tissue between the glands. Most of the amyloid bears no definite relation to the glands. Although there is some disappearance of glands the majority are intact and appear normal. There is no involvement of layers other than the mucosa.

Small Intestine:

Extensive amyloid deposits in the submucosal arteries. A few small deposits of amyloid in the interstitial tissue of the mucosa.

Gall Bladder:

Rather extensive deposits of amyloid in the interstitial tissue of the mucosa.

Lungs:

Precipitated serum and polymorphonuclear leukocytes in a number of alveoli. A few alveoli contain fibrin plugs, which in some areas are arranged as acellular pink fibrillar bands lining the alveolar walls. Numerous organisms are present throughout the section. The pulmonary arteries are free from change.

Tricuspid valve:

Loose subintimal amyloid deposits with a sharp inner border and without ulceration of the endothelium. The deposits occupy about one half the thickness of the valve.

Myocardium:

Extensive replacement of muscle fibers by pale, acellular, faintly fibrillar deposits. The deposits here do not take as eosinophilic a stain as elsewhere. Here these deposits can be seen actually replacing the muscle fibers, the latter running directly into the acellular, faintly fibrillar amyloid deposits without any sharp transition between the two. At one point there is a focal accumulation of bacteria with a surrounding polymorphonuclear leukocytic infiltrate.

Uterus and Ovary:

Amyloid deposits limited to walls of arteries, and far more extensive in the arteries of ovary than in those of uterus.

Pelvic Fibrous Tissue:

Dense collagenous tissue with a few foci of lymphocytes.

Hilar Lymph Node:

Calcified caseous tubercle with a dense wall of hyaline connective tissue. No deposit of amyloid.

Skin:

No significant change. No deposits of amyloid. Sections of liver, spleen, kidney, thyroid, pancreas and heart stained with methyl violet give positive reactions for amyloid. A section of the myocardium stained with Congo Red is positive for amyloid.

Discussion

Reimann, Koucky and Eklund and Koletsky and Stecher classify amyloid disease in four groups:

1. Primary amyloidosis
2. Secondary amyloidosis
3. Tumor forming amyloidosis
4. Amyloidosis associated with multiple myeloma.

Although this appears to be the most satisfactory classification which can be made in the present state of our knowledge, this is admittedly not entirely satisfactory, as overlapping cases have been reported by several authors. The following, however, are given by the above authors as characteristics of the four groups:

1. Primary amyloidosis
   a. Absence of an etiological disease.
   b. No involvement of organs or tissues usually involved in secondary type.
   c. Involvement of mesodermal tissue, cardiac-vascular system, gastro-intestinal tract, smooth and striated muscle and lymph nodes.
   d. Atypical staining reactions.
   e. Tendency to nodular deposits.

2. Secondary amyloidosis
   a. Presence of etiological disease.
   b. Deposits in spleen, liver, kidneys and adrenals.
   c. Typical staining reactions.

3. Tumor forming amyloidosis
   a. Usually primary in type.
   b. Tumors in eye, bladder, urethra, pharynx, tongue and especially respiratory tract.

4. Amyloidosis with multiple myeloma
   a. Deposits resemble those of primary type, but large deposits occur in joints and elsewhere.
   b. Occasional small deposits in the blood vessels of heart, spleen, etc.

