Case Report

A rare case of carotid stroke in young due to Takayasu Arteritis with positive anti-cardiolipin antibodies – A case report

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Abstract

Takayasu arteritis is a well-known yet rare form of large vessel vasculitis. Takayasu arteritis affects mainly women, and is most commonly seen in Japan, South East Asia, India, and Mexico, where it usually presents in the 2nd or 3rd decade of life. It is seen usually as pulseless disorder often with bruit at the stenosed arteries. Manifestations range from asymptomatic disease, to catastrophic strokes. Angiography remains the gold standard for diagnosis. Approximately half of those patients treated with steroids will respond, and half of the remaining patients respond to methotrexate; mycophenolatemofetil may be useful. Fertility is not adversely affected and pregnancy does not appear to exacerbate the disease, although management of hypertension is essential. We herewith report a rare case of an11 year old girl, who presented with left-sided hemiparesis, dysarthria, left UMN facial palsy, feeble pulses on right side, high Blood Pressure recordings and positive anti-cardiolipin antibodies. Imaging studies revealed Occluded right Common carotid artery, occluded right subclavian artery and stenotic right renal artery and MRI showed Acute Infarcts in Right Basal Ganglia and Right High Parietal Region, Hemorrhagic infarct in right MCA subcortical area.The diagnosis of Takayasu arteritis with recent cerebrovascular accident (left hemiparesis) with hypertension was made and the patient was started on steroids, anti-platelets, anti-hypertensives and physiotherapy.

Key words

Stroke in Young, Takayasu Arteritis, Anti-Cardiolipin Antibodies.

Introduction

Stroke occurs when blood flow to the brain is diminished, resulting in dysfunction in one or many parts of the brain. The two main types of stroke include ischemic, due to lack of arterial blood flow to the brain, and hemorrhagic due to bleeding (intracerebral hemorrhage). Although a majority of strokes occur in people aged above 65 years, when it occurs to an adult between the age of 18 and 65 years, it is referred to as a young stroke. A stroke may also occur in children, and its causes may be different [1]. It includes TA (Takayasu Arteritis).

Incidence

Takayasu arteritis is rare, but most commonly seen in Japan, South East Asia, India, and Mexico. In 1990, it was included in the list of intractable diseases maintained by the Japanese government, [2] and to date 5000 patients have been registered. Hall, et al. found the incidence to be 2.6/million/year in North America [3]. The UK incidence is unknown.Stroke affects approximately 5% to 15% of people with TA [4]. Takayasu arteritis is a well-known yet rare form of large vessel vasculitis.

Clinical features

The clinical features have been well documented by cohort studies of over 570 patients from different countries [3, 5, 6, 7, 8, 9, 10, 11, 12]. Manifestations range from asymptomatic disease found as a result of impalpable pulses or bruits, to catastrophic neurological impairment. The two phases are described 1) A “pre-pulseless” phase characterised by non-specific inflammatory features, followed by 2) A chronic phase with a) the development of vascular insufficiency, in few cases with intermittent flares. [3]. As the inflammation progresses and stenoses develop, the more characteristic features become apparent, influenced by the development of collateral circulation. Stenotic lesions predominate and tend to be bilateral. Nearly all patients with aneurysms also have stenoses and most have extensive vascular lesions [8, 9]. Takayasu arteritis appear to be very similar to those of adults [11]. Non-specific features include fever, night sweats, malaise, weight loss, arthralgia, myalgia, and mild anemia [3].

Diagnosis and differential diagnosis

From the more typical features of Takayasu’s arteritis, the American College of Rheumatology (ACR) defined specific diagnostic criteria for this disorder in 1990 (**Table - 1**) [13].

Angiography remains the gold standard for diagnosis. Doppler ultrasound is a useful non-invasive procedure for the assessment of vessel wall inflammation. In view of the vessels involved, histological diagnosis is usually impractical and histological assessment is limited to those cases undergoing revascularization procedures.

