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Takayasu Arteritis Affecting Only Renal Arteries: A Rare Cause of Renovascular Hypertension

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ABSTRACT

Takayasu Arteritis (Ta) Is An Inflammatory Process Frequently Associated With Stenosis And Obliteration Of The Aorta And Its Primary Branches With Unknown Etiology. This Manifestation Can Lead To Renovascular Hypertension Then To Renal Failure. Renovascular Hypertension Is Generally Resistant To Medical Therapy And Often Requires Additional Invasive Management Strategies, Such As Angioplasty Or Surgical Bypass. We Will Present A Case Of 20-Year-Old Patient That Had Severe Hypertension Due To Takayasu Arteritis Affecting Only The Renal Arteries. Our Case Aims To Improve Awareness Of This Condition Among Clinicians Because Early Diagnosis And Treatment Are The Only Way To Avoid The Onset Of Serious Complications Of Hypertension And To Preserve Renal Function.

KEYWORDS: Takayasu Arteritis, Renal Hypertension, Young Age.

Introduction

Takayasu Arteritis (Ta), Also Known As Aortoarteritis And Pulseless Disease, Is A Rare Disorder. It Is A Chronic Granulomatous Vasculitis Of Unknown Cause And Affects Large-And Medium-Sized Arteries, Primarily The Aorta And Its Main Branches. It Can Lead To Progressive Stenosis, Occlusion, Or Aneurismal Transformation. Ta Can Involve The Renal Arteries, Resulting In Renal Artery Stenosis (Ras) And, Consequently, Renovascular Hypertension. Diagnosis Is Based On The Combination Of Suggestive Clinical Picture, Increased Levels Of Inflammatory Markers, And Diagnostic Imaging. Because Early Symptoms Often Are Nonspecific, Prompt Diagnosis Of Ta Often Is Challenging. In This Report, We Present The Case Of A 20-Year-Old Patient In Whom We Diagnosed Takayasu Arteritis While Investigating The Etiology Responsible For severe Hypertension.

Case Report

A 20 -Year-Old Man, With More Than 6 Months history Of headache Mainly Localized At The Retro-Orbital Area, Moderate In Intensity, Temporarily Releaved By Medications. He Also Had Complaints Of Palpitation And Fatigability .Currently, He Is Admitted In The Emergency Department Because Of hypertensive Crisis .The Family History Was Negative For Essential Hypertension And Cardiovascular Diseases. The Physical Examination Revealed High Blood Pressure (240/130 Mm Hg) In Both Arms. The Heart Rate Was 92 /Min. The Respiratory Rate Was 20 Breaths Per Minute And The Oxygen Saturation Was 98% While Breathing Ambient Air. Peripherals Pulses Were Palpable And Symmetric. There Were No Signs Of Volume Overload Manifested By Clear Chest And Absence Of Peripheral Edema .The Neurological Examination Was Unremarkable .No Other Pathological Signs Were Detected.

Ecg Shows Sinus Rhythm With Sign Of Left Ventricular Hypertrophy (Figure1). Echocardiography Revealed Moderate Left Ventricular Hypertrophy And No Coarctation.

Laboratory Investigations Revealed C-Reactive Protein (Crp) Level: 10 Mg/Dl (Reference Range, <0.7 Mg/Dl), Erythrocyte Sedimentation Rate (Esr): 8mm/H, Normal Kidney Function, Serum Creatinine Level: 0.5 Mg/Dlleukocyte Count: 90.5 × 10^3 /Ml, Urinalysis: Moderate Proteinuria, Without Hematuria Or Pyuria. Serum Potassium Levels Were Low And Varied Between 3and 3.4 Meq/.Rheumatoid Factor, Anti-Cardiolipin Ig M And G, Anti-Nuclear Antibody And Antiphospholipid Antibodies All Negative.

Ultrasound Showed A Small Left Kidney Measuring 8 Cm In The Longitudinal Axis With Decreased Parenchymal Thickness And Increased Echodensity. The Right Kidney In Normal Range With Normal Echodensity. Because Of Renal Size Asymmetry, The Possibility Of Renal Artery Stenosis Was Raised. Color Doppler Indicated Occlusion Of Right Renal Artery, Turbulent Flow And Spectral Analysis Suggested High Peak Velocity At Left Renal Artery And A Decreased Resistive Index In The Interlobular Arteries, Consistent With High-Grade Stenosis. The Resistive Index

Was Severely Decreased At The Left Side Due To The Proximal Stenosis Of Artery (0.3).

