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MENINGOCOCCUS ENDOCARDITIS*

REPORT OF A CASE

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The number of cases of meningococcus endocarditis reported in the literature is still so small that the meningococcus may be regarded as one of the rarest organisms producing lesions of the endocardium. In more than half of these cases the endocarditis has occurred merely as a complication of meningitis. However, there are rare instances of the meningococcus gaining entrance to the blood stream and involving the endocardium and other structures, leaving the meninges unaffected throughout the course of the illness. The accuracy of the diagnosis of a few of these cases may be questioned because in some the autopsy report is incomplete, and in others there is insufficient bacteriologic evidence.

REVIEW OF LITERATURE

The first case of meningitis in which the meningococcus was isolated from the blood stream was briefly reported by Osler[1] in 1898; and, with regard to the autopsy and bacteriologic findings, was more fully described by Gwyn[2] one year later. The bacteriologic diagnosis was based purely on the morphologic and cultural characteristics of the organism.

We have been able to find but two cases in the literature of endocarditis associated with a meningococcus septicemia in which the existence of meningitis can be definitely excluded. The first of these was reported by Cecil and Soper.[3,4] The patient, a male, 31 years of age, entered the hospital complaining of sore throat, feverishness

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and tenderness of the wrist and elbow of two weeks duration. Physical examination revealed an acute polyarthritis involving the joints of ankles and fingers, and a presystolic and systolic murmur at the apex. The provisional diagnosis was rheumatic fever and endocarditis. A gram negative diplococcus in pure culture was isolated from the blood, but there was no evidence of meningitis and the spinal fluid was normal. Pyrexia increased and the patient became delirious and died on the tenth day of observation. At the autopsy a soft cauliflower vegetation was found on the anterior curtain of the mitral valve. The spleen was large and contained an infarct. The brain, meninges and prostate were grossly and microscopically negative, and smears and cultures from the latter organ showed no gonococci. Gram negative intracellular diplococci were seen in smears and sections from the vegetations. The organism isolated from the blood stream resembled the meningococcus in morphology, cultural characteristics and fermentation reactions, and was feebly agglutinated by antimeningococcus serum.

In the second case, which was reported by Worster-Drought and Kennedy,[5] the clinical course ran over six weeks duration yet no signs of meningitis were present at any stage of the disease. The patient, a male aged 37, entered the hospital with the diagnosis of influenza. The temperature was 104° F yet there were no definite physical signs. After returning to normal for a few days the temperature gradually rose to 105° F. In the second week of observation mitral and aortic murmurs were heard; cyanosis and dyspnoea increased, and in the last weeks, the heart enlarged and the lower extremities became edematous. A gram negative diplococcus isolated from the blood culture in the second week was agglutinated by Gordon's Type II monovalent antimeningococcus serum, but not by the other three types. At autopsy, the brain and meninges were normal. The heart was enlarged, soft and dilated. A fairly tough massive fibrinous vegetation projected into the ventricle from one of the cusps of the aortic valve. The spleen contained infarcts and the kidneys were enlarged.

There are two other cases reported of meningococcus endocarditis without clinical signs of meningitis, one by Warfield and Walker [6] and the other by Rhoads.[7] In neither of these did the autopsy include an examination of the brain, hence the existence of a meningitis cannot be positively excluded.

In the case reported by Warfield and Walker, death occurred five weeks after the onset of symptoms. One week before admission the patient became delirious and developed an herpetic eruption; while under observation there was a high intermittent temperature and a gram negative diplococcus in pure culture was isolated from the blood culture. In the fourth week of the disease a blowing systolic murmur appeared and a few days later the patient died without ever showing signs of meningitis. At autopsy a large gray and red fungating vegetation firmly attached to the mitral valve, projected into the left auricle, while both the spleen and kidneys contained infarcts. The bacteriologic diagnosis of meningococcus was based simply on the morphology and cultural characteristics of the organism.

In the case reported by Rhoads, the patient, a male negro 21 years of age, died in the third week of his illness. Two weeks before admission to the hospital the patient experienced some dyspnoea, palpitation and attacks of precordial pain. Physical examination revealed a diastolic murmur over the aortic area and a loud blowing systolic murmur at the apex. The clinical diagnosis was acute endocarditis with aortic regurgitation. Death followed on the third day after admission without signs of meningitis. At autopsy a smooth, purple, friable vegetation filled a ragged perforation extending through the interventricular septum and involved both the tricuspid and aortic valves. Both kidneys contained infarcts. Smears and sections of the vegetations revealed gram negative diplococci. The diagnosis of meningococcus was based on the morphology of the organism and its colonies, and on agglutination, precipitation, and complement fixation tests.

