HEMOPHILUS HEMOLYTICUS ENDOCARDITIS *

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Since the publication in 1923 by Miller and Branch [1] of a case of bacterial endocarditis caused by the hemolytic hemophilic bacillus, a bacterium that is normally non-pathogenic, no new cases of endocarditis caused by this organism have appeared in the literature. Russell and Fildes [2] reported a case of endocarditis caused by the so-called *Bacillus parainfluenzae*, an organism that more closely resembles *Hemophilus hemolyticus* than the ordinary strains of the Pfeifer bacillus. Neither the latter nor the *Bacillus parainfluenzae*, however, is hemolytic.

The hemolytic hemophilic bacillus was first described by Pritchett and Stillman [3] in 1919 in an investigation of the bacteriology of the then pandemic influenza, and was labelled by them "Bacillus X." This microörganism is morphologically and culturally similar to the Pfeifer bacillus, but differs in its ability to hemolyze red blood cells. Stillman and Bourn [4] recovered the bacillus from the sputum of patients suffering from acute influenza and lobar pneumonia and from the throat and saliva of healthy persons.

We quote from Miller and Branch a summary of the characteristics distinguishing this microörganism from the influenza bacillus: (*a*) it causes hemolysis; (*b*) it occurs only as a saprophyte; (*c*) it is less strictly dependent on hemoglobin; (*d*) it has a coarser morphology; and (*e*) it is slightly more difficult to keep in culture. Bergey [5] suggested the name *Hemophilus hemolyticus*, which we shall employ in the present discussion.

We shall proceed to the report of a case of bacterial endocarditis complicated by pregnancy, in which the blood culture yielded *Hemophilus hemolyticus* and also *Streptococcus viridans*. The presence of the former organism was also demonstrated in the microscopic lesions postmortem.

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Report of Case

Clinical history: The patient, F. F., a married female 32 years of age, an office worker, born in Czechoslovakia, was admitted to the Fourth Medical Division of Bellevue Hospital on November 22, 1931 with a complaint of chills, fever and cough of 3 weeks duration. The patient stated that she had enjoyed good health until the onset of the present illness, which was sudden and occurred about the evening of November 1, when she was taken with a severe chill lasting about 4 hours, followed by fever and sweating. She was prostrated the next morning and called her family physician, who treated her at home for 3 weeks. Her symptoms during that time were chills, fever and cough. The patient had had dyspnea on exertion and occasional edema of the ankles for the past 4 years. There was no previous history of rheumatic fever, chorea, scarlet fever or acute tonsillitis. Her last menstrual period she believed was about 3 months before admission.

Physical Examination: The patient was acutely ill, flushed, dyspneic and slightly orthopneic. Petechiae were present in the right conjunctiva and in the mouth. The teeth were carious and the gums showed marked gingivitis. The heart was not enlarged, the rate was rapid (120) and the rhythm regular. There was a soft blowing systolic murmur at the apex that was transmitted to the axilla. This was heard upon admission and thruoghout the course of illness. The chest findings consisted of dullness and bronchial breathing over the right lower lobe and a friction rub over the left lower lobe. The spleen was not palpable. The fingers showed slight clubbing and cyanosis. There was slight pretibial edema. On vaginal examination the uterus was felt to be enlarged to the size of a 4 months' pregnancy.

Laboratory Findings: Temperature 103° F, pulse 120, blood pressure on admission 90/60. The urine contained 2 plus albumin and a few red cells per high power field. Hemoglobin 50 per cent, red cells 3,500,000, color index 0.75, leukocytes 12,000, polymorphnouclears 75 per cent, and lymphocytes 25 per cent. Wasserman negative, blood chemistry essentially normal.

Course of Illness: The temperature was of the septic type — 103–4° F in the evenings with morning remissions to about 100° F. Pulse was always rapid, out of proportion to temperature. Blood pressure determinations remained fairly constant, about 120/80. Repeated urine analyses showed 2 plus albumin and casts. On one occasion red blood cells were noticed in the urine.