The differential diagnoses include other causes of large vessel vasculitis: inflammatory aortitis (syphilis, tuberculosis, lupus, rheumatoid arthritis, spondyloarthropathies, Behçet’s disease, Kawasaki disease, and giant cell arteritis); develop- mental abnormalities (coarctation of the aorta and Marfan syndrome), and other aortic pathologies, such as ergotism and neurofibromatosis. Most of these have specific features that enable diagnosis, but tuberculosis has remained an important differential and possible etiological factor. However, tuberculous aortitis tends to cause erosion of the vessel wall with the formation of true or false aneurysms, particularly affecting the descending thoracic and abdominal aorta. Dissection and rupture are important complications rather than the stenoses typical of Takayasu arteritis. The incidence of rupture and bleeding complications of aneurysmal Takayasu arteritis is low. Syphilis tends to affect an older age group, with calcification, sparing the

descending thoracic aorta, and stenoses are not a feature [8]. Hypertension as a result of fibromuscular dysplasia is an important differential diagnosis.

Classification

An attempt has been made to classify the disease on the basis of angiographic findings. The early system, revised by Lupi-Herrera et al in 1977 [5], has been superseded by the new classification of Takayasu arteritis (**Table - 2**) [10]. These systems are useful in that they allow a comparison of patient characteristics according to the vessels involved and are helpful in planning surgery.

Table – 1: 1990 ACR criteria for the classification of Takayasu arteritis [13].

Criterion	Definition
Age at disease onset <40 years	Development of symptoms or findings related to Takayasu arteritis at age <40 years
Claudication of extremities	Development and worsening of fatigue and discomfort in muscles of 1 or more extremity while in use, especially the upper extremities
Decreased brachial artery pulse	Decreased pulsation of 1 or both brachial arteries
Blood pressure difference >10 mm Hg	Difference of >10 mm Hg in systolic blood pressure between arms
Bruit over subclavian arteries or aorta	Bruit audible on auscultation over 1 or both subclavian arteries or abdominal aorta
Arteriogram abnormality	Arteriographic narrowing or occlusion of the entire Aorta, its primary branches, or large arteries in the proximal upper or lower extremities, not caused by arteriosclerosis, fibromuscular dysplasia, or similar causes; changes usually focal or segmental

A diagnosis of Takayasu arteritis requires that at least 3 of the 6 criteria are met.

Table – 2: New angiographic classification of Takayasu arteritis, Takayasu conference 1994 [10].

Type	Vessel involvement
Type I	Branches from the aortic arch
Type IIA	Ascending aorta, aortic arch and its branches
Type IIB	Ascending aorta, aortic arch and its branches, thoracic descending aorta
Type III	Thoracic descending aorta, abdominal aorta, and/or renal arteries
Type IV	Abdominal aorta and/or renal arteries Type V , Combined features of types IIb and IV

According to this classification system, involvement of the coronary or pulmonary arteries should be designated as C (+) or P (+), respectively.

Table – 3: Ishikawa clinical classification of Takayasu arteritis [6].

Group	Clinical Features
Group I	Uncomplicated disease, with or without pulmonary artery involvement
Group II A	Mild/ moderate single complication together with uncomplicated disease
Group II B	Severe single complication together with uncomplicated disease
Group III	Two or more complications together with uncomplicated disease

The natural history of any disorder can only be elucidated by following patients in the absence of specific treatment. Ishikawa defined clinical groups based on the natural history and complications of the disease (**Table – 3**) [6]. The four most important complications were defined as Takayasu retinopathy, secondary hypertension, aortic regurgitation, and aneurysm formation, each being graded as mild/moderate or severe at the time of diagnosis. Four grades of disease are described

Histology, immunology, and pathogenesis

Macroscopically, in the chronic phase, the aorta is thickened secondary to fibrosis of all three vessel layers. The lumen is narrowed in a patchy distribution, often affecting multiple areas. If disease progression is rapid, fibrosis can be inadequate with subsequent aneurysm formation. The intima may have a "tree bark" appearance, a feature common in many cases of aortitis [14].