The present report is another instance in which, although obviously secondary in type, the location of the amyloid deposits in some respects resemble primary amyloidosis. Amyloid deposits are present in the spleen, kidneys and adrenals, but are absent from the hepatic parenchyma. The staining reactions, as tested by iodine and sulfuric acid, Congo Red and methyl violet everywhere conform to the typical staining reactions of secondary amyloidosis. Thus, with the exception of hepatic involvement all the criteria for secondary amyloidosis are fulfilled. In addition, however, there is extensive involvement of the cardiovascular system, involvement of gastro-intestinal tract, gall bladder, pancreas and thyroid. There is no involvement of smooth or striated voluntary muscle or lymph nodes, and there is no tendency to nodular deposits. In the affected areas the deposits primarily involve mesodermal derivatives, i.e., the deposits occur primarily in connective tissues or about blood vessels, with only a secondary loss of epithelium. Here it might be pointed out, in regard to the above classification, that this is true even in typical secondary amyloidosis, the destruction of epithelial tissue merely occurring as a secondary result of amyloid deposits in mesodermal derivatives. The present example can thus best be considered as a case of secondary amyloidosis with an atypical distribution of amyloid deposits, and it well illustrates the inadequacies of the present classification. An improvement on the latter can only be made by a more exact knowledge of the true chemical nature of the various types of amyloid and of the underlying metabolic processes in the formation of such material.

Small deposits of amyloid in the pancreas are not infrequent in secondary amyloidosis, but extensive involvement of the pancreas and thyroid, with a resultant loss of large portions of parenchyma is unusual. Amyloid deposits in the heart have been described by a few authors. Koletsky and Stecher describe a case of primary amyloidosis in which the mitral and aortic valves were distinctly stenotic as a result of amyloid deposits within them. There was also extensive involvement of small arteries, muscles, joints and tendons. Pearson, Rice and Dickens report two cases of primary amyloidosis in negroes. In both there was involvement of the heart, and in one definite amyloid deposits were described in the mitral and tricuspid valves.

From a clinical standpoint the extensive involvement of the pancreas and thyroid is of considerable interest. The involvement of the islands of Langerhans (in the given section) is more extensive than is present in some cases of diabetes mellitus. Yet the extent of destruction of the islands in diabetes mellitus does not of necessity parallel the severity of the diabetes, so that a purely pathological diagnosis of diabetes mellitus is difficult or impossible. Glycosuria was never present in this case, but this might be explained on the basis of the renal damage resulting from amyloid deposits. [illegible]

values are recorded, there never having been any symptoms of disturbed carbohydrate metabolism.

In the present instance the possibility exists that destruction of the thyroid might counteract, to a certain extent, the diabetogenic effect of island destruction. Certainly the adrenal involvement is not sufficient to be anti-diabetogenic. Involvement of the pituitary might also explain the absence of diabetes as in the Houssay dog. In the present case the hypophysis was not examined.

Summary

An example of generalized amyloidosis following chronic osteomyelitis is reported. It is of particular interest because of the extensive involvement of organs and tissues not usually involved in secondary amyloidosis. It illustrates the fact that an entirely satisfactory classification of amyloid disease cannot be made at this time. The relation of the amyloid deposits in the various endocrine organs to diabetes mellitus is discussed.

References

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VII. GOSSIP

Each year an increasing number of physicians and institutions have asked to have the Bulletin sent to them. This year, because of a decrease in available funds, it was necessary to make a charge for subscriptions. (In other years money for postage was sent in advance, but this has now been discontinued.) We notified our old friends and staff members with offices in the Twin Cities of our problem. The following list represents the response up to Thursday noon, October 9. We are deeply grateful to these good friends for their continued support and cooperation. We take this means to thank them for their contribution. From time to time additions to this list will be published.

Of special interest is the group in Missoula, Montana, where former Minnesotans and friends of the institution gather each week at luncheon to review a Staff Meeting Bulletin. The group is headed by M. B. Hesdorffer, Director of the Student Health Service, University of Montana. Anyone interested in this plan may write to Dr. Hesdorffer for details. Another supporter on the group basis is F. P. McNamara of Dubuque, Iowa. He has 10 physicians on his list who receive the bulletin. He is better known as "100-bed" McNamara because he has found so many different phases of staff education and service which can be carried on in an institution of 100 beds.