On November 24 the spleen was enlarged to percussion and on December 1 it became palpable for the first time. New crops of petechiae were discovered almost daily. A friction rub persisted over the left lower lobe. Numerous moist râles were heard from time to time, particularly over the right chest.

On the evening of December 3 the patient had a severe chill and the temperature rose to 105.4° F. New petechiae were noticed in the left conjunctiva. The patient was unable to move her legs and femoral pulsations were absent. A diagnosis was made of a saddle thrombus at the bifurcation of the aorta. Next morning both legs were cyanosed but warm, the body temperature dropped to 95° F and the patient appeared in a terminal toxic state. She died rather suddenly shortly thereafter, the temperature having meanwhile risen to 98.4° F.

The final clinical diagnosis was subacute bacterial endocarditis, saddle thrombus at bifurcation of aorta, and pregnancy.

Bacteriology

Three blood cultures were taken during life. The first taken on November 24 was negative after 5 days. On November 30 blood was withdrawn and cultured aerobically in dextrose broth, anaerobically in vitamin broth and in plain agar plates. The plates remained sterile for 10 days. After 48 hours spreads from the dextrose broth were stained and examined daily for 10 days. No organisms were obtained in any of the spreads. The anaerobic vitamin broth was examined on the 7th day and smears revealed an occasional small Gram-positive diplococcus. Subcultures were then made on blood-streaked agar plates. Growth appeared on the 4th day as thin, green, non-hemolytic colonies which, when transplanted to vitamin broth, grew in long chains. Blood plates streaked from these broth cultures showed typical colonies of *Streptococcus viridans*.

On December 3 blood was cultured as follows: 5 cc. were placed in each of two vitamin broth flasks and in one dextrose broth flask, and 1 cc. in each of three plain agar plates. The plates yielded no growth after 10 days. Smears of each broth culture were made after 24 hours and showed small Gram-negative coccoid bacilli occurring singly and in pairs. Some of the bacilli stained irregularly, showing slight polar bodies. Cultures in broth at 48–72 hours showed larger, non-mature Gram-negative bacilli.

Subcultures of the broth cultures were made on blood-streaked agar plates, blood-drop dextrose agar, ascitic agar and plain agar plates. No growth was obtained in the two latter. At 48 hours the two former showed several pin-point colonies, each surrounded by a small hemolytic area. After 72 hours the zone of hemolysis became much larger. On blood-drop agar plates the colonies were "influenza-like," minute, round, smooth, transparent and finely granular with a coarse, granular, central heaping. Growth on "chocolate slants" was abundant, gray and moist. No growth of transplants was obtained on plain or ascitic agar slants. Sodium oleate agar containing 1 per cent heated blood showed large, spreading, brown, moist, influenza-like colonies. When they were smeared the organisms appeared larger, were Gram-negative, and occurred in short filaments, pairs and groups.

POSTMORTEM EXAMINATION

Autopsy was performed within 4 hours of the patient's death. The anatomical diagnoses were: pericardial effusion, vegetative endocarditis of mitral valve, focal embolic myocarditis of left ventricle; infarcts of kidney and spleen; thrombosis of both iliac arteries; pregnancy estimated to be about 6 months; a Meckel's diverticulum; petechial hemorrhages in skin, conjunctivae and pericardium.

Autopsy: The body is that of a white female about 35 years of age with a well developed frame in a poor state of nutrition. Reddish purple petechiae are present over the right shoulder, neck and in both conjunctivae and sclerae. The left leg and thigh are deep purple in color. Both legs are slightly edematous. The abdomen is greatly distended. Slight clubbing and cyanosis of digits is observed.

On opening the abdomen the uterus projects from the pelvis to the level of the umbilicus and contains a ballotable fetus.

The chest contains scanty thymic remains, the pleural cavities are dry and the right lung is bound down posteriorly by dense, fibrous adhesions.