Microscopically, the vasculitis may be divided into two phases 1). an acute florid inflammatory phase and 2). a healed fibrotic phase. In the acute phase a vasa vasoritis is seen in the adventitia. The media is infiltrated by lymphocytes and occasional giant cells with neovascularisation. Mucopolysaccharides, smooth muscle cells, and fibroblasts thicken the intima. In the chronic phase there is fibrosis with destruction of elastic tissue. Similar histopathological findings are also seen in giant cell arteritis; therefore, biopsy results may not differentiate between these two. Clinical features usually allow correct diagnosis, [15] but difficulties are faced in older patients with Takayasu arteritis, as the timing of disease onset is not known.

Infection has been considered to play a role in the pathogenesis of Takayasu arteritis. Tuberculosis has remained an important differential and possible etiological factor. Tuberculosis has been particularly implicated in view of the high prevalence of infection, past or present, in affected patients. [16]. Hoffman GS, in a study [17] reported in 1998 concluded that no known serological test was able to supplant vascular histopathology in determining disease activity.

Cardiolipin (CL) is a unique phospholipid which is localized and synthesized in the inner mitochondrial membrane (IMM) [18]. Anti-cardiolipin antibodies are antibodies against cardiolipin found in various diseases like syphilis, antiphospholipid syndrome, livedoid vasculitis, vertebrobasilar insufficiency, Behçet's syndrome and systemic lupus erythematosus. The role of anti-cardiolipin antibodies (aCLs) as novel risk factors for ischemic stroke and transient ischemic attacks (TIAs) has been a matter of debate. Prior cohort studies included only selected subjects, mostly men. Elevated serum concentrations of aCLs, independently of other cardiovascular risk factors, significantly predict the risk of future ischemic stroke and TIA in women but not in men [19].

Case report

An 11 year old girl presented with sudden onset of weakness of left Upper Limb and Lower Limb since one day duration.

History of presenting illness - Patient was apparently asymptomatic a day earlier, when she noticed sudden onset of weakness of left Upper Limb& Lower Limb while she was playing in her school. History of deviation of mouth to right +, History of slurring of speech +, No history of headache or vomiting. No history of loss of consciousness or seizures/ head injury, No history of visual disturbances, No history of fever/ arthralgia or rash.

Past history - No history of similar complaints in the past, No history of HTN/ DM/ CAD/ CVA/ TIA/ CRHD in the past.

Personal history - Mixed diet, Normal appetite, Adequate sleep, Bowel and bladder movements regular, No addictions, No known drug allergies,

Family History - Not significant

General Examination

Patient was conscious, coherent and cooperative. There was no pallor/ icterus/ cyanosis/ jaundice/ clubbing/ pedal edema or lymphadenopathy

Vitals; Temperature - 98.4 F, PR-86/min, regular, Right radial, ulnar, brachial pulses were feeble

No subclavian bruit, BP - Right Upper Limb 100/90 mm Hg systolic, Left Upper Limb 164/90 mmHg

Systemic Examination-

CVS - S1, S2 +, Carotid bruit heard over Rt. carotid

Lungs - Clear

PA - Soft, No renal bruit

CNS - Intellectual functions: - Patient was conscious/coherent/co-operative, oriented to time, place and person. Memory - Intact. Speech – dysarthria +.

Cranial nerves - all cranial nerves normal except Left UMN Facial palsy +

Pupils NSRL

Fundus – Revealed dilatation of central retinal artery and vein, no micro aneurysms

No signs of meningeal irritation

Motor system

Bulk of the muscles was normal and equal on both sides, There was hypotonia on left sided limbs and power was '0' on left sided limbs. Deep tendon reflexes were lost in left sided limbs and plantars was extensor on left side

Cerebellar functions: No titubation, no nystagmus, no pendular knee jerk, no dysdiadokinesia, no past pointing, finger nose test was normal on right side and could not be tested on left side. Gait – could not be tested because of '0' power

Sensory system – all modalities of sensations werenormal, Peripheral nerves – not thickened, Skull and spine: Normal

