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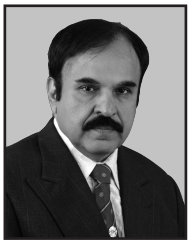

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Editor's Note

Dear colleagues,

Greetings of the season!

The objective of TAPIJ is to publish up-to-date, quality original research papers alongside relevant and insightful reviews. I am delighted to present the first issue of TAPIJ of 2018, which has several informative articles, which covers various aspects of Medical sciences and unusual case reports.

This issue has an interesting review article on "Medical Nutrition Therapy" which highlights the importance of tailored dietary prescription that involves diet counselling depending upon the person's dietary requirements. This issue consists of two interesting case reports on 'Effort Thrombosis - Paget Schroetter Syndrome' and 'Congenitally Corrected Transposition of Great Arteries'.

In this edition, we are introducing a new section where we are bringing back some of the novel concepts in Biochemistry and other basic medical sciences. Besides these, we have valuable articles in the areas of ECG, Dermatology and Toxicology.

This edition comes as a combination of various specialties to enlighten the readers and will add food for thought. I am sure that this issue of TAPIJ with its articles on various aspects of medicine will explore both clinical and academic knowledge.

With warm regards,

Dr. Vijay Viswanathan

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Review Article

TAPI Journal Vol. 10, Issue 1, January – April 2018

Medical Nutrition Therapy

Vijay Viswanathan[1*], Sanjay Kalra[2]

[1]*M.V. Hospital for Diabetes & Prof. M. Viswanathan Diabetes Research Centre, Chennai*

[2]*Bharti Hospital, Karnal*

[*]*Corresponding author: drvijay@mvdiabetes.com*

Introduction:

MNT is a tailored dietary prescription that involves diet counselling depending upon the person's dietary requirements.

The concept of medical Nutrition Therapy (MNT) was introduced in 1994 by the American Dietetic Association which is the world's largest organization of food and nutrition professionals. MNT was formulated with the view to propagate the importance of therapeutic nutrition. MNT or nutrition therapy is thus defined as nutritional diagnostic, therapy, and counselling services for the management of disease.[1,2,3] Research suggests that the health and the quality of life of the patients who received MNT intervention improved. Academy of Nutrition and Dietetics suggests that the MNT is an "integral component" of the health care and the management of the different diseases such as diabetes, heart diseases, osteoporosis, chronic kidney disease and cancer.[3,4,5] Thus, MNT is a tailored dietary prescription that involves diet counselling depending upon the person's dietary requirements. It involves an individualized dietary assessment and can be offered to anyone who has an altered nutrition profile, including infants, children, adolescents and adults.6 It is also of significant importance in pregnant and nursing women and in different medical conditions (diabetes, obesity, hypertension, dyslipidaemia, and cardiovascular diseases).[6]

Goals of MNT[3,5]

- Prevent and manage disease
- Enhance medication effectiveness,
- Maintain nutritional status
- Prevent adverse complications associated with the disease
- Help patients to recover quickly and spend less time in hospital

Process of MNT

"There are 2 steps of Medical Nutrition Therapy.

MNT consists of 2 steps[7]

Step 1 – Conduction of nutrition assessment

Step 2 – Development and implementation of the nutrition care plan

Step 1 – Conduction of nutrition assessment - The first step of MNT includes the complete analysis of the medical, nutrition and medication history of the patient. The data thus obtained help the Registered Dieticians (RDs) to stratify the patients based on the risk of nutritional deficiencies. This step includes checking the person's nutrition status, and providing the correct food or nutrients to manage the conditions such as diabetes, heart disease and cancer.[5,7]

Step 2 - Development and implementation of the nutrition care plan – The second step of MNT is to develop a nutrition plan based on the assessment. The nutrition plan may include providing simple changes in the diet of the patient. RDs ensure the implementation of the nutrition care plan. The nutrition therapies suggested by the RDs help the patients to manage the disease condition.[5,7]

MNT in chronic diseases

In chronic diseases such as diabetes mellitus, gestational diabetes, obesity, hypertension, disorders of lipid metabolism, heart failure, osteoporosis, celiac diseases, and chronic

kidney diseases, adoption of MNT has proved beneficial.[8]

Diabetes – In comparison to the pharmacotherapy, or no intervention, adoption of MNT has proven to be cost-effective in prevention of diabetes.[8] In a study, in persons with diabetes, use of MNT for 3 to 6 months resulted in the reductions in HbA1c between 0.25% to 2.9% depending on the type and duration of diabetes. Persons who adhere to MNT also showed an improved lipid profile, weight management, and decreased need for medication.[8]

American Association of Clinical Endocrinologists (AACE) / American Diabetes Association (ADA) nutritional guidelines for the management of diabetes[2]:

Intake of hypocaloric (weight loss) diet: 250 to 1000 kcal/day deficit

Target: decrease weight by 5% to 10% for overweight/obese, 15% for class 3 obesity

Target: decrease BMI by 2 to 3 units

Intake of carbohydrates (preferably low-glycemic index): 45% to 65% daily energy intake and not less than 130 g/d in patients on low calorie diet

Intake of protein: 15% to 20% daily energy intake

Intake of dietary fat: <30% daily energy intake

Intake of saturated fat: <7% daily energy intake

Intake of cholesterol: <200 mg/day

Intake of fiber: 25 to 50 g/day

Intake of trans fats: minimize or eliminate

Gestational diabetes – The main objective of MNT in managing gestational diabetes is to maintain adequate pregnancy weight along with fetus growth while maintaining euglycemia and without ketosis, which overall improves glycemic control.[9] In women with gestational diabetes, an analysis of the evidence recommended the implementation of MNT within 1 week of identification of impaired glucose tolerance. An early MNT implementation has proven to reduce hospital admissions and insulin use.[8]

Obesity – The measures of obesity used in the clinical practice are BMI and waist circumference.[10]

- Normal BMI: 18 to 22.9 kg/m^2
- Overweight: 23 to 24.9 kg/m^2
- Obesity: >25 kg/m^2