Heart: The pericardium contains about 50 cc. of yellow fluid. The heart weighs 375 gm. The wall of the left ventricle appears slightly hypertrophied but the chambers appear normal. Along the line of closure of the aortic leaflet of the mitral valve is a mass of friable vegetations measuring 2 by 1.5 by 1 cm. that are dark red, granular and crumbly in consistence. There are no extensions along the chordae tendineae to the papillary muscles or to the auricular endocardium. The line of closure of the posterior mitral leaflet is thickened and contains some minute vegetations. Some of the chordae tendineae are slightly thickened and shortened. No old or recent aortic, tricuspid or pulmonary valvulitis is present. Coronary orifices are patent. Vessels show no sclerosis. Myocardium is dark reddish brown. Over the anterior surface of the left ventricle near the apex, directly along the course of the anterior descending branch of the coronary artery, is a circumscribed area of pallor and softening without thinning of the wall. There is a similar area over the posterior surface of the left ventricle near the apex along the course of the circumflex branch. These are recent infarctions.

Lungs: The lungs are smooth save for dense fibrous adhesions over the right lower lobe. The bronchi contain a slight amount of

mucopus. Both lungs contain diffusely scattered, subpleural, dark red areas of infarction, presenting a contrast to the pink lung. These are even more noticeable in the parenchyma.

Spleen: The spleen weighs 800 gm. and measures 15 by 9 by 5 cm. Its surface is slate gray with contrasting yellow areas of infarction varying from 1 to 3 cm. in diameter. On section pulp is soft and bright red, with ragged subcapsular infarcts occupying about 50 per cent of the organ.

Liver: The liver is large, yellow and fatty, and weighs 3000 gm.

Kidneys: The kidneys are slightly enlarged. Capsules strip readily exposing a smooth purplish red surface interrupted by depressed, indented, irregular infarcts varying from 1 to 2 cm. in diameter. Cut surfaces are bluish red in color with infarcts cutting deeply into the organs. The kidney substance does not bulge beyond the capsule. The markings are obscured. Pelves appear congested. The glomeruli are not enlarged. No petechiae are present.

Uterus: The uterus contains a well formed fetus 26 cm. in length. Its placenta is intact.

Iliac Arteries: Distal to the bifurcation of the aorta both common iliac arteries are thrombosed as follows: the proximal 2 cm. of the right common iliac is uninvolved; the distal portion contains a firm, yellow-gray thrombus about 2.5 cm. long extending for about 0.5 cm. into the corresponding hypogastric artery. On the left side the clot begins about 0.5 cm. below the bifurcation of the aorta and is about 4 cm. long. The proximal 3 cm. is dark red, the distal cm. yellow and firm, extending for about 0.5 cm. into the left hypogastric artery.

Microscopic Examination

Tissues for microscopic sections were removed from the various organs and fixed in 10 per cent formol and in Zenker's fluid and then embedded in paraffin. Heart sections were removed after the routine manner suggested by Gross and coworkers.[6] These were stained with hematoxylin and eosin, by Giemsa's method, and by Brown and Brenn's picric acid differential method.[7]

Heart: Gram stains of smears from the vegetations reveal large numbers of Gram-negative bacilli arranged in clusters and short

chains. These organisms exhibit bipolar staining and vary considerably in size and length.

The anterior leaflet of the mitral valve is largely replaced by a mass of vegetations in the base of which a remnant of old valve substance can be recognized. This remnant shows no vascularization or evidence of antecedent rheumatic change. The vegetation consists of a crumbly mass of fibrin and leukocytes in the midst of which Giemsa sections disclose the presence of enormous numbers of Gram-negative bacilli. Many are intracellular both in polymorphonuclear leukocytes and in underlying connective tissue macrophages. The deeper zone of the vegetation is composed of cellular granulation tissue.

The posterior mitral leaflet is covered with several minute verrucae that consist of small subendothelial elevations covered with fibrin. The bases of these elevations consist of closely packed fibroblasts. No microörganisms can be found in this leaflet. Between the small verrucae the valve surface is covered with a column of cells forming a palisade usually 2 or 3 rows in thickness. These cells are directed obliquely or at right angles to the valve surface, and are often covered with a thin layer of fibrin. The cells are elongated with large basal nuclei. Within the valve substance appear foci of fibrinoid necrosis. The subendothelial connective tissue is swollen and hyalinized and surrounding these foci are accumulations of round cells. Large numbers of basophilic mast cells lie beneath the endocardium of the left ventricle, which like the valvular endothelium, is often replaced by a cellular palisade. Mast cells also appear throughout the interstitial tissue of the myocardium where, together with polymorphonuclear and mononuclear leukocytes, they surround coronary venules. The vascular endothelium is flattened and beneath it is a row of clearly staining cylindrical cells directed at right angles to the endothelium.