Investigations

CBP - Hb - 11.4 gm/dL, PCV - 35%, TLC - 10500/mm3, Platelet count - 3.6 lakh/mm3, T – 2 to 7 min, CT - 5 to 11 min, Sickling Test-Negative, Peripheral Smear-Normal, Viral markers – HBS Ag – negative, HIV –negative, Anti HCV antibodies – negative, Lipid profile - Total Cholesterol - 129 mg/dL, HDL - Cholesterol 45 mg/dL, LDL Cholesterol - 74 mg/dL, VLDL Cholesterol - 10 mg/dL, Triglycerides - 50 mg/dL, Total Cholesterol/ HDL ratio - 3, Iron studies - Homocysteine- 18 mcmol/L, Iron – 25 mcg/Dl, %TIC Saturation-4%, Ferritin – 14 ng/mL, TIBC - 699, Renal function tests - Blood Urea – 21 mg/dL, Serum Creatinine - 0.6 mg/dL, Liver function tests - S. Bilirubin (total) - 0.8 mg/dl, S. Total Proteins – 7.50 mg/dl , S. Albumin – 4.2 mg/dl, SGOT- 33 IU/L, SGPT – 32 IU/L , S. Alkaline Phosphatase – 611 IU, Serum electrolytes – sodium - 136 meq/L, potassium - 4.3 mEq/L, chloride - 100 meq/L, Random Blood Sugar - 97 mg/dL, Prothrombin time - 13.5 sec, Activated partial thromboplastin time - 24 sec, CUE - Normal, ECG - normal, CXR- NAD

Immunology Report – ANA - negative, Anti ds DNA - negative, Anti PR3 - negative, Anti MPO - negative, Cardiolipin Ab IgA - 1.5 u/ml (neg) (N - < 12 u/ml), CardiolipinAb, IgG - 2.0 u/ml (neg) (N - < 20 GPL U/ml), CardiolipinAb – IgM - 15 /ml (Positive) (N < 13MPL U/ml), Beta 2 Glycoprotein IgGAb - 0.46 u/ml (neg) (N - <12 u/ml), Beta 2 glycoprotein IgM ab-0.2 u/ml8 (neg) (less than 12 u/ml), Lupus anticoagulant - 29.50 secs (n - 39.20 secs), Factor 5 Leiden mutation – RT PCR not detected, Protein C - 82% (N -70-140), Protein S - 88.3% (N - 63.5-149)

MRI brain findings (**Figure - 1**) - Acute Infarcts in right basal ganglia and right high parietal region, Hemorrhagic infarct in right MCA subcortical area.

MRA - Right ICA, MCA show narrowed caliber throughout the course. Paucity of distal branches of right MCA (**Figure – 2, 3**).

2D ECHO Report - Global Hypokinesia of LV with mild LV dysfunction, EF -50%, No MR/AR/TR, No LA/LV Clot, No pericardial effusion.

TEE - Intact IAS/IVS, Normal valvular Echos, Mild LV dysfunction, No intracardial thrombus / mass, Trivial MR / AR+. Proximal aorta up to arch was normal.

Figure – 1: MRI – Shows Acute Infarcts in right basal ganglia and right high parietal region, Hemorrhagic infarct in right MCA subcortical area.

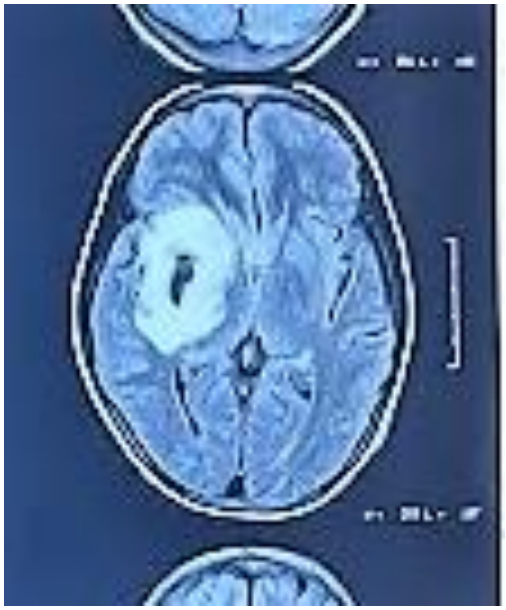


Figure – 2: MRA - Right ICA, MCA show narrowed caliber throughout the course. Paucity of distal branches of right MCA.

