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Primary aortitis in childhood

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The case of a 5-year-old boy affected by autoimmune aortitis is reported. The onset was acute, the progression rapid. Hypertension, absence of right radial pulse, elevated ESRand immune globulin levels, cellular and humoral immunity against blood vessel wall were observed. The tests suggested an autoimmune origin of the aortitis. On immunosuppressive treatment the signs of activity disappeared and the condition improved and after captopril treatment the blood pressure decreased.

During the last thirty years an increasing body of reports has been published on obstructive panarteritis of the aorta and the great arteries. The disorder is rare in Europe, especially in children. We describe here a case in whom immunological studies relevant to the aetiology of the disease have been carried out.

REPORT OF A CASE

The five years old boy was first admitted on 25 September, 1979. He had fever, experienced thickening of his fingertips and pain in the extremities and the chest one month before admission. At admission we saw a normally developed boy with dyspnoea, pale skin and clubbed fingers. A marked jugular and epigastrial pulsation could be observed. No pulse was palpable on his right arm and above the right subclavian artery. Pronounced pulsation was present in his left extremities while a reduced pulse was observed in the right lower extremity. No difference could be demonstrated in the circumference of the extremities. Oscillation was hardly measurable on his right arm, it was reduced on the right leg. No blood pressure could be measured on the right arm, the value was 180/120 mmHg on the left arm. The flush method gave a blood pressure of 105 on the right arm, 150 on the left arm, 120 on the right and 155 mmHg on the left leg. Above the abdominal aorta a systolic murmur was heard. The liver was enlarged by 2 cm. The eyegrounds were normal.

Chest X-rays revealed an enlarged heart with a dilated left ventricle and markedly dilated aorta. The ECG showed signs of left ventricular hypertrophy. Intravenous pyelography gave normal findings.

Laboratory findings: ESR: 40 mm/h; WBC: 10.5 G/l; haemoglobin: 1.44 mmol/l (9.3 g/dl). Blood smears urinalysis, platelet count, serum iron, total lipid, cholesterol, creatinine, glu-

cose, urinary vanylmandelic acid, liver function tests, LE cell tests, VDRL were normal. The Mantoux test showed a normergic reaction. Serum total protein was 69 g/l; albumin: 0.39; alpha$_1$: 0 07; alpha$_2$: 0.14; beta: 0.18; gamma globulin: 0.22. Plasma sodium and chloride, were normal, plasma potassium 3.6 mmol/l.

Vasodilator treatment resulted a drop of the blood pressure measured on the left arm to a value of 135/90 mmHg.

As the parents refused the recommended aortography, the child was discharged after prescription of antihypertensive treatment.

He was readmitted on 2 March 1980, in an unconscious state and having clonic convulsions. His blood pressure was 240/130 mmHg on the left arm. Anticonvulsive, antihypertensive and dehydrating treatment resulted in regain of consciousness. The eyeground arteries showed constriction, the Gunn sign was positive. This time the laboratory findings were ESR: 60 mm/h; WBC: 11.4 G/l; plasma potassium: 4.1 mmol/l; serum total protein: 78 g/l; gamma globulin: 0.28; IgG: 20.5 g/l; IgM: 2.80 g/l; IgA: 3.20 g/l; CRP: 50 mg/l; alpha$_1$-antitrypsin: 6.1 g/l; alpha$_1$ acid glycoprotein: 2.54 g/l; haptoglobin: 4.25 g/l; C_3 0.90; C_4, 0.48 g/l; no antinuclear factor was found in the serum.

The following blood pressure values were obtained by the ultrasound method: right brachial artery: 120, left brachial artery: 170, arteria dorsalis pedis, right: 140, left: 175; arteria tibialis posterior, right: 142, left: 190 mmHg.

This time the parents consented to the aortography. The hepatic and lienal arteries showed normal filling, the distal section of the aorta showed unevenness and multiple constrictions. The initial section of the left renal artery was narrow, the right renal artery could not be distinguished (Fig. 1). The left iliac artery showed two constricted sections shortly after the bifurcation of the aorta, on the right side the common iliac artery showed a complete obstruction over a two cm long segment adjacent to the bifurcation. The external and internal iliac artery was intensively filled up through collateral vessels (Fig. 2).