The muscle fibers in the mitral ring are hyalinized. The endothelium of the left auricle appears normal, but the subintimal tissue is infiltrated with fibroblasts, polymorphonuclear cells and histiocytes. No Aschoff bodies are found in the auricular myocardium.

Purulent emboli are seen in the small branches of the left anterior and posterior descending coronary arteries and in small branches to the papillary muscles. These consist of fibrin clumps with necrotic polymorphonuclear cells, but in places contain proliferating fibro-

blasts. The media is destroyed at one side with resultant formation of a mycotic aneurysm. This purulent process undergoes extension into the adjacent myocardium where extensive infarction, muscle degeneration, polymorphonuclear infiltration and scars of granulation tissue are found. Giemsa sections reveal no microörganisms in the infarcted areas.

Lungs: Marantic infarcts occupy large areas of the sections. The smaller bronchi and terminal bronchioles contain plugs of mucopus. The bronchial walls are infiltrated with lymphocytes and plasma cells. Surrounding them are well circumscribed islands of connective tissue densely infiltrated with round cells and containing "glandular structures" lined by cuboidal epithelium.

Liver: The liver sinusoids are packed with every variety of bone marrow cell including all types of granular leukocytes, together with megakaryocytes and large mononuclear cells. The lymphoid elements in the portal spaces are hyperplastic and contain many myeloid cells. There is an astonishing number of immature forms, myelocytes, band forms and metamyelocytes of the neutrophilic and eosinophilic series. These are found in the portal veins where they appear to marginate and invade surrounding sinusoids.

Spleen: The spleen contains wedge-shaped peripheral infarcts. In the splenic arterioles there are granular leukocytes of varying stages of maturity. The lymphoid follicles are hyperplastic. Many leukocytic cells and megakaryocytes of the same type as were present in the liver lie beside the endothelial sinuses of the pulp.

Kidneys: The majority of the glomeruli are intact but in many of them focal embolic lesions are identified by necrosis of one or more tufts. In some instances emboli lie within afferent glomerular arterioles. Some emboli contain bacteria morphologically identical with *Hemophilus hemolyticus*. The capsules of the affected glomeruli are surrounded by zones of polymorphonuclear leukocytes and round cells. A few epithelial crescents are present in Bowman's capsule. There is a moderate degree of tubular degeneration with exudation of albumin into the lumen and occasionally hemorrhage. The vessels appear intact.

Aorta: In sections through each common iliac artery the middle coat is invaded by polymorphonuclear leukocytes separating the smooth muscle fibers. Both the cells and the medial coat are under-

going necrosis. The vasa vasorum contain numerous polymorphonuclear leukocytes in process of margination. Overlying the intima is a huge thrombus which contains no bacteria.

DISCUSSION

So far as can be determined this is the second case of bacterial endocarditis to be reported in which *Hemophilus hemolyticus* has been the cause. There are several features in this case that call for comment and it is proposed to discuss them in the following order: (1) the bacteriology indicating a mixed infection with *Hemophilus hemolyticus* and *Streptococcus viridans*; (2) the association with pregnancy; (3) the probable primary focus; (4) the nature and significance of a myeloid reaction in the liver and spleen; (5) the unusual embolic phenomena; and (6) the relation of the verrucous endocarditis on the posterior mitral leaflet to the vegetative endocarditis on the anterior mitral leaflet.