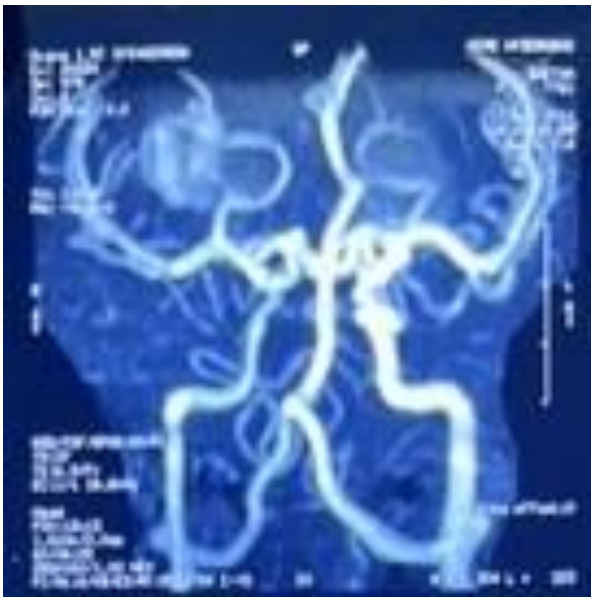
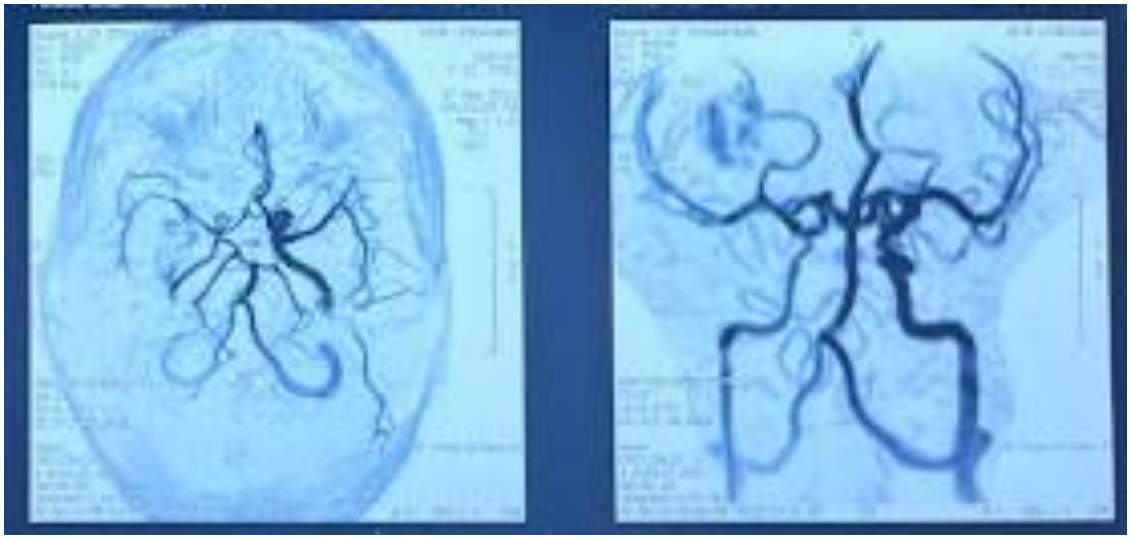


Figure – 3: MRA - Right ICA, MCA show narrowed calibrethroughout the course. Paucity of distal branches of right MCA



Color Doppler Report- Near total thrombosis with central (minimal) recanalization of right proximal CCA with altered dampered monophasic waveforms in right distal CCA, ECA, ICA. Left CCA ICA and ECA were normal. Right VA could not be visualized.