In Abdominal Tomography, Nothing Pathological Was Detected About The Surrenal Glands, But There Was Concentric Wall Thickening At The Abdominal Aorta That Was Consistent With Arteritis (Figure 2).

Mr Angiography Showed That The Arch Of Aorta, Major Branches And Carotids Were All Potent With Normal Calibration. At Infrarenal Abdominal Aorta, There Was Obvious Pathological Involvement, And Calibration Became Thinner. There Was Severe Stenosis Just At The Origin Of Left Renal Artery. Superior Mesenteric Artery And Celiac Trunk Were Potent.

Intra-Arterial Aortography Was Applied To The Patient, And Pathological Involvement Was Detected At Abdominal Aorta Just Below Renal Artery .The Calibration Became Thinner At That Segment And Returned To Normal Just Beyond Iliac Bifurcation. High-Grade Stenosis At The Origin Of Both Renal Arteries Was Seen, And Other Branches Of The Abdominal Aorta Were All Normal (Figure 3,4). Infectious Causes Of Vasculitis Were Excluded. Serologic Tests For *Borrelia* Species, Hepatitis B, And Syphilis Were Performed With Negative Results. Polymerase Chain Reaction For Mycobacterium Tuberculosis Was Negative. Thus,The Definitive Diagnosis Of Takayasu Arteritis Which Involves The Infrarenal Segment Of The Abdominal Aorta And Renal Arteries Was Made By Clinical And Imaging Data.

Treatment Based Of Methotrexate Was Introduced In A Dose Of 20 Mg/M2/Week, Hypertension Was Treated Using Two Medications For Hypertension, Namely Amlodipine 10 Mg /Day, Acebutolol 200 Mg/Day. In The Following Days, The Blood Pressure Values Decreased But Didn't Reach Normal Values (160/98 Mm Hg). 2 Months Later, After A Multidisciplinary Discussion, Balloon Dilatation And Stenting Were Applied To Save Right Stenotic Artery With Excellent Result And Efficient Control Of Hypertension. The Patient Is Still Being Followed At Nephrology And Cardiology Department.

Discussion

First Described By The Ophthalmologist Mikito Takayasu In 1908, Takayasu's Arteritis (Ta) Is A Rare Disorder Characterized By A Granulomatous And Necro-Inflammatory Disease Of Aorta And Its Major Branches [1]. Ta Affects Predominantly Women Of Childbearing Age [2, 3]. The Diagnosis Of Ta Is Reached On The Basis Of Clinical Presentation And Imaging Results. Histopathologic Confirmation Can Be Obtained In Patients Who Undergo Vascular Surgery. The Diagnosis Ofta Is Particularly Challenging Because This Disease Can Affect A Variety Of Organ Systems, Producing Diverse Clinical Scenarios, The American College Of Rheumatology Proposes The Following Classification Criteria For Ta (Figure 5) .When 3 Or More Of These 6 Criteria Are Present, A Diagnosis Of Ta Can Be Rendered With A Sensitivity Of 90.5% And A Specificity Of 97.8%. Renal Arteries Are Affected In 24% To 76% Of Cases [2, 4, 5].

Takayasu Arteritis Is Classified According To The Angiographic Findings (Lupi Herrera Classification). These Are As Follows[6]:

- Type I. Aortic Archus Or Main Branches Of Aorta Involved
- Type II. Thoracoabdominal Aorta Involved
- Type III. Diffuse Involvement
- Type IV. Pulmonary Involvement

In Our Case, It Was Type 2takayasu Arteritis, Affecting Only Renal Arteries. The Initial Inflammatory Phase Of Ta Did Not Occur. The Onset Of His Disease Was Undetectable, And Severe Systolic Arterial Hypertension, Which Was Revealed Accidentally, Was The Predominant Clinical Manifestation.

The Differential Diagnosis Can Include Certain Infectious Causes Of Large-Vessel Aneurysm (Mycobacterial, Syphilitic, Or Fungal) , These Are Similarly Not Associated With Stenotic Arterial Lesions. Certain Autoimmune Illnesses (Systemic Lupus And Behçet's Disease) Can Be Associated With Large-Vessel Vasculitis; However, These More Typically Have Other Distinguishing Factors And Distinct Age Predilection (Kawasaki Disease And Giant Cell Arteritis). Perhaps, The Most Difficult Distinction Is Between Ta And Giant Cell Arteritis. Both Conditions Involve Large Arteries, Which Show Granulomatous Vasculitis On Histologic Examination, And Both Respond To Corticosteroids. Sarcoidosis Should Also Be Included In The Differential Diagnosis Of Ta. The Correct Diagnosis Usually Depends Upon The Presence Of Other Characteristic Features.