As an autoimmune process against the blood vessel wall was suspected, immunological studies were carried out. Human aorta affected by lipid plaques and normal vena cava intima were used as antigens. The antigens were applied as an extract prepared with calcium chloride — tris — citrate buffer [46]. Since it was supposed that the specifically reacting fraction of the blood vessel antigen is a low density lipoprotein, LDL antigen prepared by gradient ultracentrifugation was used for testing the cell-mediated immunity. The tests performed were a modified leucocyte migration test [45] for cellular immunity (normal value MI = 0.8 to 1.2), passive haemagglutination [4] for humoral immune responses (normal value below 1 : 32), for circulating

immune complexes a modified method of complement consumption and Clq solubility [9] (normal values below 15% and 0.28, respectively) and a method utilizing polyethyleneglycol (PEG) precipitation [13] (normal value below 0.04).

The following results were obtained.

Cellular immune response: MI = = 0.32 in the presence of aorta antigen, 0.39 with venous antigen, 0.31 with LDL antigen. The MI value was 0.55 in the presence of aorta antigen plus autologous serum, 0.32 with LDL plus adresone, 0.43 with LDL plus azathioprine, 0.30 with LDL plus clofibrate. Humoral immune reponse: 1 : 256 with aorta antigen, 1 : 2048 against venous intima. Circulating immune complexes: negative result with complement consumption test, 0.35 with Clq solubility test, 0.02 with PEG precipitation.

The inhibition of leucocyte migration in the presence of aorta and vein wall antigen or LDL pointed to cellular sensitization and, at the same time, elevated levels of humoral antibodies against blood vessel wall antigens were demonstrated. It was therefore decided to apply immunosuppressive treatment with dexamethasone and chlorambucil and, in addition, vasodilatory, sympathicolytic and diuretic therapy.

As a result, blood pressure stabilized at 200/120 mmHg. The steroid was administered in a gradually

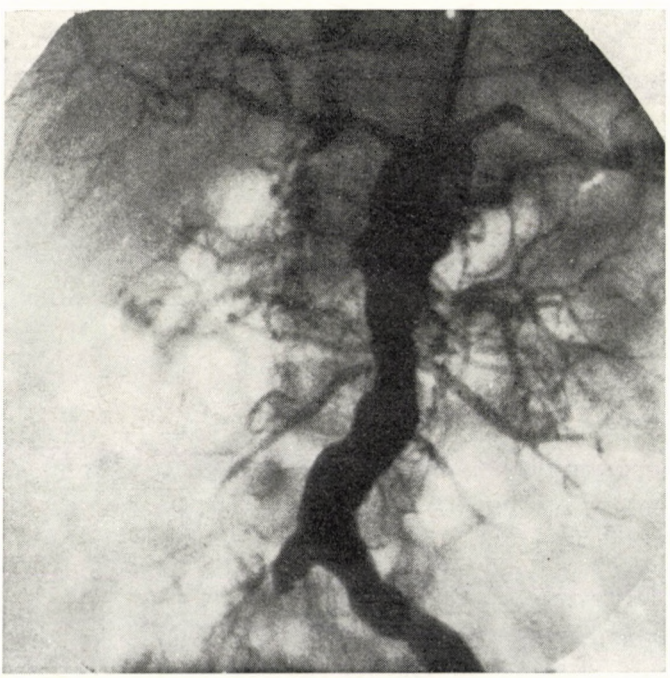

FIG. 1 Lumbar aortography. The aorta has roughly uneven contours with multiple constrictions. The left renal artery is constricted near the aorta, no right renal artery can be distinguished