There can be little doubt that in this case death was due to infection by *Hemophilus hemolyticus*, probably associated with *Streptococcus viridans*. Proof of infection by the former organism consists in the recovery in blood culture, its presence in smears and in the substance of the vegetation, and finally in its presence within the embolic glomerular lesions. Because the valvular and embolic glomerular lesions were partially productive in character and hence of some chronicity, it would seem reasonable to assume that *Hemophilus hemolyticus* was the primary infective agent. Moreover, in terminal invasions associated with endocarditis it is uncommon to find the infective microörganisms deep in the substance of the vegetations, as in this case, although they may occur on the surface.

The rôle of *Streptococcus viridans* is difficult to interpret. Although the organism is the most frequent single causative agent of bacterial endocarditis, its absence from the heart valve *in the presence of a bacteremia* is unusual. In twelve cases of bacterial endocarditis in which blood culture yielded non-hemolytic streptococci Wright [8] was able to demonstrate the microörganisms in the valve section in eleven instances. It would seem, therefore, that this organism is to be found in the valve substance in a high percentage of cases where it is the responsible agent and when blood culture is positive. The frequency with which *Streptococcus viridans* is a secondary invader in rheu-

matic and other infections, together with its absence in the histological preparations, are strong arguments in favor of a secondary infection.

Pregnancy as a complication of bacterial endocarditis is of some interest. The literature mentions relatively few cases with this feature. Kobacker [9] recently reported a case and quoted Walser who, in an extensive review of the literature, was able to find but two bona fide cases of endocarditis lenta complicated by pregnancy. Walser added two cases of his own. Blumer's comprehensive monograph [10] fails to mention the subject, from which it may be deduced that such an association is infrequent. The fetus in our case was well formed and corresponded to that of a 6 or 7 months pregnancy.

The organizing bronchopneumonia suggests itself as a probable portal of entry. The connective tissue reaction in and about the bronchial wall indicated a process of some chronicity. Blumer's analysis mentions the bronchi as a probable focus of infection in five out of fifty-eight cases of bacterial endocarditis.

The myeloid reaction present in the liver and spleen, as revealed by Giemsa's stain, was so striking as to border on the changes of a myelogenous leukemia. *A priori* there are two sources from which bone marrow cells in extramedullary sites may be derived: (1) from preëxisting cells present in those organs by a process of so-called extramedullary myelopoiesis, and (2) as circulating deposits from the bone marrow.

Maximow [11] has demonstrated the formation of bone marrow within the kidney of the rabbit after ligation of the renal arteries and vein. He believed that the endothelial cells of the liver, along with the fixed tissue cells or histiocytes, were endowed with unrestricted mesenchymal potentialities, among which was the ability to form hemocytoblasts from which all successive varieties of bone marrow cells could be derived. Whenever extramedullary myelopoiesis occurred myelocytes appeared first, followed in order by megakaryocytes and erythroblasts, but often the erythroblast stage was not reached.

Evans [12] studied the splenic tumors resulting from infections and distinguished a gray and a red variety. The former occurred in bacterial endocarditis and was characterized microscopically by the presence of numerous myeloid forms. These, he believed, arose from mononuclear myeloid cells normally present in the pulp, but indis-

tinguishable by ordinary staining methods. The red variety was typified by the splenomegaly of typhoid fever in which the predominant cell was an endothelial or mononuclear cell.

Histological examination will not absolutely solve the problem of the origin of these extramedullary myeloid cells. We feel, however, that origin from circulatory deposits is the more likely because the myeloid cells could be traced to the splenic arterioles and to the portal veins. The presence of the young myeloid forms indicates a severe and overwhelming demand upon the bone marrow for leukocytic cells which can be met only by discharge of immature forms.

The embolic phenomena here call for some consideration. Embolic deposits in the left coronary artery with accompanying focal purulent myocarditis were present and appeared identical with lesions reported by Miller and Branch, and by Russell and Fildes. These emboli were probably derived directly from the anterior mitral vegetation. Histologically they were identical with the superficial layers of the vegetations. The infrequency of such emboli may be inferred from the failure of Blumer to mention them in his monograph.

The embolic phenomena in the kidneys are the classical focal Löhlein and Baehr [13] lesions with *Hemophilus hemolyticus* demonstrable in glomerular tufts. The splenic and renal infarcts are typical. The thrombi in the iliac arteries are localized over areas of purulent destruction of the media, probably from small emboli in the vasa vasorum.