Studies in persons with obesity have suggested that MNT can statistically and significantly reduce the body weight.[8] A dietician can help in making definitive lifestyle changes such as including regular exercise in daily regime, moderation in alcohol consumption, abstaining from smoking, stress reduction, and modifications in diet such as lowering the intake of total and saturated fatty acids, increasing plant based diets along with monitoring the sodium content in the diet.[11]

Disorders of lipid metabolism – MNT has proved to be a cost-effective method to prevent and treat lipid disorders in patient. The combination of medications and MNT for managing severe forms of lipid disorders has resulted in lower dosage and lesser side-effects.[8]

Chronic kidney failure – Renal diseases are often the complication of diabetes and hypertension. In patients with renal disease, the diet therapy helps to maintain good nutrition, slows down the progression of disease, and helps to treat complications. The main diet constituents that slows down the progression of disease are[8,12]:

1. Blood pressure should be controlled (less than 140/90 mm Hg) by limiting sodium intake to 2300 mg a day and by delimiting the potassium intake if serum potassium is greater than 5 mEq/L.
2. Albuminuria should be controlled by reducing the dietary protein intake

   For nondiabetic: 0.8 g protein/kg/day

   For diabetic: 0.8 to 1 g protein/kg/day
3. By controlling diabetes.

Integrating MNT and Pharmacotherapy

In cases where optimal management of specific disease states and conditions cannot be

achieved with MNT alone and pharmacotherapy is required, a team approach with active collaboration among registered dieticians and other health care team members should be promoted.[8]

Role of dietician in management of MNT for patients receiving pharmacotherapy[8]

- Prepares a customized nutrition prescription depending upon the patient's energy, macronutrient, mineral, vitamin, fibre and fluid requirements, and the effect of medicines.
- Intervenes with foods, supplemental nutrients, parenteral and enteral nutrition, and also basic lifestyle and exercise adjustments.
- Collaborates with pharmacists and gains information on potential drug-nutrient interactions, and adjusts patient's diet accordingly to achieve optimal therapeutic benefit.

Need for Nutrition Supplementation

As the nutritional professionals possess the expertise in the composition of food and nutrient metabolism, as MNT providers they customize food intake in collaboration with food preferences and lifestyle habits of an individual.[8]

Nutrition supplements are not intended to substitute food and cannot replicate all the nutrients present in whole foods; however supplements are a useful and effective way to obtain essential nutrients that might otherwise be lacking in routine diet.[13]

Use of supplements is recommended in the following situations[13,14]:

- There is an increased demand of nutrients in the body during pregnancy, especially of protein, iron, folate and calcium.
- People who are vegan or vegetarians eat a limited variety of foods and may suffer from nutritional deficiencies.
- People who consume less than 1600 calories a day.
- Older adults have higher requirements of vitamin B12 and Vitamin D.
- Women who experience heavy bleeding during menstrual cycles.
- People who have a medical condition which affects the metabolism of nutrients.
- People who have had a surgery and are unable to digest and absorb nutrients adequately.
- Children who are picky eaters and consume a diet deficient in nutrients such as proteins, DHA, vitamins and minerals (especially calcium and iron).

Summary

MNT is a tailored dietary prescription that involves diet counselling depending upon the person's dietary requirements. MNT is of significant importance in pregnant and nursing women and in different medical conditions. In chronic diseases adoption of MNT has proved beneficial and is an "Integral component" of the health care and management of different diseases such as diabetes, heart diseases, osteoporosis, chronic kidney disease and cancer. Active collaboration among registered dieticians and other health care team members including pharmacists should be promoted in cases where optimal management of specific disease conditions requires pharmacotherapy along with MNT. Nutrition supplements are recommended for those who lack nutrients in their diet, have a condition associated with altered nutrient metabolism, or have an increased nutrient requirement due to certain physiological states.

References

1. Morris SF, Wylie-Rosett J. Medical nutrition therapy: a key to diabetes management and prevention. Clin Diabet 2010;28(1):12-18.
2. Mechanick JI, Marchetti AE, et al. Diabetes-specific nutrition algorithm: a transcultural program to optimize diabetes and prediabetes care. Curr Diab Rep. 2012;12:180–94.
3. American Dietetic Association. Initial Step: American Dietetic Association says medical nutrition therapy is "integral component" of disease treatment [Press Release]. 2010 [cited 2017 Oct 04]. Available from

http://www.eatrightpro.org/resource/media/press-releases/positions-and-issues/medical-nutrition-therapy-is-integral-component-of-disease-treatment.

4. Taber's Medical Dictionary Online. 22nd ed. F.A. Davis Company; 2014. Available from http://www.tabers.com/tabersonline.
5. National Cancer Institute. NCI Dictionary of Cancer Terms. 2016. Available from http://www.cancer.gov/dictionary.
6. Kentucky Public Health. Community nutrition and medical nutrition therapy. 2015. Available from http://www.eatrightpro.org/resource/media/press-releases/positions-and-issues/medical-nutrition-therapy-is-integral-component-of-disease-treatmenthttp://chfs.ky.gov/NR/rdonlyres/46C003CD-D57F-4523-82A4-55E1CBE31E45/0/6CommunityNutritionandMNT.pdf
7. Gilbreath JL, Biesemeier C. Medical nutrition therapy: a powerful toll in disease management. Am J Manage Care. 1999;5(1):81-88.
8. American Dietetic Association. Position of the American Dietetic Association: integration of medical nutrition therapy and pharmacotherapy. J Am Diet Assoc. 2010;110:950-56.
9. Moreno-Castilla C, Mauricio D, Hernandez MN. Role of medical nutrition therapy in the management of gestational diabetes mellitus. Curr Diab Rep., 2016;16:22.
10. Misra A, Chowbey P, Makkar BM, et al. Consensus statement for diagnosis of obesity, abdominal obesity and the Metaboli Asian Indians and recommendations for physical activity, medical and surgical man. J Assoc Physicians India., 2009;57:163-70.
11. Raj S. Introduction. In: Misra R, editor. Indian foods: AAPI's guide to nutrition, health and diabetes. 2nd ed., Chapter 1. Anna Salai, Chennai: Allied Publishers Pvt. Ltd., p. 1-5.
12. National Kidney Disease Education Program. Chronic kidney disease (CKD) and diet: assessment, management, and treatment. Treating CKD patients who are not on dialysis: an overview guide for dietitians. 2015. Available from https://www.niddk.nih.gov/health-information/health-communication-programs/nkdep/a-z/Documents/ckd-diet-assess-manage-treat-508.pdf.
13. Mayo Clinic. Healthy lifestyle: nutrition and healthy eating. 2014 [cited 2017 Oct 11]. Available from http://www.mayoclinic.org/healthy-lifestyle/nutrition-and-healthy-eating/in-depth/supplements/art-20044894.
14. Signutra. Growth and nutrition [Internet]. 2017 [cited 2017 Oct 11]. Available from http://signutra.com/nutrition.php?pageid=13.