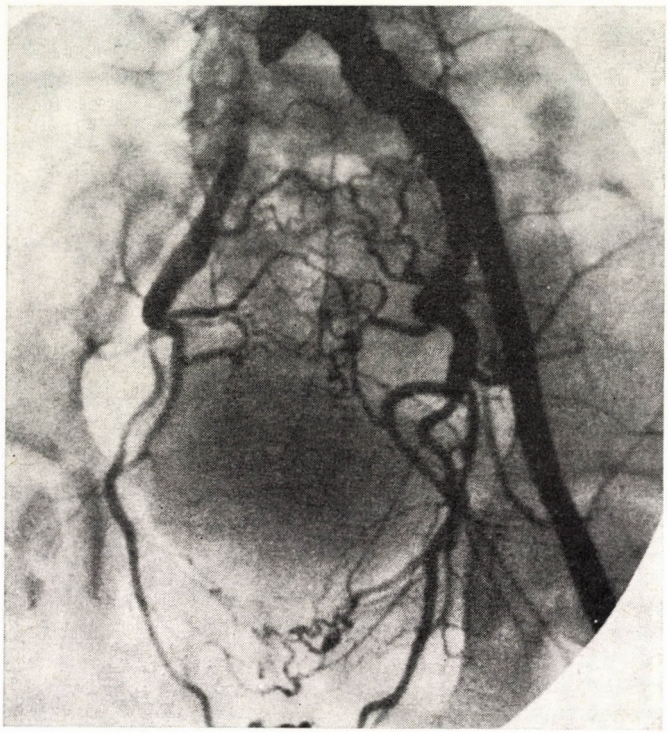

FIG. 2 The left common iliac artery shows two narrow segments after the bifurcation. The right common iliac artery displays a two cm long completely obliterated segment. The right external and internal iliac arteries fill through the extensive collateral network

decreasing dose over 4 months, chlorambucil for 5 months. The boy's condition improved, he became free of complaints, the laboratory findings normalized. The circumference of the right arm showed a reduction by 1.5 cm in comparison to that of the left arm. and a 1 cm difference developed between the femoral circumference values. By January, 1981, blood pressure became 150/110 mm Hg on the right arm, the right radial pulse became palpable. By this time the ESR again increased to a value of 45 mm/h, the previously normal gamma globulin value was again elevated to 0.25, and IgG was 12.8 g/l, with normal values for all other immune globulins. Renin activity in blood plasma gave a basal value of 33 ng/ml/24 h, after 2 hours of walking it was 40 ng/ml/24 h (with our method the normal basal value lies between 5 to 16 ng/ml/24 h).

Histological examination of a specimen removed from the pectoralis major muscle showed normal arterioles. In the specimen, by immunofluorescent methods no IgA, IgE, IgG, IgM, C' or fibrin could be found. The studies involving immunity against blood vessel wall antigens were again carried out, with the following results: MI: 0.79 in the pres-

ence of aorta antigen, 0.68 with venous antigen. Humoral immune response against aorta antigen: 1 : 1024, against venous wall antigen: 1 : 128. Circulating immunocomplexes: negative with complement consumption test, 0.46 with the Clq solubility test, 0.03 with PEG precipitation.

The relapse of the autoimmune process has made us to reintroduce dexamethasone and chlorambucil treatment. Chlorambucil was applied for 4 months and in October, 1981, the steroid is still taken. On captopril with diuretic treatment the blood pressure decreased to 125/80 mmHg. The patient's condition now is satisfactory and the laboratory findings are normal.

Discussion

Primary aortitis attacks the great vessels rich in elastic fibres. The inflammatory process begins in the adventitia near to the media, with round cell infiltration. As the disorder proceeds, panarteritis and periarteritis develop, with destruction of the elastic fibres [6, 17, 18, 21, 22, 23, 30, 41]. Weakening of the blood vessel wall can lead to development of saccular aneurysms, proliferation of the intima may narrow the lumen which then may completely be obstructed by thrombi formed in the constricted segments. In most cases the aortic disorder is segmental, alternation of diseased and uninjured segments results in an irregular lumen. The great arteries are usually attacked at their arisal from the aorta. The blood vessels are obstructed gradually, leaving time for development of a collateral circulation. The disorder may also involve the pulmonary artery [19, 22, 26, 30, 31].

Any segment of the aorta may be involved, the symptoms may thus be variable. The various forms of localization were regarded as separate disorders, therefore the condition has many synonymous descriptions: aortic arch syndrome, pulseless disease, young women's arteritis, (obliterative) brachiocephalic aortitis, atypic coarctation, "inverse" coarctation, middle aorta syndrome, panaortitis syndrome, epiaortic arteritis, Takayasu's disease, Takayasu's syndrome, Takayasu's aortitis, etc.