The Occurrence Of Ras In Ta Follows The Typical Sequence Of Events: Acute Inflammatory Arteritis And Lymphocytic Infiltration, Intimal Thickening, Elastic Tissue Destruction, fibrosis And Patchy Luminal Narrowing Of Renal Arteries Leading To Reduction In Blood flow And Development Of Renovascular Hypertension. Acute Renal Failure Can Occur With The Rapid Rise In Serum Creatinine And The Marked Decrease In Creatinine Clearance, Particularly With The Use Of Angiotensin-Converting Enzyme (Ace) Inhibitors.

The Pathophysiologies Of Renovascular Hypertension Is Based On The Fact That Decreased Renal Perfusion Triggers Cause Renin Release From The Juxtaglomerular Apparatus With Increased Production Of Angiotensin Ii. Angiotensin Ii Causes Vasoconstriction Of Both The Afferent And The Efferent Arterioles, With The Latter Affected More Because Of Their Smaller Caliber. It Also Causes Mesangial Constriction That Reduces The Effective filtration Surface Area. In The Ischemic Kidney, Efferent Vasoconstriction Maintains The Glomerular filtration. With The Use Of Ace Inhibitors, This Compensation Is Blocked, Causing A Drop In filtration And Progression Of The

Disease Scenario, Particularly With Preexisting Ras. The Development Of Renovascular Hypertension Is Initiated By Increased Renin Secretion, Leading To The Increased Production Of Angiotensin Ii And The Consequent Aldosterone Release, Which Causes Excessive Salt And Water Retention. If There Is Unilateral Ras, Pressure Diuresis By The Contralateral Kidney Prevents Volume Overload, And The Angiotensin-Mediated Hypertensive Effect Is Dominant. However, In The Case Of A Solitary Kidney, The Volume Component Predominates Because Of The Ineffective Volume Handling By The Single Ischemic Kidney. This Sets Up A Vicious Cycle With Further Decrease In Effective Renal Perfusion, Third Spacing Of fluid And Extremely High Compensatory Renovascular Hypertension. If Sufficient Compensatory Renal Perfusion Is Achieved, Blood Pressures Will Normalize In Days To Weeks. However, If Ischemic Nephropathy Occurs As A Result Of Continued Hypoperfusion, Drug-Resistant Hypertension Occurs And Often Requires Multiple Drugs To Achieve Reasonable Control. This Situation Can Persist Even After Correction Of The Underlying Stenosis.

Ta Is A Chronic, Progressive Disease. Its Degree Of Activity Varies Over Time; The Intensity Of Its Inflammatory Processes Typically Fluctuates Between Exacerbation And Reduction Or Remission. Vascular Involvement Tends To Be Progressive And Vascular Complications Of The Cardiac, Renal, And Central Nervous Systems Are The Major Causes Of Morbidity And Death In Ta, Which Is Usually Fatal When It Remains Untreated. Therefore, Remission Remains The Goal Of Therapy.

The Mainstay Of Therapy In Ta Is Immunosuppression, Primarily With Steroids Or Methotrexate .Alternative Therapies Such As Azathioprine, Cyclophosphamide, Mycophenolate Mofetil, And Tacrolimus Hydrate Are Also Used In Ta, Especially For Corticosteroid-Resistant Disease[7,8]. Hypertension Should Be Treated Aggressively Often With Multidrug Regimen, But Cardiologist Should Be Warned Against Ace Inhibitors Until Renal Artery Stenosis Has Been Excluded. Remission Occurs In 40-60% And Resistance To Any Therapy In 20% Of The Patients. Surgery Is Required In 30% Of The Patients; If Possible, Surgery Should Be Delayed Until The Acute Phase Of The Disease Has Passed[9]. .

Conclusion

Takayasu Arteritis With Renal Artery Involvement Must Be Kept In Mind As An Etiologic Factor For Secondary Hypertension, Even If There Is No Blood Pressure Difference Between Extremities. Rapid Diagnosis And Early Treatment Are The Cornerstone Of Kidney Survival.

Consent

A Written Informed Consent Was Obtained From Patient For The Publication Of This Paper.

Conflict Of Interest

The Authors Declare That They Have No Competing Interest .