Case Report

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An Interesting Case of Effort Thrombosis–Paget Schroetter Syndrome

Madhu Mithraa S, Lakshmanan S S *

Priya Nursing Home, Chennai

**Corresponding author: priyalaksh97@yahoo.co.in*

Abstract

We present a case of Paget-Schroetter syndrome (also called effort thrombosis), one of the rare causes of upper extremity deep vein thrombosis, treated uniquely with good outcome. To the best of our knowledge, this was the first such case in our hospital.

List of Abbrevations:

PSS	Paget-Schroetter syndrome
UEDVT	Upper extremity deep vein thrombosis
DVT	Deep vein thrombosis
IJV	Internal Jugular Vein
INR	International Normalised Ratio
MRV	Magnetic Resonance Venography
CDT	Catheter-directed thrombolysis

Introduction

Paget-Schroetter syndrome was described by Paget in 1875 (London) and Schroetter in 1884 (Vienna) independently; Huges, while reviewing his cases of spontaneous venous thrombosis, gave it its name (1) .Upper extremity deep vein thrombosis (UEDVT) refers to thrombosis of the axillary and/or subclavian veins. They are classified as primary and secondary based on pathogenesis. Primary UEDVT is a rare disorder (2 per 100,000 persons per year), which comprises (1) Paget-Schroetter Syndrome, also known as effort thrombosis, and (2) idiopathic UEDVT. UEDVT has a potential for considerable morbidity because pulmonary embolism is present in up to a third of patients with UEDVT, and other complications, such as persistent pain and swelling, superior vena cava syndrome, and problems with vascular access, can be disabling. Secondary UEDVT develops in patients with upper extremity central venous catheters, pacemakers, or cancer and accounts for most cases of UEDVT (2).

Case Report

A 50-year-old, active malewas admitted to our hospital's intensive medical unit with a 3 days history of erythematous painful swelling of his right arm. He denied chest pain, shortness of breath, easy bruising, bleeding, palpitation, bone pain, lymph node or joint swelling. His systemic review was generally unremarkable. He was normally fit and well, had no family history of thrombosis, and had not undergone surgery recently or in the past.The patient was on oral antibiotics at present which a previous physician had prescribed for a presumed right upper extremity cellulitis based on his presenting symptoms yet showed no improvement. He was not on any regular medication, denied previous history of recreational drug use, and had no known history of drug allergy.The patient is non smoker but drinks alcohol socially. After repeated questioning patient gave a history of strenuous exercise prior to his presentation.

On admission, his temperature was 101.6 °F, his blood pressure was 156/98 mmHg, his pulse rate was 98 beats/minute, his respiratory rate was 16 breaths/minute, and his oxygen saturation was 96 % on room air.

On physical examination the patient had prominent superficial veins visible over the left subclavian area (Figure 1). The left upper extremity had noticeable swelling and redness from his left arm to his palm as well as mild erythema and tenderness of the affected area

(Figure 2A & 2B). His radial and brachial pulses were 2+ at rest. All of his systemic examinations were essentially within normal limits. All of his blood workup results, including the coagulation profile, were unremarkable, except for a grossly elevated d-dimer 2160 ng/ml

Figure 1

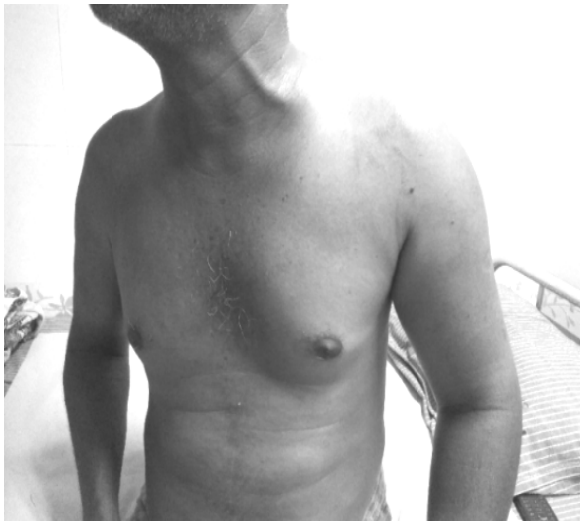


Figure 2A

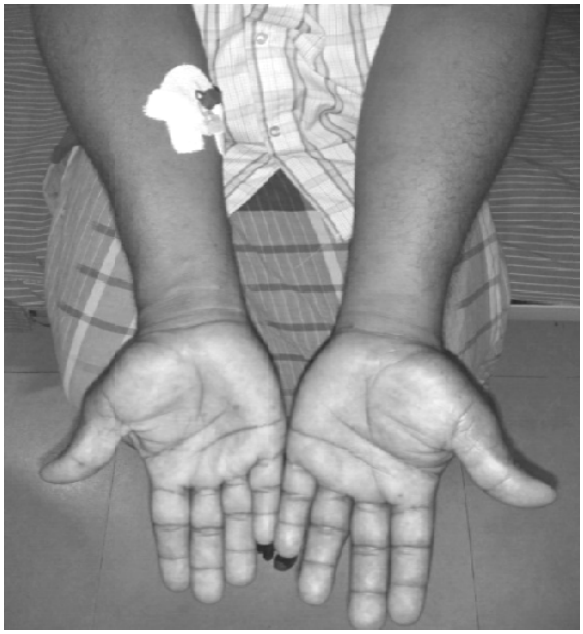
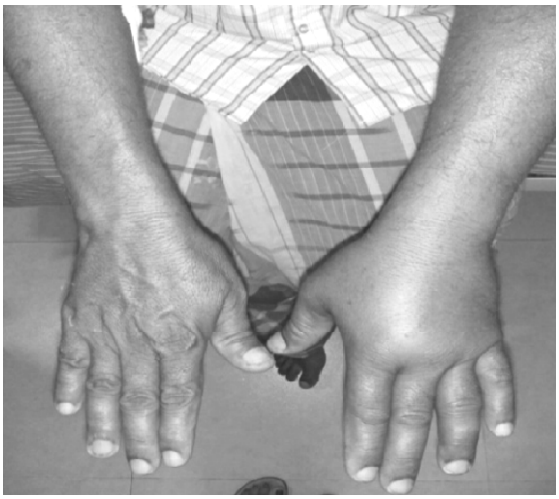