The disease proceeds in two phases. In the first, systemic symptoms are characteristic: fatigue, loss of weight, headache, pain in the chest and extremities, anaemia, oedema, fever, dyspnoea, tachycardia, clubbing of fingers, exanthem, pericarditis, iridocyclitis [8, 19, 23, 29, 35, 52]. This stage is followed by the obliterative phase in weeks or months, the symptoms greatly depend on the localization of obliteration. In our patient the occlusive symptoms developed about one month after the general complaints such as fever, chest and extremity pain, clubbing of fingers etc. had presented themselves.

If the aortic arch and the arteries arising from it are involved in the process, the term aortic arch syndrome is justified. The pulse is weak or unpalpable, blood pressure is unmea-

surable and oscillations are sharply reduced in the affected extremity. The extremity is weaker, colder and thinner than its counterpart. In our patient at the first admission the most striking finding was the unmeasurable blood pressure, the unpalpable pulse on the right arm with arterial hypertension on the left arm. When the carotid arteries are involved, symptoms of internal carotid occlusion may appear, and trophic disturbances such as loss of hair, necrosis of the nasal septum, fatigue of the masticatory muscles (so-called masticatory claudication) and hypoxic eye symptoms with transitory impairment of vision (visual claudication), iritis, iridocyclitis, cataract, microaneurysms of the eyeground arterioles, *de novo* formation of retinal blood vessels, ocular hypertension. Involvement of the lower extremities as seen in our patient, is rather unusual. Among the cases occurring in childhood, involvement of the abdominal aorta is frequent, most child patients have therefore hypertension [7, 12, 18, 23, 35, 52]. This in turn causes cardiomegaly, heart failure and eyeground symptoms in most patients.

The diagnosis can be confirmed by aortography.

In our patient blood pressure was elevated on the left arm from the beginning. This then became more pronounced and led to encephalopathy. Initially the eyeground was normal, but later the signs of arterial hypertension developed.

Primary aortitis is more frequent in Eastern countries. Nasu [31] collected 1844 cases from Japanese hospitals. Of the patients, 85 to 90% were women and only 3—4% were children, 17 were younger than 10 years and 151 were between 10 and 20 years at onset of the first symptoms. Child cases were reported from Japan, Korea, Thailand, India, Singapore, Mexico, Chile, USA, Australia, Africa, the Soviet Union and Germany [7, 10, 12, 17, 18, 22, 23, 25, 26, 30, 32, 33, 35, 37, 40, 41, 42, 43, 47, 51, 52, 53]. Among the children, 85—90% were girls. The youngest patient was 9 months old [53].

The aetiology of the disease is a a matter of controversy. The tuberculous and the autoimmune origin has the highest number of believers. Many patients had had tuberculosis in their history, in some cases the tuberculous process was still active at the time of the onset of aortitis. On the other hand the tuberculous origin of the disease cannot be accepted since the diseased arteries show no sign of tuberculosis and in the vascular wall no acid fast bacteria can be demonstrated (this happened only once, in the case cited in reference 22). In the countries with a high incidence of aortitis tuberculosis is rather frequent their coexistence may thus be a coincidence. If tuberculosis has any role in the pathogenesis it is exerted by some allergic mechanism [21, 22, 26, 52]. Others think that primary aortitis is an autoimmune disease independent of tuberculosis [16, 20, 23, 27, 29, 36, 50, 55].

In patients affected by aortitis the laboratory findings point to an auto-

immune origin: the erythrocyte sedimentation rate is accelerated in the active phase of the disease, the level of alpha$_2$ and gamma globulin, of IgG and IgM is usually elevated, mild anaemia is frequent and a moderate increase of the leucocyte count is the rule [2, 5, 14, 19, 23, 29, 36, 44, 52].

Primary aortitis may be associated with other autoimmune disorders like rheumatoid arthritis [15, 24, 25, 38], polymyositis [28], glomerulonephritis [54], ulcerative colitis [5], terminal ileitis [3], ankylosing spondylitis [34], and systemic lupus erythematosus [24].