The four preceding cases are grouped together as having two characteristics in common, first the presence of endocarditis caused by the meningococcus and secondly the absence of any signs or symptoms to suggest a pathologic lesion of the meninges.

In a case reported by Finley and Rhea,[8] meningitis occurred as a late complication. The patient was a male 47 years of age whose illness began three weeks before admission with articular manifestations and attacks of precordial pain with some dyspnoea, cough, chills and sweats. On entrance to the hospital, the heart was dilated and there was a soft systolic murmur at the apex. After being under observation for one week the temperature rose from 101° to 103° F,

the patient became dull and soporose and a hemorrhagic eruption appeared over the abdomen. At this time a positive Kernig sign was elicited, and smears of the spinal fluid showed gram negative intracellular diplococci. A similar organism was recovered from both the spinal fluid and blood. The patient died in the fifth week after the onset of symptoms. The autopsy showed a wide row of slightly pedunculated cauliflower vegetations along the mitral and aortic valves, and a yellow fibrinopurulent exudate at the base of the brain. In this case there is no report of the microscopic findings and the bacteriologic diagnosis of meningococcus is based on the morphology and cultural characteristics alone.

As pointed out earlier, meningococcus endocarditis is found slightly more commonly as a complication of epidemic meningitis, and the following cases are examples of this condition.

Weischselbaum and Ghon [9] reported a case of meningococcus meningitis in a child nine weeks old who died after an illness lasting five weeks. At the autopsy, in addition to the meningitis, fresh vegetations were found on the mitral valve. No details of the bacteriology in this case were given.

Westenhoeffer [10] reported two fatal cases; the first was a child who died after three weeks illness, and the second was a woman, 21 years of age, who lived only five days after the onset of symptoms. Autopsies were performed on both of these, and in each case showed suppurative meningitis and acute endocarditis of the mitral valve.

Markarell,[11] during an epidemic in 1915, had two cases at one time on the same ward. The first was a boy 19 years of age who was admitted with the diagnosis of meningitis. After treatment, this soon cleared up and the spinal fluid became sterile. Subsequently a loud systolic murmur was heard and a blood culture taken at this time showed gram negative diplococci. The condition of the patient gradually grew worse and death occurred twelve weeks after admission. At autopsy a very large friable vegetation was found on the mitral valve, while the brain and meninges were normal. The second case reported by Markarell was a man, 53 years of age, who lived sixteen weeks after the onset of his symptoms and meningococci were obtained from the spinal fluid during his illness. At the autopsy the spinal fluid was turbid and the meninges thickened, but in addition there was a large vegetation on the mitral valve, which almost occluded the orifice. No murmurs had been heard because of em-

physema and consequently the endocarditis had not been diagnosed clinically.

The two remaining cases of endocarditis complicating epidemic meningitis were reported by Fairley and Stewart.[12] A postmortem examination of the first showed an acute meningitis and an acute endocarditis with fresh vegetations on both mitral and aortic valves. Similarly in the second, there was, in addition to the meningitis, an acute endocarditis but in this case the lesion was confined to the mitral valve.

In almost all of these cases of acute endocarditis complicating meningitis the bacteriologic diagnosis of meningococcus endocarditis has been based simply on the morphology of the organisms seen in the spinal fluid. Probably this was sufficient because the identity of an organism is so often revealed by the part of body from which it is isolated, and yet there are exceptions in the literature bearing on this point that are of sufficient interest to warrant mentioning.

Bieck [13] on questionable evidence reported a case of gonococcus meningitis in a patient who had had a chronic urethritis for a year and who died six days after an acute exacerbation of the local condition. Autopsy revealed a patchy meningitis, while examination of the exudate showed numerous gram negative diplococci.

Teacher and Kennedy [14] had a case of micrococcus flavus meningitis following a severe head injury without rupture of the dura; this is of interest because the organism is often regarded as a harmless saprophyte.