Figure 2B



The working diagnosis of left upper limb cellulitis to rule out DVT was made. The patient was then commenced on parenteral antibiotics and fondaparinaux pending further investigations. The Roentogram of his left elbow, left arm, and chest showed no abnormalities; however, venous Doppler ultrasonography revealed a left axillary and subclavian DVT with proximal extension into left distal IJV, brachiocephalic vein and distal extension into proximal left cephalic and basilic vein. (Figure 3A & 3B)

Figure 3A

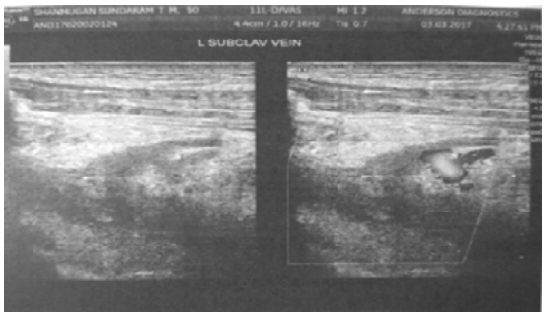
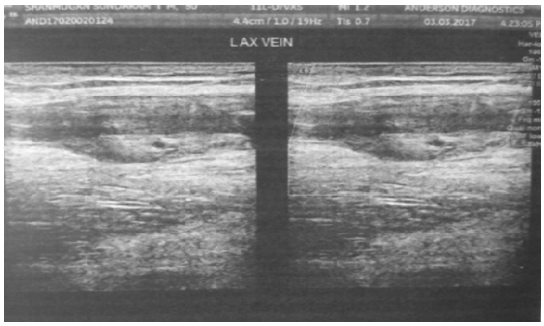


Figure 3B



CT scan of chest and neck revealed normal appearing superior vena cava with soft tissue edema in left axilla and upper arm in addition to left axillary and subclavian thrombosis (Figure 4A & 4B).

Figure 4A

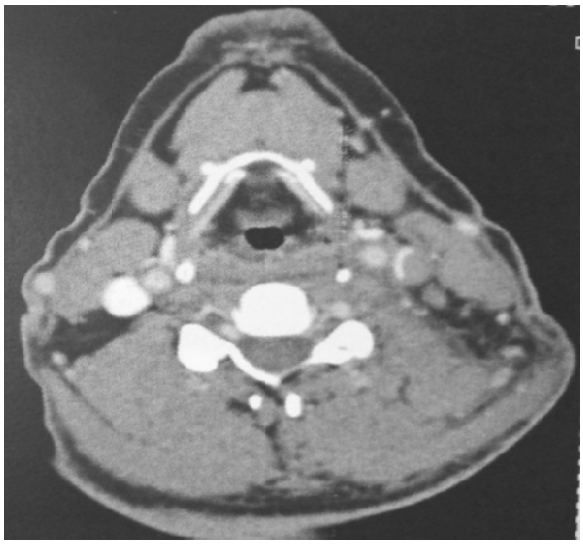
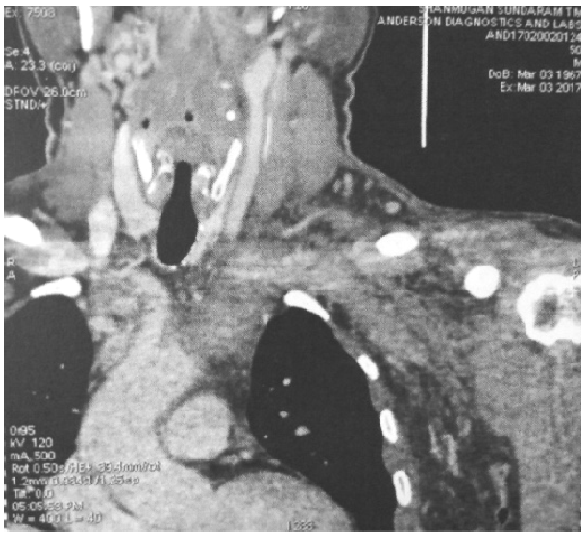


Figure 4B



Diagnosis of Paget-Schroetter syndrome of effort related UEDVT was made. Anticoagulant (Warfarin) was added and fondaparinaux was withdrawn. His INR was maintained around 2-3. On discharge, the patient's symptoms were improved. There was no swelling. He is still under regular follow up.

Discussion

PSS usually occurs in young, active, healthy, athletic people. The incidence is greater in males than females, and the mean age of occurrence is 30 years. While the dominant arm is usually involved, one-third of the patients may have the non-dominant side affected (3), as in our case. The hallmark of PSS is a lifestyle involving vigorous, repetitive upper limb movement, especially in competitive swimmers, weightlifters, and javelin throwers, all of whom develop their shoulder girdle muscles to improve their sports performance (4).

Pathogenesis

The simple pathogenesis of PSS is that heavy exertion causes microtrauma to the vessel intima and leads to activation of the coagulation cascade. Repetitive movement of the upper extremities results in scalene muscle hypertrophy, in particular the subclavius muscle. This results in compression of the subclavian vein between the ribs anteriorly, the muscle posteriorly, and the clavicle superiorly, leading to activation of the coagulation cascade. PSS may be asymptomatic, but common features include an erythematous, swollen, heavy, and painful arm, usually 24 h after the inciting event and occasionally with low-grade fever (2).