CT angiogram of aorta -
Occluded right Common carotid artery from origin up to the distal segment just before bulb; from which it's filling through collaterals from Left ECA branches, and thereafter ECA & ICA are small in Calibre. Wall thickening of CCA. Right Subclavian artery origin is occluded, and filling distally from collateral circulation. Right vertebral artery origin appears normal. S/o Subclavian steal. Coeliac axis origin is normal with distal dilatations. Superior mesenteric artery origin is occluded, filling distally from collaterals of Inferior mesenteric and prominent artery of

Drummond and mesenteric vessels. Renal vessels - Single right renal artery with significant narrowing of proximal segment and distal dilatation.Single renal artery noted at left side with mild stenosis at origin. Narrowed suprarenal segment of aorta with Thickened wall.

Collateral vessels from the left subclavian, descending and abdominal aorta with prominent intercostal, lumbar vessels and collaterals to posterior paraspinal muscles.

P/o Takayasu's Arteritis.

Diagnosis

Takayasu arteritis with Carotid Stroke with Right CCA thrombus, Right MCA subcortical Hemorrhage infarct, left hemiparesis, hypertension with positive anti cardiolipin antibodies.

Treatment given

Tab Wysolone 20 mg 1 TID
Tab Nimodip 30 mg 2 TID
Inj Ampicillin 500mg IV QID
InjAmikacin 500 mg IV BD
InjMetrogyl 500 mg IV TID
Tab. Stamlo 5mg OD
InjOptineuron 1 amp IV OD
Physiotherapy

Discussion

Takayasu arteritis (TA) is a rare inflammatory vasculopathy, affecting the aorta and its major branches and is a potential cause of stroke in young adults [20]. Takayasu arteritis (TA) is a giant cell arteritis usually affecting young women and characterized by inflammatory and ischemic signs of large vessel involvement, including extracranial cerebral arteries [21].

Characteristic features

Findings on physical examination include hypertension, loss or inequality of pulses in the upper extremities, asymmetric blood pressures between the upper extremities, or bruits heard over the neck and supraclavicular region. The loss of palpable pulses in the upper extremities gives rise to another name for TA–pulseless disease [20]. Our patient also had hypertension, different blood pressure recordings in upper limbs and also had carotid bruit.

Hypertension in most patients is due to the renal artery stenosis. Takayasu retinopathy is seen in 1/3rd of patients. Aortic regurgitation resulting from dilatation of the ascending aorta, separation of the valve leaflets, and valve thickening in 1/4 of patients Congestive cardiac failure associated with hypertension, aortic regurgitation, and dilated cardiomyopathy. Neurological features secondary to hypertension and/or ischemia, including postural dizziness, seizures, and amaurosis. 14–100% of patients will have Pulmonary artery involvement. Oligaemic lung fields on chest x ray correlate with pulmonary vasculopathy in 1/3rd of cases. Pulmonary artery disease, and the pattern of arterial involvement is useful to confirm the diagnosis of Takayasu arteritis. Other symptoms include dyspnea, headaches, carotodynia, myocardial ischemia, chest wall pain, and erythema nodosum [3, 5, 6, 8, 9, 11, 22].

The disease commonly presents in the 2nd or 3rd decade of life. In one of the largest cohorts (n = 107) 80% of patients were between 11 and 30 years, 77% had disease onset between the ages of 10 and 20 years, with time from onset of symptoms to diagnosis of two to 11 years in 78%. A study of 88 patients from India [8], gave a mean (SD) age at symptom onset of 24.0 (8.8) years and mean (SD) age at diagnosis of 28.3 (9.9) years. Kerr, et al. suggested that the delay in diagnosis was longer in juveniles, being up to four times that of adult patients [9]. However, data from India [11], shows that the patients aged under 18 years demonstrated a delay of only 2.5 to 5.5 months. This discrepancy presumably relates to the difference in disease incidence between the two populations, which results in differences in awareness. The clinical features and progress of young patients with variable disease presentation between different populations is well illustrated by Moriwaki, et al.

in their study of Indian and Japanese patients [10].