Arizona
Farness, O. J., Tucson

Arkansas
Jones, I. Fulton, Fort Smith
Walsh, W. V., North Little Rock

California
Rabwin, Marcus H., Beverly Hills

Los Angeles
Eshman, Louis
Larson, E. Eric
Nathanson, M. H.
Newman, Ben A.
Tiber, Leon Julius

California,(Cont.)
Jensen, Herman H., Oakland.
Burkland, Carl E., Sacramento

District of Columbia

Washington
Kerlan, Irvin
Lind, Carl John, Jr.
Moehring, Henry G.
Wentink, Elaine A.

Florida
Berman, Theodore, Miami Beach

Illinois
Schilla, Lt. Frederick W., Camp Grant
Papermaster, Theodore, Chicago
Canfield, Burt J., Rockford

Indiana
Bullard, Mattie, Gary
Sagel, Jacob, Gary

South Bend
Ericksen, L. G.
Holdeman, Richard
Langenbahn, C. J.

Iowa
McKean, Frank F., Allison
Fisk, Charlotte, Des Moines
McNamara, F. P., Dubuque
Glesne, Otto N., Fort Dodge
Smiley, Ralph, Mason City
Buirge, Raymond, New Hampton

Kansas
Nelson, Barrett A., Manhattan
Zagaria, James F., Topeka
Kiser, Willard, Wichita

Massachusetts
Bartels, Elmer C., Boston

Michigan
Hubly, James, Battle Creek
Brines, Osborne A., Detroit
Quigley, William G., Detroit
Llewellyn, M. B., Pontiac
Gariepy, Bernard F., Royal Oak

Minnesota
Naeve Hospital, Albert Lea
Vandersluis, Charles, Bemidji
St. Francis Hospital, Breckenridge
Rydburg, Wayne C., Brooten

Minnesota (Cont.)

Minnesota Colony for Epileptics, Cambridge
Butler, J. K., Carlton
Coombs, C. H., Cass Lake
Skaug, H. M., Chatfield
Hauge, M. I., Clarkfield
Thompson, Arthur, Cokato
The Northwestern Clinic, Crookston
Miners, G. A., Deer Rivers

Duluth
- Braverman, N. J.
- Hirschboeck, F. J.
- Tuohy, Edward L.
- Sister M. Patricia, O.S.B.

Stuurmans, S. H., Erskine
Seifert, M. H., Excelsior
Emond, Albert J., Farmington
Smith, Graham, Fulda
Hastings State Hospital, Hastings
Adams, Bert, Hibbing
Scholpp, O. W., Hutchinson
Friedell, George, Ivanhoe
Wadd, C. T., Janesville
Lohmann, John, Jasper
Sonnesyn, N. N., LeSueur
Mulrooney, Raymond, Litchfield
Hanover, R. D., Littlefork
Boysen, Herbert, Madelia
Mickelson, John, Mankato
Troost, Bradley, Mankato

Minneapolis
- Allison, R. G.
- Anderson, Ernest R.
- Arling, Leonard
- Arnold, D. O.
- Beard, Archie H.
- Bedford, E. W.
- Blumenthal, J. S.
- Buehler, Martin
- Buzzelle, Leonard K.
- Campbell, Orwood J.
- Cohen, Joseph T.
- Drake, Charles R.
- Dunn, George R.
- Ehrenberg, Claude J.
- Eitel Hospital
- Fansler, W. A.
- Hanson, Malcolm B.
- Hauge, E. M.
- Haugen, John A.
- Hayes, J. M.
- Huenekens, E. J.
- Hultkrans, Joel
- Hutchinson, C. J.
- Johnson, Reuben A.
- Kinsella, Thomas J.
- Lavake, Rae and Swanson, Roy
- Lippman, E. S.
- Lowry, Thomas
- Maxeiner, Stanley R.
- McCarthy, Donald
- McKenney, F. S.
- McKinlay, C. A.
- Meland, Ernest
- Monson, Einer
- Nicollet Clinic
- Nordland, Martin
- Olson, Frederick
- Peppard, Thomas
- Platou, E. I.
- Regnier, Edward A.
- Rucker, W. H.
- St. Andrews Hospital
- Simons, Jalmar H.
- Smith, Adam M.
- Strachauer, A. C.
- Sweetser, H. B., Jr.
- Ude, Walter H.