Lastly, a case of meningococcus conjunctivitis of a fulminating character in which no general infection occurred, was reported by Ker and Douglas.[15]

Report of Case

Clinical History: D. J. (B. C. H. 572665), a white American housewife, 28 years of age, was referred into the hospital on Oct. 30, 1928 because of malaise, weakness and shortness of breath.

Present Illness: The patient was in moderately good health until three months ago when she had an abscessed tooth extracted. Very soon afterward she began to lose her appetite and vitality and first noticed weakness and shortness of breath which have progressively increased.

About six weeks prior to admission she sustained a transient hemiplegia. Shortly thereafter she gave birth to a child, and within a day or two of this she developed a mild fever, due possibly to a sore throat which she had and which her attending physician, feeling there was no question of puerperal sepsis, con-

sidered an adequate cause. At this time a loud blowing systolic murmur was first noted, and the case was then considered as probably one of endocarditis.

During the six weeks interval preceding admission and following childbirth we have little data. Her appetite diminished, she vomited frequently and lost considerable weight. We are told that she frequently spoke of pains in her finger tips and of numbness and tingling of her hands, also of slight tenderness over her anterior chest wall, and at one time of a single attack of precordial pain.

Her past history is rather vague. We do know, however, that she denied having had tonsillitis, rheumatic fever or chorea; and that her attending physician knew of no time during which she showed signs of meningitis or nephritis.

Physical Examination: A young woman, strikingly pale, moderately dyspneic, poorly nourished and acutely ill, with a temperature of 102° F. Respiration rapid (30), and rather shallow. The skin slightly icteric, warm, soft and dry, with no petechiae. The lips and extremities cyanotic, the hands and feet cold. Sclerae subicteric, mucous membranes pale. Tongue smooth, atrophic and tender. Gums showed some erosion but no bleeding. Few very small pea-sized lymph nodes bilaterally palpable and freely movable in posterior cervical triangles. Thorax not remarkable.

Heart slightly enlarged; apex impulse visible and palpable in fifth intercostal space, 8.5 cm. to left of midsternal line. Greatest diameter of cardiac dullness, 8.5 cm. to left and 2 cm. to right of midsternal line. Sounds are regular but rapid (140) and of poor quality. A loud, rough, rasping systolic murmur and coarse palpable thrill at the apex. The murmur was transmitted to the axilla and over the base of the heart. The pulmonary second sound was accentuated.

The rest of the physical examination revealed no positive findings.

Provisional Diagnosis: Subacute bacterial endocarditis.

Laboratory Findings: Blood showed a marked secondary anemia with slight leucocytosis. Red blood cells 2,200,000, and somewhat achromic. Hemoglobin 34 per cent. Leucocytes 15,500 with 68 per cent polymorphonuclear leucocytes, 9 per cent lymphocytes, 15 per cent large mononuclear phagocytes, many of which were enormously enlarged, irregular, vacuolated and contained both red blood cells and polymorphonuclear leucocytes. The remaining 8 per cent were smudges that could not be identified.

The urine showed a trace of albumen and the sediment contained many white and red blood cells with few casts.

The blood non-protein nitrogen on admission was 100 mg. Two days later it was 195 mg.

The admission blood culture showed gram negative bean-shaped diplococci; a second culture on the following day confirmed this finding.

Progress Note: The second morning the temperature was 103° F, pulse 150 and respiration 40. The patient was irritable, restless and at times irrational.

Discharge Note: No outspoken embolic lesions appeared until the day of death when a few small purplish areas were found on the fingers and toes. The patient became comatose and delirious by turns, screamed that she could not get her breath, became more cyanotic, pulseless and died on the fourth day apparently in uremia.

Final Diagnosis: Uremia, acute nephritis, acute endocarditis, secondary anemia.

Autopsy Report

An autopsy (A28–382), was performed on November 7, five days postmortem. There is edema of the lower extremities. The breasts are slightly enlarged, the areola pigmented and milk is expressed from the nipples. A deeply pigmented line extends from the umbilicus to the symphysis. The vagina is red and ulcerated, and the cervix is unusually soft. No cutaneous petechiae can be distinguished with certainty.

The peritoneal, pleural and pericardial cavities contain 500 cc., 60 cc., and 50 cc. respectively of clear yellow watery fluid.