Diagnosis

Duplex Ultrasound is the initial imaging test of choice for diagnosis because it is noninvasive and demonstrates a high sensitivity and specificity for peripheral (jugular, distal subclavian, axillary) UEDVT. False-negative studies do occur, however, because of acoustic shadows from the clavicle, which limits complete visualization of the subclavian vein (5).Computed tomography and MRV magnetic resonance venography are also useful tools for diagnosis. However, these modalities could be technically difficult to use, especially in an already swollen arm (2).

Management

Traditionally treatment has consisted of limb elevation and anticoagulation. More recently, catheter directed thrombolytic therapy CTT followed by surgical decompression of the thoracic outlet has been advocated (1). For recurrent symptoms, removal of incompletely resected or regenerated rib and lysis of adhesions can best be accomplished through the posterior high thoracoplasty approach (6).

Conclusion

PSS remains largely a condition with which many clinicians are unfamiliar and that the tendency for many patients to leave the hospital undiagnosed is high. Though there are reports in literature, an individual physician's experience with this condition is usually limited, so the diagnosis requires a high index of suspicion.

References

1. Paget-Schroetter Syndrome Kavita Krishna*, Ami Shah**, A Garg***, S Pathan
2. © JAPI • VOL. 55 • MAY 2000.
3. A case of Paget-Schroetter syndrome (PSS) in a young judo tutor: a case report; Ruth Ijaopo, Victor Oguntolu et al; Journal of Medical Case Reports2016.
4. Paget-Von Schrötter Syndrome in a Non-Dominant Arm: A Case Report; Cailee E. Welch Bacon et al; Article in Athletic Training and Sports Health Care 3(6):280-283 · November 2011.
5. Paget-schroetter syndrome in an overhead athlete; Brian D. Keisler
6. Thomas D. Armsey; Current Sports Medicine Reports; July 2005, Volume 4, Issue 4.
7. Paget-Schroetter Syndrome in the Young and Active; VijuVijaysadan, MD,The Journal of the American Board of Family Practice, July-August 2005 vol. 18 no. 4 314-319.
8. Paget-Schroetter syndrome: what is the best management? Ann Thorac Surg. Urschel HC; Razzuk MA; 2000; 69(6):1663-8; discussion 1668-9 (ISSN: 0003-4975)

Congenitally Corrected Transposition of Great Arteries: A Case Presentation

Anantha Subramaniam G*, Udhaya Balasubramanian, Dhriti Shukla, Chitra Ayyappan

Apollo Hospitals, Chennai

**Corresponding author: gananthasubramaniam@gmail.com*

Abstract

A congenitally corrected transposition of great arteries is a rare congenital heart defect due to left looping of the heart tube and failure of the aorto-pulmonary septum to rotate. It occurs in 0.5 to 1% of congenital heart diseases, with an incidence of 1 in 33,000 live births (2). The venous blood returns from the body to the right atrium and flows through mitral valve to morphological left ventricle. Blood then enters the lung via the pulmonic valve into the main pulmonary artery. Pulmonary venous blood returns to the left atrium and then passes through the tricuspid valve to the morphological right ventricle, exiting the heart through the aorta via the aortic valve. The aorta is positioned anterior and to the left of pulmonary artery. Thus despite atrioventricular and ventriculo-arterial discordance, normal circulation is restored.

Case

A 52 years old lady came for a routine health check up with an echo report of CCTGA with complaints of left sided chest pain, radiating to the back, associated with dizziness and nausea, exacerbated by strenuous activity. She also complained of palpitations, paroxysmal nocturnal dyspnea and increased frequency of urination. She had no family history of congenital heart disease. On examination, she presented with pallor and anasarca. Her blood pressure was 80/60 mmhg, with a regular pulse rate of 64/min. On auscultation, an early diastolic murmur was heard along the left sternal edge with a loud S2. Apical impulse was normal. Rest of the physical examination was normal. Lab reports were normal except for anemia. Her ECG showed junctional rhythm with T-wave inversion in inferolateral leads with intra-ventricular conduction delay. Chest X-ray showed cardiomegaly. ECHO confirmed congenitally corrected transposition of great arteries and 2+ aortic regurgitation. She could not afford CT or MRI. The patient was started on a regimen of diuretics, aspirin and iron supplements and was advised to schedule regular follow-ups.

Figure 1: Schematic diagram of congenitally corrected transposition of great arteries (Image courtesy: Abigail Maja)