Immunity against the vascular wall has been investigated by several authors with various results. Many [2, 14, 39, 44] were unable to demonstrate antibodies against the wall of aorta or large arteries in the serum of their patients. Others [16, 20, 27, 29, 48, 50, 55] succeeded in finding such antibodies. In addition to differences in the methods, it may be anticipated that the patients were in different stages of the disease. It may also be supposed that the aetiology of the disorder is not quite homogeneous.

At the first admission of our patient, ESR, and the serum levels of alpha$_2$ and gamma globulin were elevated. During the most active phase of autoimmune processes, markedly increased IgG, moderately increased IgM and IgA, increased levels of acute phase proteins in the presence of normal complement levels, were encountered. Studies of immune reactivity against vascular wall performed in this stage showed an exaggerated immune response to aortic and venous wall antigens, both cellular and humoral. The level of circulating immune complexes was slightly increased.

Treatment led to some improvement but then a relapse ensued. In this period the IgG level was a multiple of the normal, the acute phase proteins, however, gave a normal value. The cellular reaction to the vascular wall could not be demonstrated while the humoral response to aortic wall was markedly augmented and the level of circulating immune complexes showed an increase over the previous values. The immunological findings therefore suggested an autoimmune origin.

The therapy of primary aortitis is an unresolved problem. Some think that steroid treatment is ineffective [5, 23, 26, 41], others reported on good results with corticosteroids [1, 8, 14, 20, 29, 32, 50]. Antimetabolite treatment has been thought to be effective but experience with it is scarce [11, 54]. A good effect can be expected in the early phase of the disease thus an early diagnosis is of importance [19 44].

In our case the disease showed rapid progress as long as no treatment was given. Corticosteroid and antimetabolite therapy resulted in improvement, the laboratory parameters improved, the aortitis became inactive. The previously impalpable pulse on the right arm reappeared but this had to be ascribed to an improvement of the collateral circulation since the right subclavian artery showed no

pulsation. Three months after termination of the first course of treatment there was a relapse as expressed by the increased ESR and gamma globulin level. A second course of immunosuppressive treatment led to disappearance of the signs of activity without decreasing the blood pressure. This then decreased on captopril treatment.

Severe arterial occlusions may be treated surgically, but the results are questionable and this kind of treatment does not resolve the underlying disease; progression of the latter may abolish the beneficial effect of surgery. Therefore, surgery is indicated only if there occur severe functional disturbances [8, 20, 26], especially in connection with stenosis of the aorta or the renal arteries. After unilateral constriction of the renal artery, nephrectomy may abolish hypertension but later the other renal artery may become involved and the hypertension will return [7]. Since in our case the abdominal aorta was involved together with both renal arteries, there is no chance for reconstructive surgery.

The prognosis of primary aortitis cannot be predicted exactly. Out of the 1844 patients of Nasu [31] 100 died, but only one out of the 17 patients under 10 years. The prognosis seems to be independent of age, laboratory findings or localization; it is said to be favourable if there is no complication or only one of mild degree (retinopathy, hypertension, aortic regurgitation or aneurysm [9]. With severe or multiple complications, 30% of the patients die within 5 years.