Heart: Weight 330 gm., slightly enlarged with moderate dilatation on the right side. The myocardium is soft, pale and homogeneous. At about the central portion of the upper surface of each leaflet of the mitral valve, a large, elevated, nodular, yellow vegetation measuring 2 by 1 by 1.3 cm., projects into the cavity of the left atrium. Beneath these fungating vegetations the leaflets are thickened in some places and perforated in others. The adjacent portions of the valve, except for a few delicate pink fibrinous granulations and two thickened and injected chordae tendineae are normal. The remaining valves, the pericardium, endocardium and coronary vessels are normal.

Lungs: They are distended, pale and edematous. The bronchi and bronchioles are filled with watery blood-tinged fluid.

Spleen: Weight 440 gm., is uniformly enlarged and varies in color and consistence. A large, firm, irregular yellow infarct passing through the central portion stands out in sharp contrast to the surrounding soft maroon parenchyma.

Liver: Weight 1740 gm., is pale, moderately firm and slightly enlarged. The fresh surfaces are homogeneous but the finer markings are obscured by a swelling of the parenchyma.

Kidneys: Weight 390 gm., large and firm. The capsules strip readily from smooth pale surfaces. The cortex is regular and 0.7 cm. in thickness. The fresh surfaces bulge slightly and show marked pallor of both cortex and medulla.

Genital Organs: The cervix is congested. Part of the endometrium covering the fundus of the uterus is dark red and covered with a fibrinous exudate. The tubes and ovaries are negative.

Meninges: The inner surface of the dura covering the parietal and frontal lobes is coated with a very thin yellowish glistening rather mucoid exudate. There are three minute petechial hemorrhages in the pia arachnoid covering the cerebrum but there is no visible inflammatory reaction.

Brain and Cord: These were fixed before sectioning and later carefully examined by serial sections. No gross lesions are found throughout the brain, in the ventricles or in the choroid plexus.

Anatomic Diagnoses: Endocarditis; meningitis; infarction of the spleen; ascites; pulmonary edema; endometritis.

Microscopic Report

Heart: The myocardium shows surprisingly little pathology. There are a few small perivascular foci of polymorphonuclear leucocytes; and occasional necrotic muscle fibers are invaded by endothelial leucocytes. The vegetations are composed of a coarse network of fibrin surrounded by clumps of bacteria and covered at the periphery by a layer of polymorphonuclear leucocytes. The bacteria are small gram negative diplococci and many are intracellular. The underlying valve leaflets are necrotic. The pericardium and vessels are normal.

Lungs: The structure of the lung is well preserved. The alveoli contain a little precipitated albumen with scattered foci of fibrin and red blood cells.

Spleen: There is a large area of infarction bordered by an acute inflammatory exudate and extravasated red blood cells.

Kidneys: These show a rather diffuse lesion involving primarily the glomeruli. The capillary tufts are large, cellular and contain little blood. Groups of tubules also show a moderate inflammatory reaction with degenerative changes within the cells and numerous polymorphonuclear leucocytes within their lumina.

Uterus: Slight cystic dilatation of the glands of the cervix, few foci of lymphocytes in the stroma, and an acute inflammatory exudate on the surface of the endometrium.

Tubes and Ovaries: These organs are normal.

Breast: A normal involuting breast, the cells are atrophic and there is little secretion within the glands.

Meninges: The under surface of the dura is covered with a narrow layer of fibrin containing red blood cells and polymorphonuclear leucocytes. A collection of lymphocytes, plasma cells and endothelial leucocytes lies beneath the lining endothelium of the sagittal sinus, and infiltrates the wall of one of the larger vessels of the choroid plexus.

The pia arachnoid is normal except for three small perivascular foci of acute inflammation and hemorrhage. The inflammatory exudate is composed of well preserved polymorphonuclear leucocytes, endothelial leucocytes and little fibrin.

Cerebrum and Cerebellum: Sections from many different parts including the walls of the ventricle and the cervical portion of the cord, show no histologic lesions.

Pituitary: In the anterior lobe there is a small area of necrotic cells that are undergoing phagocytosis.

Microscopic Diagnoses: Endocarditis; meningitis; infarction of spleen; pulmonary edema; subacute intracapillary glomerular nephritis; endometritis. Gram negative bean-shaped diplococci, intra- and extracellular in vegetations on mitral valve.

