CARDIO-VASCULAR SYPHILIS.

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The incidence of syphilitic cardio-vascular disease is small in comparison with heart disease resulting from acute rheumatism and arterio-sclerosis; and of all patients suffering from cardiac disease less than one in twenty is found to be suffering from disease due to syphilis. Autopsies on syphilitic subjects have shown the aorta to be diseased in half the cases examined and recent observations suggest that the incidence of such changes has increased, despite the introduction of the arsenical preparations.

Infection with syphilis having occurred, numerous factors appear to determine the occurrence of clinical heart disease due to this cause. The initial treatment may be faulty, the social status of the patient may result in neglect of treatment owing to indifference or inability to undergo a long course of treatment. The majority of victims are males and this suggests that heavy manual labour may be conducive to the production of vascular changes in infected subjects.

The symptoms of cardiac disease arise some twenty or more years after the primary infection, although sometimes symptoms may arise within five years. The condition is commonly diagnosed between the ages of forty and sixty.

Pathology.

Brown and Pearce[1] have shown by experiments on rabbits that the duration of a syphilitic lesion is inversely proportional to the intensity and extent of the local reaction, and the latter determine the clinical manifestations of the disease (which may explain the frequency with which a history of the primary infection in man cannot be obtained). The spirochætes lodge in the perivascular lymph spaces and excite a typical reaction, consisting of an accumulation of lymphocytes and plasma cells. The endothelial lining of the capillaries swells, resulting in narrowing or obstruction of the lumen. Proliferation of fibroblasts occurs and when healing sets in there may be considerable fibrosis. The gross changes found in the aorta result from this syphilitic reaction. Endarteritis of the vasa vasorum of the adventitia and media of the aorta produces lymphocytic infiltration with consequent destruction of the elastic laminæ, producing a replacement of the elastic fibres by fibrous tissue, which yields under pressure within the aorta and leads to fusiform or saccular dilatation and the formation of aneurysms.

Syphilitic aortitis usually commences about one inch above the aortic valves, it may extend to the aortic ring causing dilatation of the aortic orifice and incompetence of the valves. The cusps of the valves may be normal, but often become sclerosed and contracted with a cord-like thickening of the free edges, and the commissure between the adjacent cusps may be widened owing to disease of the aortic wall. Changes in the sinuses of Valsava may produce stenosis of the orifices of the coronary arteries, at times so marked that the orifices can be penetrated by only the finest of probes. Distally the changes may extend to the commencement of the abdominal aorta, and rarely the abdominal aorta itself may be diseased.

The macroscopic changes in the aorta are characteristic ; soft, raised, gelatinous-looking swellings appear in the intima, at first about one inch above the aortic valves; as the disease advances fibrosis occurs and the surface becomes scarred and pitted with the production of longitudinal wrinkles, and usually the change from obviously diseased to apparently healthy tissue is abrupt. The heart shows hypertrophy of the left ventricle, and may show gummatous deposits or fibrotic changes. Acute syphilitic myocarditis is also described. Syphilitic endarderitis and periarteritis may be found in the small vessels of the brain and meninges, and a few cases have been described in which similar changes have been found in the smaller branches and arterioles of the pulmonary artery.

Clawson and Bell[2] record the causes of death in 126 cases of syphilitic aortitis revealing the following conditions: aortic insufficiency 46, coronary occlusion 25, ruptured aortic aneurysm 35, and myocardial gummata 3. Death from other causes 17.

Symptoms.

The symptoms of cardio-vascular syphilis are those produced by the pathological changes and vary from case to case. In the early stages of syphilitic infection, fatigue, dyspnœa on exertion, and tachycardia may be present, and suggest the presence of a myocarditis, but as a rule cardiac symptoms do not arise until aortic regurgitation, coronary ischæmia, aortitis or other gross changes are present, and then symptoms of myocardial insufficiency present themselves.

The earliest symptom is a vague præcordial discomfort on exertion, and within a short time dyspnœa and early fatigue become noticeable. The præcordial discomfort then progresses to a definite substernal oppression or anginal pain and the attacks tend to increase in frequency and severity. In like manner dyspnœa becomes more marked, and attacks of paroxysmal dyspnœa may occur, later assuming the character of "cardiac asthma." Palpitations of the heart and vertigo are common complaints at the onset of symptoms. Finally definite signs of congestive heart failure such as orthopnœa and œdema may occur.