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References

1. Ask Upmark E: On the "Pulseless disease" outside of Japan. Acta Med Scand 149:161, 1954
2. Asherson RA, Asherson GL, Schrire V: Immunological studies in arteritis of the aorta and great vessels. Br Med J 3:589, 1968
3. Bigot R, Cateau P, Chermet J, Doyon D, Monnier JP: Iléite terminale et maladie de Takayasu. Ann Radiol (Paris) 16:767, 1973
4. Boyden SV: The adsorption of proteins on erythrocytes treated with tannic acid and subsequent hemagglutination by antiprotein sera. J Exp Med 93:107, 1951
5. Chapman R, Dawe C, Whorwell PJ, Wright R: Ulcerative colitis in association with Takayasu's disease. J Am Dig Dis 23:660, 1978
6. Cheitlin MMD, Carter CPB: Takayasu's disease. Unusual manifestations. Arch Intern Med 116:283, 1965
7. Danaraj TJ, Wong HO, Thomas MA: Primary arteritis of aorta causing renal artery stenosis and hypertension. Brit Heart J 25:153, 1963
8. Fraga A, Mintz G, Valle L, Flores Izquierdo G: Takayasu's arteritis: Frequency of systemic manifestations (study of 22 patients) and favorable response to maintenance steroid therapy with adrenocorticosteroids (12 patients). Arthritis Rheum 15:617, 1972
9. Füst G, Szondy É, Székely J, Nánai I, Gerő S: Studies on the occurrence of circulating immune complexes in vascular diseases. Atherosclerosis 29:181, 1978
10. Gelfand M: Case reports: Giant cell arteritis with aneurismal formation in an infant. Br Heart J 17:264, 1959

11. Gilligan BS: Brachiocephalic arteritis in a young female (Takayasu's disease). Clin Exp Neurol 14:100, 1977
12. González-Cerna JL, Villavicencio L, Molina B, Bessudo L: Nonspecific obliterative aortitis in children. Ann Thorac Surg 4:193, 1967
13. Hasková V, Kaslik J, Riha I, Matl I, Rovensky J: Simple method of circulating immune complex detection in human sera by polyethylene glycol precipitation. Z Immunitaetsforsch 154:399, 1978
14. Hirsch MS, Aikat BK, Basu AK: Takayasu's arteritis. Report of five cases with immunological studies. Bull Johns Hopkins Hosp 115:29, 1964
15. Hope-Ross P, Bien EJ, Palladino VS, Graham G: Rheumatoid arthritis. Report of an unusual case. Ann Int Med 52:682, 1960
16. Ikeda M: Takayasu's disease. Immunological studies. Jpn Circ J 30:87 1966
17. Isaacson C, Klachko DM, Wayburne S, Simson JW: Aortitis in children. Lancet 2:542, 1959
18. Isaacson C: An idiopathic aortitis in young Africans. J Pathol 81:69, 1961
19. Ishikawa K: Natural history and classification of occlusive thromboaortopathy (Takayasu's disease). Circulation 57:27, 1978
20. Ito I: Aortitis syndrome with reference to detection of anti-aorta antibody from patients' sera. Jpn Circ J 30:75, 1966
21. Jellinek H, Littmann I, Süle É, Földi M, Máthé Z: Takayasu's disease and tuberculosis. Acta Med Acad Sci Hung 16:5, 1960
22. Kinare SC: Nonspecific aortitis (Takayasu's disease). Autopsy study of 35 cases. Path Microbiol 43:134, 1975
23. Lee K, Sohn K, Hong C, Kang S, Berg K: Primary arteritis (Pulseless disease) in Korean children. Acta Paediatr Scand 56:526, 1967
24. Lesoff MH, Glynn LE: The pulseless syndrome. Lancet 1:799, 1959
25. Lomas RW, Bolande RP, Gibson WM: Primary arteritis of the aorta in a child. Am J Dis Child 97:87, 1959
26. Lupi-Herrera E, Sánches-Torres G, Marcushamer J, Mispireta J, Horwitz S, Vela JE: Takayasu's arteritis. Clinical study of 107 cases. Am Heart J 93:94, 1977
27. Maekawa M, Ishikawa K: Immunological studies of aortitis syndrome. Jpn Circ J 30:79, 1966
28. Miller GAH, Thomas ML, Medd WE: Aortic arch syndrome and polymyositis with LE cell in peripheral blood. Br Med J 1:771, 1962
29. Nakao K, Ikeda M, Kimata S, Niitami H, Miyakora M, Ishimi Z, Hoshiba K, Takeda Y, Ozawa T, Matsushita S, Kuramochi M: Takayasu's arteritis. Clinical report of eighty four cases and immunological studies of seven cases. Circulation 35:1141, 1967
30. Nasu T: Pathology of pulseless disease. A systemic study and critical reviews of 21 autopsy cases reported in Japan. Angiology 14:225, 1963
31. Nasu T: Takayasu's truncoarteritis in Japan. A statistical observation of 76 autopsy cases. Path Microbiol 43:140, 1975
32. Numano F, Isohisa I, Kishi U, Arita M, Maezawa H: Takayasu's disease of twin sisters. Possible genetic factors. Circulation 58:173, 1978
33. Okumi M, Iijima MD, Kobayashi N, Saity I: Primary arteritis in children: Report of three cases. Paediatr Univ Tokio 7:32, 1962
34. Paloheimo JA, Julkunen H, Siltanen P, Kajander A: Takayasu's arteritis and ankylosing spondylitis. Acta Med Scand 179:77, 1966
35. Paton BC, Chartikavanij K, Buri P, Prachuabmoh K, Jumbala MRB: Obliterative aortic disease in children in the tropics. Circulation 31:(Suppl I) 197, 1965
36. Riehl JL, Brown JB: Takayasu's arteritis. An autoimmune disease. Arch Neurol 12:92, 1965
37. Roberts WC, Wibbin EA: Idiopathic panaortitis, supra-aortic arteritis, granulomatous myocarditis and pericarditis. Am J Med 41:453, 1966
38. Sandring H, Welin G: Aortic arch syndrome with special reference to rheumatoid arthritis. Acta Med Scand 170:1, 1961
39. Sano K, Saito I: Immunological studies of pulseless disease. Neurol Med Chir (Tokyo) 8:28, 1966
40. Saveleva LA, Sivkova A: Pulseless disease in childhood. Pediatrija 42:67, 1963
41. Sen PK, Kinare SG, Kulkarni TP, Parulkar GB: Stenosing aortitis of unknown etiology. Surgery 51:317, 1962
42. Sen PK, Kinare SG, Engineer SD, Parulkar GB: The middle aortic syndrome. Br Heart J 25:610 1963
43. Steeger A, Vargas L: Enfermedad de Takayasu. Rev Chil Pediatr 31:377, 1960
44. Strachan RW, Wigzell FW, Anderson JR: Locomotor manifestations and serum studies in Takayasu's arteriopathy. Am J Med 40:560, 1966