Finally it is interesting to contrast the reactions on the anterior and posterior mitral leaflets. The former was essentially an exudative reaction to bacteria, the latter essentially proliferative and bacteria-free. In fact, the gross and the histological appearance of the vegetations on the posterior leaflet was similar to that seen in acute rheumatic valvulitis, a similarity that has previously been recognized by Clawson and Bell.[14] Recently Leary [15] has described a proliferative reaction of the valve surface in rheumatic valvulitis which he has termed a "palisade formation" and which he regards as the earliest valvular response to the action of bacteria. Von Glahn,[16] Swift [17] and others have called attention to focal degeneration of collagen and elastic tissue of the rheumatic valve surrounded by proliferating cells as a pathognomonic feature of rheumatic inflammation and a precursor perhaps to the Aschoff body. Histologically, it will be recalled, the posterior mitral leaflet showed a type of

reaction similar to, if not identical with, the palisade reaction, as well as a fairly extensive area of fibrinoid swelling and degeneration within the substance of the valve surrounded by cellular reaction. The valvular responses of bacterial endocarditis may thus occasionally imitate those of rheumatic endocarditis.

Summary

1. A case of *Hemophilus hemolyticus* endocarditis associated with *Streptococcus viridans* infection is reported. It represents the second recorded instance of infection by the former microörganism.

2. The infection was complicated by pregnancy.

3. A probable portal of entry is demonstrable in the form of an organizing bronchopneumonia.

4. A myeloid reaction is exhibited in the liver and spleen and probably represents the effect of a severe and prolonged over-stimulation of the bone marrow by a bacterial toxin, with a resultant deposition of many immature marrow cells in the hemopoietic organs.

5. Some unusual embolic phenomena are present in the coronary arteries.

6. A villous endocarditis on the anterior mitral leaflet abounding in bacteria is contrasted with a proliferative reaction on the posterior leaflet. This latter reaction, together with a type of recently described "palisade formation," is here shown for the first time in subacute bacterial endocarditis. The existence of an additional lesion, described as a fibrinoid or hyaline swelling and degeneration of collagen valve substance previously demonstrated in rheumatic valvulitis and in rheumatic auricular endocarditis is shown in subacute bacterial endocarditis.

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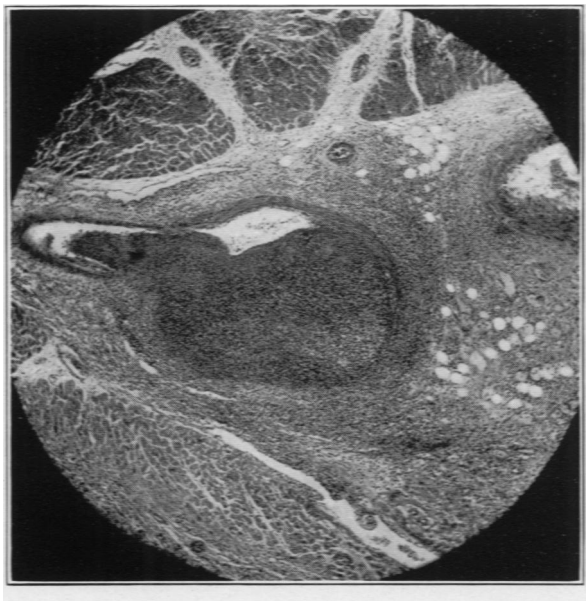
DESCRIPTION OF PLATE

PLATE 62

FIG. 1. A branch of the left anterior coronary artery with an embolic occlusion is seen. The surrounding myocardium is infarcted. Hematoxylin-eosin stain. × 70.

FIG. 2. Myeloid reaction in liver. Many immature leukocytes may be seen in a portal space and among the sinusoids. Giemsa stain. × 150.

FIG. 3. Focal embolic glomerulonephritis. Note the bacteria within the afferent arterioles and the surrounding cellular reaction. Hematoxylin-eosin stain. × 250.