Bergh, George S., Montevideo
Korp, M. W., Moose Lake
Francis, David, Morristown
Seifert, Otto J., New Ulm
Mears, R. F., Northfield
Schaefer, J. F., Owatonna
Stransky, Theodore W., Owatonna
Benjamin, Walter B., Pipestone
Cooney, H. C., Princeton
Tyler, Stanley H., Raymond
Hedin, R. F., Red Wing
Lewis, C. B., St. Cloud
Sister M. Rosaria, St. Cloud

St. Paul
- Birnberg, T. L.
- Calvin, A. M.
- Carroll, William C.
- Colby, Woodward
- Daugherty, L. E.
- Freidman, Louis L.
- Geer, Edward K.
- Gruenhagen, A. P.
- Hanson, Norbert O.
- Harmon, G. E.
- Herrmann, Edgar T.
- Karman, Gordon R.
- Lynch, Francis
- Madden, John F.
- Midway Hospital
- Moren, L. A.
- Mounds Park Hospital

Minnesota (Cont.)

St. Paul (Cont.)
Ouellette, A. J.
Richardson, Harold Edward
Ruhberg, George N.
Schwyzer, Arnold
Stewart, Alexander
Wilson, J. Allen
Sister Regina Clare

Valentine, W. H., Tracy
Johnson, C. P., Tyler
Vadheim, A. L., Tyler
Welton, P. C., Wabasha
Bakke, G. Marie, Wayzata
Branton, A. F., Willmar
Schroeppel, J. E., Winthrop
Schade, Frederick, Worthington

Missouri

Bowers, Captain Warner F., Fort Leonard Wood
Clarke, William, Fort Leonard Wood

Montana
Olson, S. A., Glendive
Attix Clinic, Lewistown

Missoula
Alderson, L. R.
Blegan, Holward M.
Brewer, L. W.
Duffalo, J. A.
Foss, A. R.
Frederickson, Clyde
George, Elmer
Haas, A. T.
Hall, Horace
Hesdorffer, M. B.
Lowe, F. H.
Morrison, Wm. F.
Murphy, E. S.
Nelson, John
Noble, P. C.
Preston, Stephen
Ritchey, J. S.

Nebraska
Bancroft, Paul, Lincoln

Omaha
Bantin, E. W.
Isaacson, Svon
Baker, Charles P.

New Jersey
Miller, Cecil Eugene

New York
Jones, O. P., Buffalo

New York City
Jett-Jackson, C. E.
Manson, M. H.
Youel, Milo A.

North Dakota
Quain and Ramstad Clinic, Bismarck
Beck, Charles, Harvey
Olesky, Elmer, Mott
Gilsdorf, W. H., New England

South Dakota
Merryman, M. P., Rapid City
Radusch, Frieda, Rapid City
Schwartz, E. Robert, Wakonda

Texas
Concklin, C. L., Corpus Christi
Cutler, Haydn, H., Houston
Ershler, Irving, Randolph Field
Mitchell, Mancel T., San Antonio

Utah
Westwood, James B., Provo

Virginia
Landis, Eugene M., Charlottesville

Washington
Richardson, J. P., Ellensburg
Flynn, John E., Everett

Wisconsin
Martin, Ann, Ashland
Hertzog, A. J., Eau Claire
Bayley, W. E., LaCrosse
Doege, Paul F., Marshfield

Milwaukee
Lieberman, Benjamin
Schulz, Irvin
Winters, Kenneth J.

Simenstad, L. O., Osceola
Focke, William J., Poynette
Wright, R. S., Racine
Vickers, E. Smith, Sparta
Weisberg, J. H., Superior

Canada
Alberta
Spackman, E. V., Lethbridge