45. Szabó G, Szegedi Gy, Fekete B, Petrányi Gy: The investigation of cellular immune reactivity by leukocyte migration test. Orv Hetil 113:134, 1972
46. Szondy É, Jobbágy A, Link E, Gerő S: Immunological studies with aortic and venous tissue antigens I. The antigen structure of vascular tissues. Arterial Wall 3:81, 1975
47. Ueda H, Ito I, Saito Y: Studies on arteritis, with special reference to pulseless disease and its diagnosis. Naika (Japan) 15:239, 1965
48. Ueda H, Saito Y, Ito I, Yamaguchi H, Sugiura M, Morooka S: Immunological studies of aortitis syndrome. Jpn Heart J 8:4, 1967
49. Ueda H, Saito Y, Morooka S, Ito I, Yamaguchi H, Sugiura M: Immunological studies of aortitis syndrome. Jpn Heart J 9:73, 1968
50. Ueda H, Saito Y, Yamaguchi H, Takeda T, Morooka S, Ito I: Further immunological studies of aortitis syndrome. Jpn Heart J 12:1, 1971
51. Wagenvoort CA, Harris LE, Brown AL, Veeneklaas GMH: Giant cell arteritis with aneurism formation in children. Pediatrics 32:861 1963
52. Warshaw JB, Spach MS: Takayasu's disease (primary aortitis) in childhood. Case report with review of literature. Pediatrics 35:620, 1965
53. Westphal J, Löbbecke F, Mietens C: Arteritis der von Aorta abgehenden großen Gefäße (Takayasu-arteritis) im Säuglingsalter. Klin Paediatr 188:570, 1976
54. Zillulero GE, Ferrer P, Garcia OL, Moore M, Pardo V, Strauss J: Takayasu's arteritis associated with glomerulonephritis. A case report. Am J Dis Child 132:1009, 1978
55. Yoshitoshi Y, Seki K, Susuki H, Masuyama K, Yamana Y, Koida K, Eto S: Immunological studies in Takayasu's disease. Jpn J Intern Med 22:1450, 1968

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