Bacteriology

An excellent opportunity was offered to study the bacteriology in this case and the results are unusually interesting. A gram negative bean-shaped diplococcus was recovered from the blood cultures taken ante mortem and later from cultures from the vegetations on the mitral valve and from the spleen. Of the smears taken at the time of the autopsy from the base of the brain, from the vagina, cervix and fundus of the uterus and from the vegetations and spleen, only those from the latter two situations revealed a similar organism.

Morphology and Cultural Characteristics: The organism was a small gram negative bean-shaped diplococcus. It varied in size and stained with dilute carbol fuchsin with different degrees of intensity.

In spite of the fact that the organism remained viable in the body for five days postmortem it grew with difficulty under artificial conditions. The most favorable media were glucose ascitic agar and chocolatized sheep blood agar. A scanty growth was obtained on sheep blood agar, Loeffler's serum media, ascitic broth and blood

serum broth, whereas no growth occurred on plain agar or on beef infusion broth.

After twenty-four hours incubation at 37° C the colonies were minute, discrete, moist, round and elevated. They had a grayish blue translucency, produced no hemolysis and formed no pigment. No growth was obtained at room temperature or at 40° C. Cultures placed in the icebox died out in three to four days.

Fermentation Reactions: These were obtained with difficulty using 1 per cent dextrose broth and 1 per cent maltose broth enriched with horse serum. Control tubes were inoculated with known cultures of meningococci and gonococci. No change in reaction was observed in the tube containing the unknown organism after twenty-four hours incubation, while only at the end of six days was the change in the reaction complete. The unknown organism resembled the meningococcus by attacking both sugars and differed from the gonococcus which fermented only the dextrose (Table I).

TABLE I

Fermentation Reactions

Organism	Dextrose	Maltose
Unknown	+	+
Meningococcus	+	+
Gonococcus	+	−
Tube uninoculated	−	−

SEROLOGY

Arkwright [16] pointed out that to identify a gram negative diplococcus accurately one must take into account not only the morphology of the cocci and their colonies, their cultural characteristics and fermentation reactions but also their agglutination and agglutinin absorption reactions with known antimeningococcus sera.

Gordon,[17] in his bacteriologic studies of cerebrospinal fever among the troops, showed that about 98 per cent of all meningococci fall into four groups with minor bonds of relationship between his types I and III, and types II and IV. Furthermore, the monovalent agglutinating sera of types II and IV of the meningococci contain abundant coagglutinins for many strains of gonococci, and different

strains of gonococci vary greatly in their serologic resemblance to meningococci.

Because of this close relationship between these two groups of organisms we performed agglutination tests to determine into which of these groups the unknown organism should fall.

AGGLUTINATION *

Agglutination with Polyvalent Antimeningococcus Serum: Faintly turbid suspensions of twenty-four hour cultures of the unknown organism as well as of four types of meningococci grown on chocolate agar slants were made in salt solution and kept at 56° C for one hour. Four-tenths of a cubic centimeter of bacterial suspensions were added to one-tenth of a cubic centimeter of graded dilutions of standard polyvalent antimeningococcus serum. As controls we used normal horse serum and bacterial suspension, and saline and bacterial suspension. The tubes were stoppered, kept at 55° C for twenty-four hours and placed in the icebox for twenty-four hours before reading.

The agglutination reaction of the unknown organism corresponded very closely to the reactions of the four known types of meningococci. It was complete in a dilution of 1:200 and partial up to 1:800. This, together with the results of the control tests, is shown in Table II.

TABLE II

Agglutination with Antimeningococcus Serum

Strain	Final dilutions Antimeningococcus serum					Saline	Normal horse serum 1:100	Partial agglutination	Complete agglutination
	1:200	1:400	1:800	1:1600	1:3200				
Unknown organism.....	4†	3	2	0	0	0	0	1:800	1:200
Meningococcus Type I.....	4	4	2	0	0	0	0	1:800	1:400
" " II.....	4	3	2	1	0	0	0	1:1600	1:200
" " III.....	4	3	2	0	0	0	0	1:800	1:200
" " IV.....	4	3	2	0	0	0	0	1:800	1:200

† Figures refer to degree of agglutination reaction: 4 = complete.

* The polyvalent antimeningococcus serum, the four known types of meningococci, and the normal horse serum used in these experiments were obtained from the Commonwealth of Massachusetts Antitoxin and Vaccine Laboratory. The types of meningococcus were: Type I, No. 123; Type II, No. 55; Type III, No. 57; Type IV, No. 60, (United States Hygienic Laboratory).