The Japanese patients (n = 80) were predominantly female (96%), presenting with dizziness, vertigo, pulselessness, more prolonged and severe inflammation, and more aortic regurgitation, reflecting involvement of the aortic arch and its main branches. This contrasted with the Indian patients (n = 102), 37% of whom were male. They tend to present with headache, hypertension, and left ventricular hypertrophy as a result of vasculitis affecting the abdominal aorta and renal vessels. However, most patients in both countries had diffuse disease.

Although similar in many respects, including aortic involvement in 10–15% of patients with giant cell arteritis, Michel, et al. [16] suggest that giant cell arteritis and Takayasu arteritis can be differentiated on clinical grounds. In a study of 280 patients, 217 with giant cell arteritis and 63 with Takayasu arteritis identified through the ACR vasculitis criteria data bank, they found that age of 40 years at disease onset was the single most discriminatory factor. Excluding age from the analysis, ethnic background and clinical signs of upper limb vascular insufficiency, shoulder stiffness, and scalp tenderness were variables that led to correct diagnoses in 95% of patients [15].

Ishikawa retrospectively studied 54 Japanese patients over six months to 18 years of follow up between 1957 and 1975. The overall five year survival rate after diagnosis was 83.1%. Seven patients died within five years of diagnosis, all were in groups IIB and III, and deaths were mostly from cerebrovascular disease and congestive cardiac failure. All patients with aortic regurgitation were in group III. The five year survival rate in combined groups IIB and III was 70%, compared with 100% in group I. Five acute events occurred in the survivors during follow up, three of five occurring in patients from groups IIB and III. No acute event occurred in patients from group I. Nineteen of the 54 patients were treated with steroids. The experience from India supports this classification for prognostic assessment [8]. Cumulative survival at five years after disease onset was 91%, after 10 years the figure was 84%, whereas event free survival figures were 74.9% and 64%, respectively. Patients with a single mild complication or no complication at diagnosis had a five year event free survival of 97%, compared with 59.7% in patients with a single severe or multiple complications. No deaths occurred in patients in groups I and IIA, whereas 19.6% of patients in groups IIB and III died during follow up, mostly from cerebrovascular disease and cardiac failure. Twenty two major non-fatal events occurred during follow up, with 20 of 22 occurring in groups IIB and III. In this study, 63 of 88 patients received no specific disease modifying treatment. Other studies, which have included patients treated more aggressively, give five year survival rates of 90–94% [3, 12]. Therefore, classification according to this system (**Table - 3**) appears to give useful prognostic information at diagnosis and may help to guide treatment.

Management

Young stroke survivors have substantial rates of modifiable vascular risk factors, which contribute significantly to stroke recurrence and mortality [23].

Medical Treatment

Steroids are the mainstay of treatment for Takayasu arteritis and reports of efficacy vary. This may relate to the stage of disease at which treatment is introduced and to the disease extent. As there is no single cytotoxic drug which appears to be better than any other in terms of efficacy, side effect profiles have been an important driving force in determining treatment.

Currently, the best evidence based treatments include steroids, to which 50% respond, and methotrexate to which a further 50% respond. The use of methotrexate as a steroid sparing drug is logical and safe. Twenty five percent of patients with active disease will not respond to current treatments and care should be taken not to expose these patients to the hazards of

prolonged immunosuppression in the absence of clinical benefit [4].

Apart from treatment of hemiparesis, physiotherapy has a vital role in preventing contractures and stiffness. The other important medical issues relate to the management of hypertension and the prevention and treatment of thrombosis. Hypertension can be particularly difficult, and worsened with the use of steroids. The use of ACE inhibitors requires careful monitoring in view of the frequency of renal artery stenosis [24].