List of Abbreviations

Ta: Takayasu Arteritis.
Ras: Renal Artery Stenosis.
Ace: Angiotensin Converting Enzyme;

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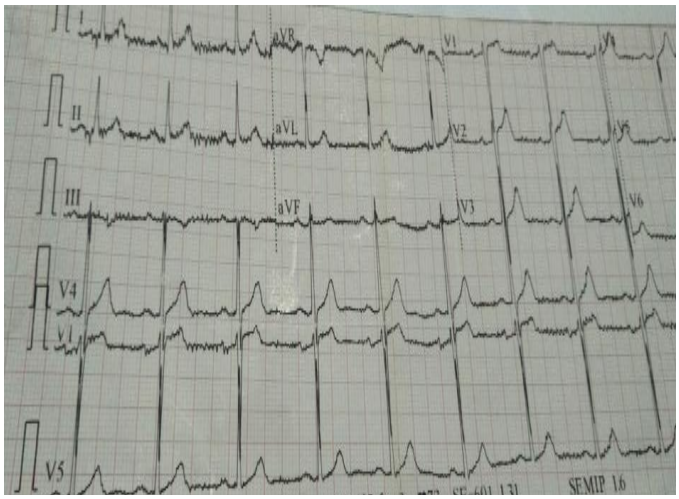

Figure 1. Ekg Showing Ventricular Hypertrophy.

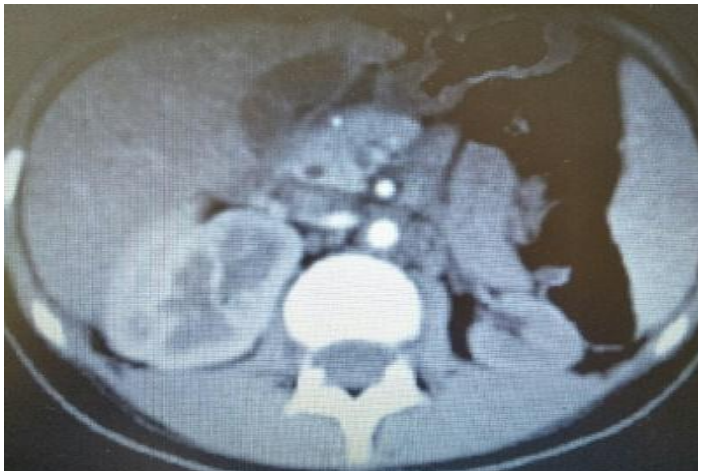

Figure 2 : Computed Tomography Of Abdomen Shows Left Kidney Smaller Than Right One And Circumscribed Thickening Of The Infrarenal Aortic Wall.

Figure 4 : Renal Angiogram Demonstrating Occlusion Of Right Renal Artery, Right Kidney Is Infused By 2 Polar Arteries.

Figure 3 : Renal Angiogram Demonstrating Severe Proximal Left Renal Artery Stenosis And Decreased Calibration At Infrarenal Aorta.

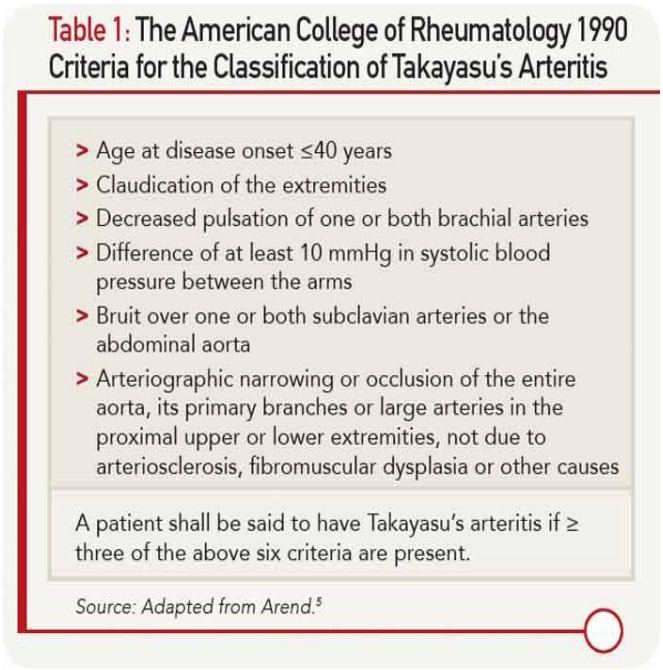

Figure 5: Criteria For The Classification Of Takayasu Arteritis.