Agglutination with Polyvalent Antigonococcus Serum: Using the same technic as in the former test we replaced the polyvalent antimeningococcus serum with polyvalent antigonococcus serum, and added a known strain of gonococcus as an additional control (Table III).

TABLE III

Agglutination with Polyvalent Antigonococcus Serum

Strain	Final dilutions Antigonococcus serum					Saline	Normal horse serum 1:100	Partial agglutination	Complete agglutination
	1:200	1:400	1:800	1:1600	1:3200				
Unknown organism.....	4	3	2	0	0	0	0	1:800	1:200
Meningococcus Type I.....	4	3	2	0	0	0	0	1:800	1:200
" " II.....	3	2	0	0	0	0	0	1:400	...
" " III.....	2	0	0	0	0	0	0	1:200	...
" " IV.....	0	0	0	0	0	0	0	....	...
Gonococcus.....	4	4	4	3	1	0	0	1:3200	1:800

In this, agglutination of both the unknown organism and meningococcus Type I was complete in a dilution of 1:200, and partial up to 1:800, which resembled the results of the previous test in which antimeningococcus serum was used. Only partial agglutination resulted with meningococcus Types II and III in dilutions of 1:400 and 1:200 respectively, while the gonococcus agglutination was complete up to 1:800.

The identity of the unknown organism could not be determined from the results of these two agglutination tests alone (Tables II and III), because it was agglutinated equally well by both the antimeningococcus and antigonococcus sera.

AGGLUTININ ABSORPTION

Agglutinin absorption tests were carried out using both polyvalent antimeningococcus and polyvalent antigonococcus sera, a mixed suspension of the four types of meningococci and the unknown organism.

The technic of the first test consisted in absorbing the agglutinins against the four types of meningococci from an homologous polyvalent antimeningococcus serum, and then mixing this absorbed serum with a suspension of the unknown organism. This was accomplished in three steps. First, five-tenths of a cubic centimeter of a mixed

suspension of the four types of meningococci were added to five-tenths of a cubic centimeter of the homologous polyvalent antimeningococcus serum diluted 1:20. After twelve hours at 55° C followed by two hours in the ice chest, agglutination was strongly positive. The mixture was thoroughly centrifuged and eight-tenths of a cubic centimeter of clear supernatant fluid were pipetted off without disturbing the precipitate.

The second step, which was rather a repetition of the first, was carried out simply to insure the complete absorption of the agglutinins against the meningococci. To the supernatant fluid obtained after the first agglutination we added two-tenths of a cubic centimeter of a fairly heavy mixed suspension of the four types of meningococci, giving a final dilution of 1:50. This mixture was kept at 55° C for twelve hours and later placed in the ice chest for two hours. Only very slight agglutination occurred. The suspension was cleared by centrifuging and the supernatant fluid was saved for the agglutination test with the unknown organism.

In the last step, three-tenths of a cubic centimeter of a suspension of the unknown organism were added to one-tenth of a cubic centimeter of the absorbed serum diluted 1:50, giving a final dilution of 1:200. A final reading was made after twelve hours at 55° C followed by twelve hours in the ice chest.

The following protocol (Table IV) shows that the absorption of antibodies from the antimeningococcus serum by a mixture of the

TABLE IV

Absorption Test with Polyvalent Antimeningococcus Serum Absorbed with Mixed Meningococci

Strain	Absorbed serum 1:200	Unabsorbed serum 1:200
Unknown organism	0	4
Mixed meningococci	0	4

four types of meningococci completely removed the agglutinins against the unknown organism. To certify that the agglutinins against the four types of meningococci had been completely removed, a mixed suspension of the meningococci was likewise added to the absorbed serum; and no agglutination occurred. The unabsorbed antimeningococcus serum that was used as a control in this

protocol (Table IV), had been previously subjected to the same treatment as the absorbed serum because the possibility was considered that the prolonged heating, the chilling, and the centrifuging might destroy or alter the agglutinins in this serum.

When the antimeningococcus serum was replaced by antigonococcus serum and precisely the same technic employed, the agglutinins against the unknown organism were again absorbed by the mixed types of meningococci (Table V).