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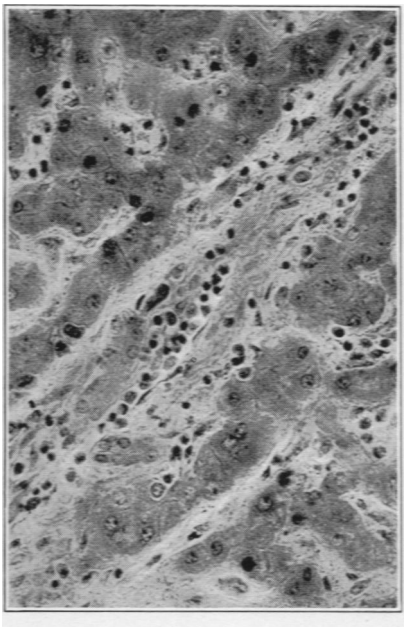

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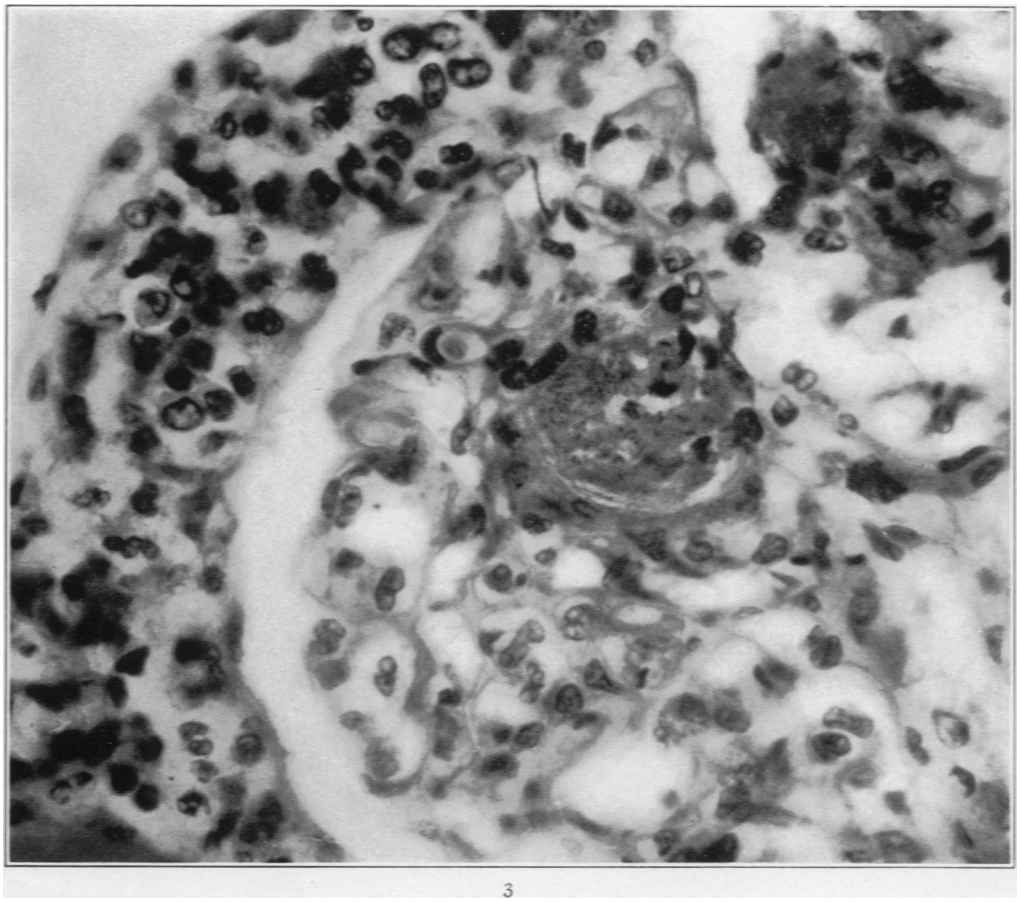

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De Santo and White Hemophilus Hemolyticus Endocarditis