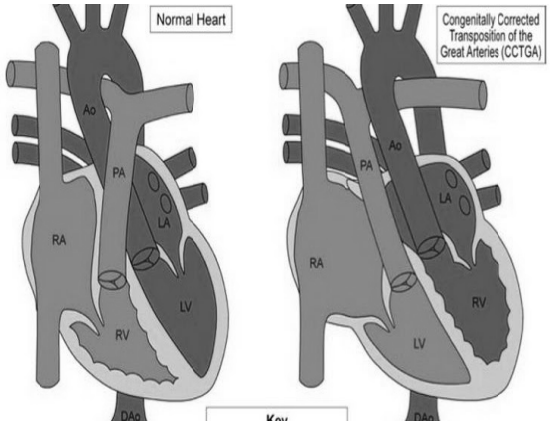


Figure 2. Echo showing atrio-ventricular discordance

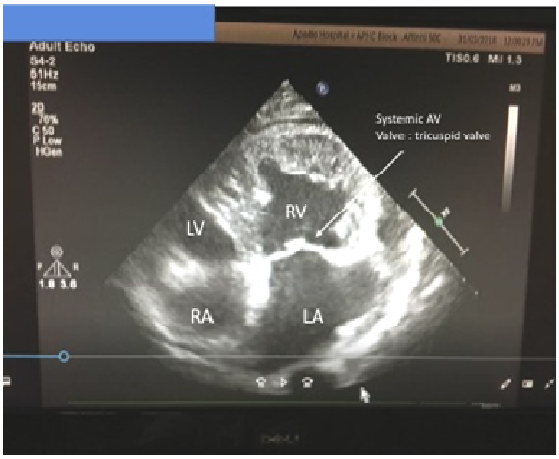


Figure 3: Echo demonstrating ventriculo-arterial discordance

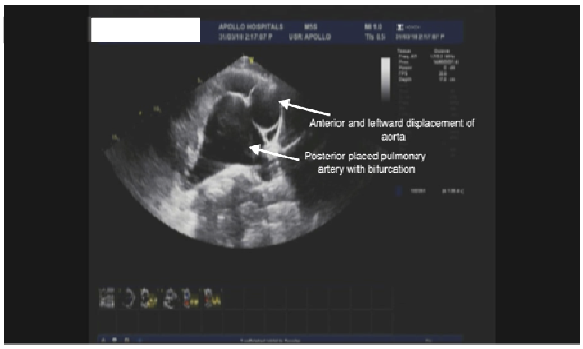
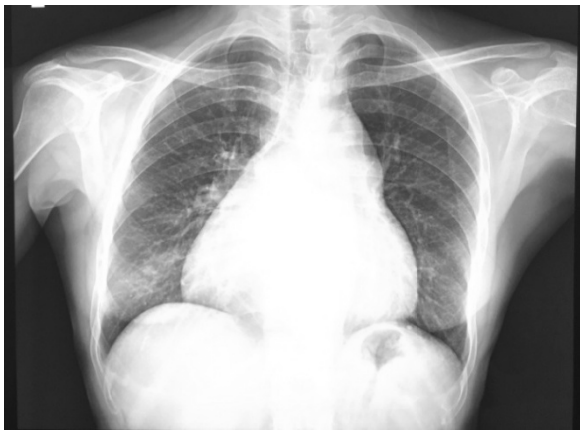


Figure 4: Chest X-ray demonstrating cardiomegaly in the patient

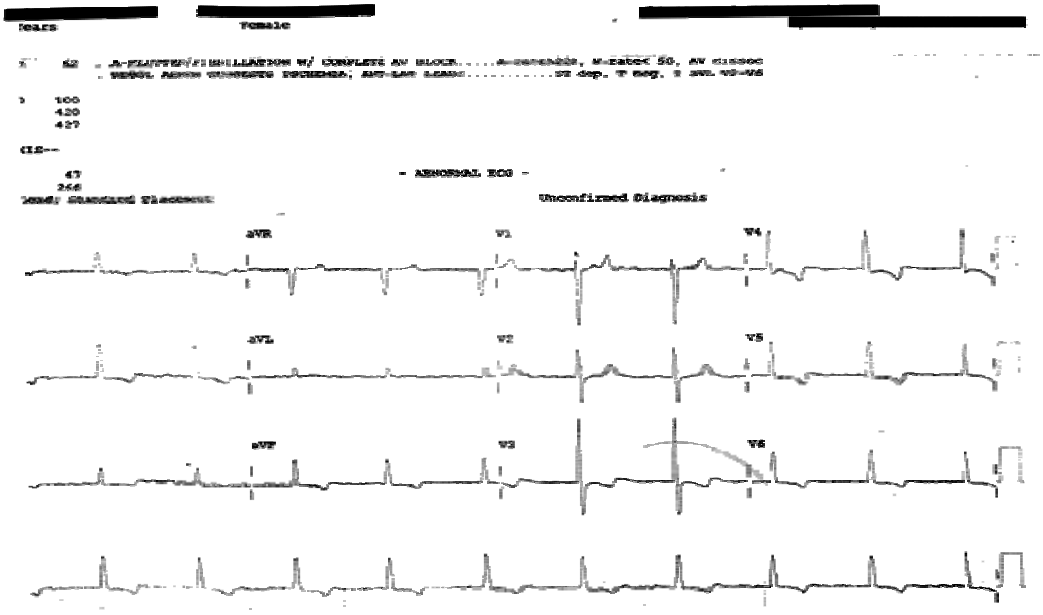


Discussion

About 150 years ago Karl von Rokitansky applied the term "corrected" to great arteries transposition. In 1961 Sheibler and associates added the word 'congenital' to avoid implication of surgically corrected transposition (5).

During childhood, patients with CCTGA are usually asymptomatic in the absence of other congenital heart defects. The most common association is ventricular septal defect in 60-80% of cases, pulmonary stenosis in 35-50% and tricuspid valve anomalies in 14-56%. Cases have been reported in new born twins (2), one in situs solitus with sinus rhythm and another in situs inversus with left atrial rhythm. CCTGA has also been reported in 83 years old male asymptomatic patient (4). The ECG could suggest CCTGA when it shows left ventricular epicardial complexes in right sided chest leads and right sided epicardial complexes in left sided leads. In our case the relatively slower heart rate, the junctional rhythm and T wave changes suggest the possibility of digitalization the patient might have had. In adult life, the patient can present either with right ventricular dysfunction since it faces systemic pressure, pulmonary valve obstruction (MLV), tricuspid valve dysfunction with Epstein's anomaly (MRV), bradycardia, complete heart block or right ventricular failure due coronary artery perfusion mismatch. Poor prognostic factors are pulmonary artery hypertension, congestive heart failure, and tricuspid regurgitation and conduction abnormalities (1) Surgery in adults is usually not required in

Figure 5: ECG demonstrating junctional rhythm in the patient



asymptomatic patients when there are no other accompanying heart defects and normal circulation is maintained. Treatment includes management of the heart failure with ACE inhibitors, diuretics, beta-blockers and nitrates. Beta-blockers must be used with caution in such cases as it can precipitate complete heart block in patients with underlying conduction abnormality. Patients with bradyarrythmia may also benefit from cardiac pacemaker implantation. Sudden death is usually due to ventricular tachyarrhythmia.

In case of CCTGA with associated defects the following surgical options could be considered (6)

Conclusion

Patients with Congenitally Corrected Transposition of Great Arteries without other congenital defects could have normal life span. However they can develop right heart failure any time and are predisposed to unexpected sudden cardiac deaths. Preferable to be under constant medical supervision.