TABLE V

Absorption Test with Polyvalent Antigonococcus Serum Absorbed with Mixed Meningococci

Strain	Absorbed serum 1:200	Unabsorbed serum 1:200
Unknown organism	0	4
Mixed meningococci	0	3

In these two absorption tests (Tables IV and V), absorption of the antibodies from both antimeningococcus and antigonococcus sera was obtained by using a mixed suspension of the four types of meningococci, and in each case the agglutinins against the unknown organism were completely removed as well. This was important for it suggested a close similarity between the agglutinins against the unknown organism and the meningococci.

TABLE VI

Absorption Test with Polyvalent Antimeningococcus Serum Absorbed with Unknown Organism

Strain	Absorbed serum 1:200	Unabsorbed serum 1:200
Unknown organism	0	4
Mixed meningococci	4	4

When the converse of the first agglutinin absorption test was performed, it was found (Table VI) that the polyvalent antimeningococcus serum absorbed with the unknown strain would still agglutinate a mixed suspension of the four types of meningococci. This indicated that by absorbing the agglutinins against the unknown organism, the agglutinins for all the four types of meningococci have

not been removed. If on the other hand polyvalent antimeningococcus serum absorbed with the unknown organism was mixed with the four types of meningococcus separately, agglutination occurred with all but Type I. This suggested that agglutinins for this type had been previously absorbed (Table VII).

TABLE VII

Absorption Test with Polyvalent Antimeningococcus Serum Absorbed with Unknown Organism

Strain	Absorbed serum 1:200	Unabsorbed serum 1:200
Unknown organism	0	4
Meningococcus Type I	0	4
" " II	4	4
" " III	4	4
" " IV	4	4

A final absorption test which suggested even more strongly the identity of the unknown organism entailed incubating polyvalent antimeningococcus serum absorbed by meningococcus Type I with the unknown organism (Table VIII). This table shows that after absorption of the agglutinins by meningococcus Type I from polyvalent antimeningococcus serum no agglutination was obtained from the unknown organism.

TABLE VIII

Absorption Test with Polyvalent Antimeningococcus Serum Absorbed with Meningococcus Type I

Strain	Absorbed serum 1:200	Unabsorbed serum 1:200
Unknown organism	0	4
Meningococcus Type I	0	4

From results of the preceding protocols it would appear that the unknown organism should be grouped as a Type I meningococcus. (U. S. Hygienic Laboratory classification.)

Discussion

Meningococcus endocarditis presents many points of clinical, pathologic and bacteriologic interest. Of the twelve cases which we found in the literature all have ended fatally. In seven of these the cardiac lesions occurred simply as a complication of meningitis and in most cases the endocarditis was not recognized clinically. On the other hand it is important to remember that endocarditis can be caused by the meningococcus without any meningeal involvement and that such cases have been diagnosed as acute rheumatic fever with endocarditis, or subacute bacterial endocarditis. The duration of such cases varies from three weeks to three months. In the case that we have reported in which there were no clinical signs or symptoms to suggest any meningeal involvement, a diagnosis of meningitis was made at the time of the autopsy that was later confirmed by the microscopic examination of the tissue.

Of unusual pathologic interest are the rather typical vegetations — large, firm, localized and fungating, with little tendency to discharge minute emboli. In eight of the cases the mitral valve alone was affected; in three, both the mitral and aortic; in one, the aortic alone, and in one other, the case reported by Rhoads, the interventricular septum was perforated and both the tricuspid and aortic valves were involved.

The bacteriology in these cases of meningococcus endocarditis presents a very interesting problem, and especially in those cases in which there is no associated meningitis. In several of the cases reported, the bacteriologic diagnosis was based merely on the morphology and cultural characteristics of the organisms isolated from blood cultures. These gram negative diplococci, however, form a group of such closely related organisms that such diagnoses are not conclusive. In identifying members of this group the organisms should be agglutinated, and in some cases agglutinin absorption tests performed.

SUMMARY

1. A case of meningococcus endocarditis is reported with the autopsy and bacteriologic findings.

2. A brief discussion deals with the clinical, pathologic and bacteriologic considerations of meningococcus endocarditis.

3. The literature is reviewed.

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DESCRIPTION OF PLATE

PLATE 41

Photograph of the heart, opened to show the characteristic large fungating vegetations, localized on the leaflets of the mitral valve.