When dilatation of the aorta occurs many other symptoms may arise as the result of pressure of an aneurysmal sac on the surrounding structures.

Syphilitic arteritis of the pulmonary arteries produces dyspnœa, cough, cyanosis and hæmoptysis.

Physical Signs.

In the early stages the most suggestive sign is a *ringing metallic aortic second sound,* and as this sign results from dilatation of the aortic ring it necessarily follows that the pitch of the sound tends to be lower than normal.

Pulsation may be seen and felt in the second right intercostal space close to the sternum.

An increase of the area of cardiac dullness may be demonstrable in the first and second intercostal spaces to the right or left of the sternum.

Radiological examination will demonstrate enlargement of the aorta, the right border of the ascending arch becoming visible lateral to the superior vena cava

and encroaching upon the shadow of the right auricle. At a later stage distortion of the aorta owing to aneurysmal dilatation may be found. The heart shadow is increased in size.

As the disease progresses a faint systolic murmur may be audible over the aortic area, and not infrequently this murmur is found to vary in intensity from one examination to another. When the aortic valves become incompetent marked increase in the size of the heart occurs and a typical diastolic murmur develops.

Diagnosis.

The diagnosis of cardio-vascular syphilis depends upon the recognition of cardiac disease together with evidence of syphilitic infection and the absence of other causes of disease. History of syphilitic infection is commonly absent, but a history of a gonorrhœal infection should suggest the possibility of a concomitant luetic infection which had been overlooked. The blood Wassermann reaction is positive in 80 per cent. of cases, but it must be remembered that a negative reaction does not exclude syphilis as the cause for the cardiac condition, nor does a positive reaction prove that the cardiac changes are necessarily due to syphilis.

Syphilitic aortitis should be considered as a possibility when a patient between the age of 40 and 60 develops symptoms of hitherto unsuspected cardiac disease; when the symptoms and signs point to involvement of the aorta, aortic valves or coronary arteries.

Signs of aortic stenosis in the absence of dilatation of the aorta may be taken as evidence that the condition is not due to syphilis.

In young adults malaise and tachycardia usually suggest a diagnosis of thyrotoxicosis or pulmonary tuberculosis, but the symptoms may be due to syphilitic infection, and it may be necessary to test the Wassermann reaction of the blood in some cases.

Prognosis.

A diagnosis of cardio-vascular syphilis implies a poor prognosis in most cases. In those patients presenting symptoms of early congestive failure the expectation of life is from six months to three years. In patients suffering from slight dyspnœa and præcordial discomfort the outlook is slightly better, but the occurrence of anginal attacks is of very grave significance, the majority of patients surviving only a few months or at the most two years.

Treatment.

Treatment of the symptoms due to myocardial insufficiency will necessitate rest in bed, digitalis, and diuretics, and is similar to that prescribed for cardiac failure due to other causes, and when relieved by such measures anti-syphilitic remedies may be applied.

Potassium iodide and mercury are the most useful drugs and in many cases are the only anti-syphilitic remedies which may be given. No case should be given bismuth or arsenic until potassium iodide and mercury have been used for at least two months, nor should they be administered if angina pectoris has occurred.

Potassium iodide should be given at first in 5 grain doses three times a day and the dose should be increased each week by 5 grains until 20 grains three times a day is being taken. Mercury may be prescribed in the form of Liquor. hydrargyrum perchloridum to be taken in one drachm doses three times a day, or it may be applied as an inunction, one drachm of mercurial ointment being massaged into the skin each day for twelve successive days, and this course should be repeated each month for three months.

If neosalvarsan is given the initial dose should be not more than 0.15 grammes intravenously, and if this dose is tolerated it may be given weekly for two months, slowly increasing the dose to 0.45 grammes. A similar course of treatment with neosalvarsan should be given every six months.

Neosalvarsan may precipitate an anginal attack, and should this occur such treatment must be abandoned.

REFERENCES.

1. Brown and Pearce: J.A.M.A. 1921, lxxvii, 1619.
2. Clawson and Bell: Arch. Path. and Lab. Med. 1927, IV, 922.

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