Acknowledgment

We would like to thank Dr. N. Sathyabama, DMS, Apollo Hospitals for permitting us to publish this article. We also thank Dr. Karthik Ananth Professor of Medicine Wayne State University & Director Non-Invasive Laboratory Henry Ford Hospital, Detroit USA for his gui dance in preparing this article and in particular the labeling of the patient's echo.

References

1. Agarwal A; Samad F, Kalvin L; Bush Met all. A great imitator in adult cardiology practice: Congenitally corrected transposition of the great arteries.
2. MD; Mark K. Friedberg, MD. Congenitally corrected transposition of the great arteries, situs solitus or inversus. Circulation- Cardiovascular imaging 2014: 7:849- 851 Kandice Mah,
3. Schmidt M, Thiessen P, Deutsch HJ, Dederichs B, Franzen D, Erdmann E, Schicha H. Congenitally corrected transposition of great arteries (L-TGA) with situs inversus totalis in adulthood: findings with magnetic resonance imaging. Magn Reson Imaging: 2000; 18:417-422.
4. Angelo Placca et al, BMJ case reports – CCTGA in 83 years old asymptomatic Patient –2014 doi: 10. 1136/bcr 2014/204228.
5. Joseph K Perloff. The Clinical Recognition of Congenital Heart Disease – Third Edition – 1988.
6. English C. Flack and Thomas P. Graham (2012). Congenitally Corrected Transposition of the Great Arteries, Congenital Heart Disease - Selected Aspects, Prof. P. Syamasundar Rao (Ed.), ISBN: 978-953-307-472-6, InTech, Available From: http://www.intechopen.com/books/congenital-heart-disease-selectedaspects/ congenitally-corrected-transposition-of-the-great-arteries

Figure 6: Surgical repair and palliation in CCTGA associated with other congenital heart defects (6)

ccTGA {S,L,L} Surgical Repair and Palliation			
Classic / Physiologic Repair		Anatomic Repair	
Associated Defect	Repair	Associated defect	Double Switch Repair
VSD	VSD closure	VSD + normal PV	* VSD closure * Atrial switch (Senning / Mustard) * Arterial switch
VSD + PS	VSD closure + PS relief	PS / Atresia + VSD	* VSD closure + RV - PA conduit * Actrial switch + Arterial switch
VSD + PS/Atresia	Biventricular Repair: *VSD closure + LV-PA conduit	PS / Atresia + routable VSD	* RV - PA conduit * Atrial switch + Rastell
	Univentricular Repair: * systermic to PA shunt * Bidirectional Glenn * Fontan	Severe RV dysfunction Small RV Abnormal atrial anatomy	* Hemi-Mustard with bidirectional Glenn (Modified atrial switch) *Arterial switch or Rastrll procedure
TR	Tricuspid valve repair or replacement	TR	PA banding to decrease TR or tricuspid valve repair / replacement

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Bitot's Spots in a Case of Phrynoderma

Deepthi Ravi, Divya G, Sobimeena R M, Jayakar Thomas*

Sree Balaji Medical College & Hospital, Chennai

**Corresponding author: jayakarthomas@gmail.com*

Introduction

Bitot's spots was described first in 1863 by the French physician Pierre Bitot. It is a sign of Vitamin A deficiency (VAD) and is also seen sometimes in chronic conjunctival inflammation. VAD is one of the major causes of preventable blindness in the world. The other ocular features of VAD include conjunctival and corneal xerosis, night blindness, corneal ulceration and scarring.

VAD occurs as a result of malnourishment in developing countries especially due to decreased intake of provitamin carotenoids. Individuals with higher nutritional demands such as young children, pregnant and lactating women are at a greater risk. VAD increases the morbidity and mortality or measles. In developed nations VAD occurs secondary to gastrointestinal disorders which leads to malabsorption of vitamin A. Further bowel, liver and pancreatic disease can also lead to impaired storage and transport of vitamin A. Strict vegetarian or vegan diets can also lead to deficiency.

Case Report

A 7 year old male patient presented with lesions over the bilateral elbows, knees, forearms, gluteal area and thighs since the last 6 months (Figure 1A). It started as a few papules over the elbows and knees and progressed to involve the forearms, thighs and gluteal area. Follicular papules with central keratotic plugs were seen over these areas. Few papules were hypopigmented. On examination of the eyes patches of xerosed conjunctiva with a foamy layer was seen temporal to the cornea (Figure 1B). Systemic examination was within normal limits.

Pathogenesis

Vitamin A is necessary for the development of normal epithelium. VAD leads to metaplasia of the conjunctival epithelium and tangled keratin mixed with Corynebacterium xerosis which is present in the conjunctival stratum corneum. The bacteria produce gas which leads the foamy appearance of the Bitot's spots.

Tears have three layers. A lipid layer at the bottom which have fatty acids that help the tears to adhere to the eyes, middle layer of water and top layer of protein and mucus which prevents the bottom two layers from drying. Goblet cell malfunction is also present which leads to protective mucus deficiency. This mucus carries away keratin and other debris. Thus, VAD leads to tiny specks of keratin which flakes off inside the eyelid leading to Bitot's spots.

Figure 1A

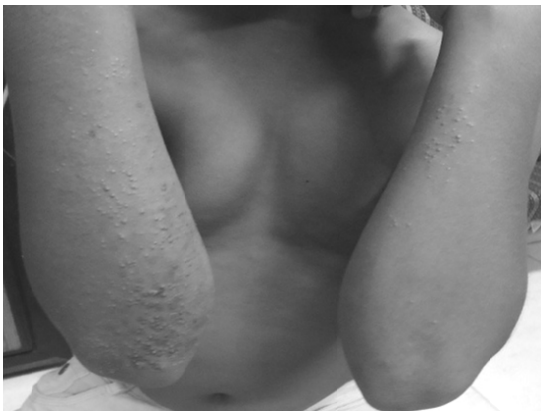
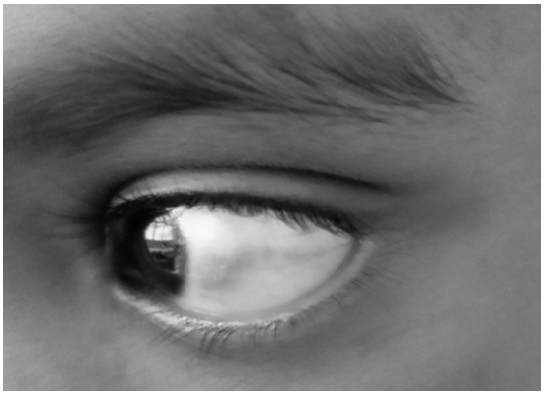


Figure 1B



Clinical features

Bitot's spots apply as triangular patches of xerotic conjunctiva with a foamy surface. It is usually seen on the conjunctiva temporal to the cornea. Wetting by the tear film is not seen in these lesions.