Surgical treatment

Indications for surgery include 1.hypertension with critical renal artery stenosis, 2.extremity claudication, 3.cerebrovascular ischemia or critical stenoses of three or more cerebral vessels, 4.moderate aortic regurgitation, and 5.cardiac ischemia with confirmed coronary artery involvement. [9]. In general, surgery is recommended at a time of quiescent disease to avoid complications, which include restenosis, anastamotic failure, thrombosis, haemorrhage, and infection [3, 9]. Surgery may be unnecessary for aortic arch and splanchnic disease as a result of extensive collateral development [24]. The critical stenoses should be corrected to prevent stroke, with grafts originating from the ascending aorta. Renal artery involvement is treated by percutaneous transluminal angioplasty [25, 26]. Stent placement following angioplasty for ostial lesions, long segment lesions, incomplete relief of stenoses, and dissection is safe and effective [27]. Radical surgical treatment of thoracic aneurysms is recommended if technically possible because more palliative procedures fail to prevent recurrent aneurysm formation or to minimise risk of later surgery [28].

Long term follow up

Takayasu arteritis is a systemic vasculopathy which can progress to cause vital organ ischemia. Hence, long term follow up is recommended and for which better tools are required. So far the focus is on vascular imaging techniques, with non-invasive methods being the most appropriate. Doppler ultrasound is easily applied to extracranial vessels and can determine vessel wall thickness. Magnetic resonance angiography (MRA) is being investigated in the evaluation of large vessel vasculitides [29]. It provides high resolution detail of vessel wall thickness and lumen configuration, and allows the measurement of wall enhancement as a reflection of edema and active inflammation. Compared with the gold standard of conventional angiography, approximately 2% of stenosed arteries are overestimated as occluded on MRA. The reduction of enhancement on follow up is presumed to reflect reduced inflammatory activity. Therefore, MRA can be used increasingly as an accurate follow up tool. The management of patients with Takayasu arteritis can be problematic. There may be uncertainty with regard to the onset and course of the disease, a poor correlation between clinical assessment and disease activity, poor disease activity markers in peripheral blood, and a lack of useful treatment in up to 25% of patients with progressive disease. The risk of increased morbidity and mortality means that most patients who present will ultimately receive immunosuppression. The vasculitides, particularly those affecting small vessels, generally require aggressive treatment. The same may not be true of all patients with Takayasu arteritis despite the angiographic appearances. Cohort studies suggest a good prognosis for those with uncomplicated or monocomplicated disease. Thus, the temptation to immunosuppress such patients aggressively should be questioned. In contrast, early treatment of those with progressive complicated disease may lead to a better prognosis for this group. Because inflammation is a risk factor for atherosclerosis, 2 more atherosclerotic complications are likely in the longer term [2].

Conclusion

Takayasu arteritis is a well-known yet rare form of large vessel vasculitis. As the inflammation progresses and stenoses develop, the more characteristic features become apparent,

influenced by the development of collateral circulation. Takayasu arteritis affects mainly women, and is most commonly seen in Japan, South East Asia, India, and Mexico.

Manifestations range from asymptomatic disease found as a result of impalpable pulses or bruits, to catastrophic neurological impairment. No known serological test was able to supplant vascular histopathology in determining disease activity. The role of anti-cardiolipin antibodies (aCLs) as a novel risk factors for ischemic stroke and transient ischemic attacks (TIAs) is known. Elevated serum concentrations of aCLs, independently of other cardiovascular risk factors, significantly predict the risk of future ischemic stroke and TIA in women but not in men.

Tuberculosis has remained an important differential and possible etiological factor.

Angiography remains the gold standard for diagnosis. Steroids are the mainstay of treatment for Takayasu arteritis. Methotrexate; Mycophenolatemofetil may also be useful.The critical stenoses should be corrected to prevent stroke, with grafts originating from the ascending aorta. Renal artery involvement is treated by percutaneous transluminal angioplasty.

Stent placement following angioplasty for ostial lesions, long segment lesions, incomplete relief of stenoses, and dissection is safe and effective. Radical surgical treatment of thoracic aneurysms is recommended. Takayasu arteritis is a systemic vasculopathy which can progress to cause vital organ ischemia. Hence, long term follow up is recommended.

We herewith reporting a rare case of Takayasu arteritis with Carotid Stroke with Right CCA thrombus, Right MCA subcortical Hemorrhage infarct, left hemiparesis, hypertension with positive anti cardiolipin antibodies and treated with steroids and antihypertensives.

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