Differential diagnosis

Focal lesions of the cornea such as burns, trachoma and pemphigoid are differential. However, Bitot's spots are more extensive than these focal lesions.

Evaluation

The age and nutritional status of the patient has to be assessed. History of weight loss, alcohol intake (in case of adults) and jaundice has to be taken. History of any previous ocular disorder, night blindness, diarrhea, intestinal or pancreatic surgery has to be taken. The patient has to be examined for jaundice. Abdominal examination has to be done to rule out hepatomegaly. On ocular examination ocular xerosis must be assessed. The examiner must look out for symblepharon and subconjunctival xerosis.

Schirmer test can be used to assess the tear secretion. Rose Bengal test and conjunctival impression cytology can be done to assess the level of damage to the conjunctival and corneal epithelium. Dark adaptometry test can be done to assess night blindness. Histological examination of the Bitot's spots reveal keratinization, inflammation infiltrates, gram positive bacilli and irregular maturation of keratinocytes. Blood tests which can be done include serum retinol binding protein, serum vitamin A levels and serum zinc levels. Liver function tests can also be done. Ultrasound of abdomen and a lower GI-scopy can also be done to rule out hepatic and Gastrointestinal causes of VAD.

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Treatment

Treatment with 50,000 IU- 200,000 IU of vitamin A supplementation is required till the serum retinol levels become normal and signs/symptoms resolve. The clinical severity and age decides the dose and duration of treatment. The recommended daily consumption of vitamin A subsequent to that is 1000-5000 IU. 200,000 IU of vitamin A in oil administered orally is given every 6 months in India to preschool children. This comes under National Vitamin A prophylaxis program for prevention of blindness in children.

It is important to treat the underlying condition if any which has led to VAD. Retinol is transported in the blood stream by being bound to a protein known as retinol binding protein (RBP) which is synthesized in the liver from zinc and amino acids. Further, RBP breaks down if the blood does not contain sufficient hemoglobin. Hence it is important to correct both iron deficiency and zinc deficiency for treating VAD

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WPW Syndrome- Can it be Dangerous?

Joy M Thomas*, Amudhanilavan R

Frontier Lifeline Hospital, Chennai

Corresponding author: joycardio@gmail.com

WPW syndrome was described in 1930 by Louis Wolff, Sir John Parkinson and Paul Dudley White. The syndrome[1] has the following characteristics:

1. Wide QRS
2. Short PR
3. Delta wave, and
4. History of Tachycardia

Fig 1. Drs Wolff, Parkinson and White in the same order from left to right. (Courtesy Google)

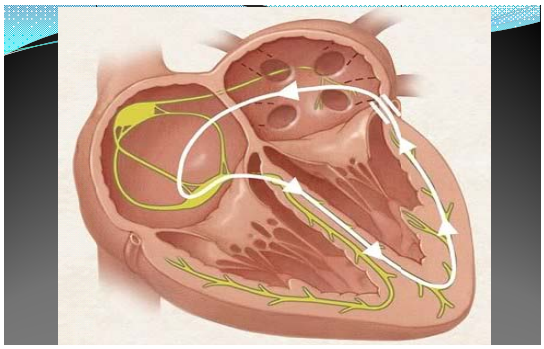

Fig 2: Section of diagrammatic representation of the heart showing pathway of an orthodromic tachycardia activating the atria through an accessory pathway located in the left lateral area.

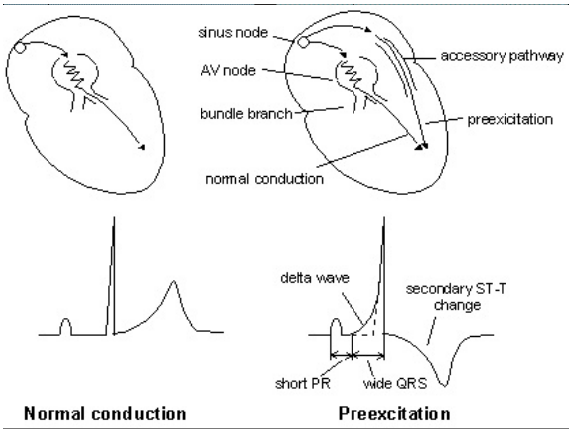

Fig 3. Cartoon depicting the Normal pathway of electrical activation in the heart on the left panel with ECG below and an activation along the accessory pathway causing pre-excitation and delta wave on the right. (Courtesy: Google)

The etiopathogenetic cause of WPW syndrome is an accessory pathway- an additional connection -between the atrium and the ventricle. This lays down the substrate for a re-entrant arrhythmia called atrioventricular re-entrant tachycardia (AVRT)[2]. During the tachycardia the ventricle can be activated through the normal conduction system resulting in a QRS that is identical in shape to the QRS during sinus rhythm. The electrical impulse from the ventricle then travels retrograde over the accessory pathway to activate the atria and the cycle is repeated in a repetitive fashion to give rise to the reentrant tachycardia. Such a tachycardia that has a narrow QRS similar to that in sinus rhythm is known as an orthodromic tachycardia[3]. On the other hand in 5 to 10 % percent of patients the atrial activation travels to the ventricle over the accessory pathway giving rise to a wide QRS. The wide QRS has a gradual upstroke in the beginning which is called the delta wave. Such a wide QRS tachycardia with a delta wave[4] is known as an antidromic tachycardia. Antidromic tachycardia indicates a greater risk factor for the patient than orthodromic tachycardia as the ventricular rates in

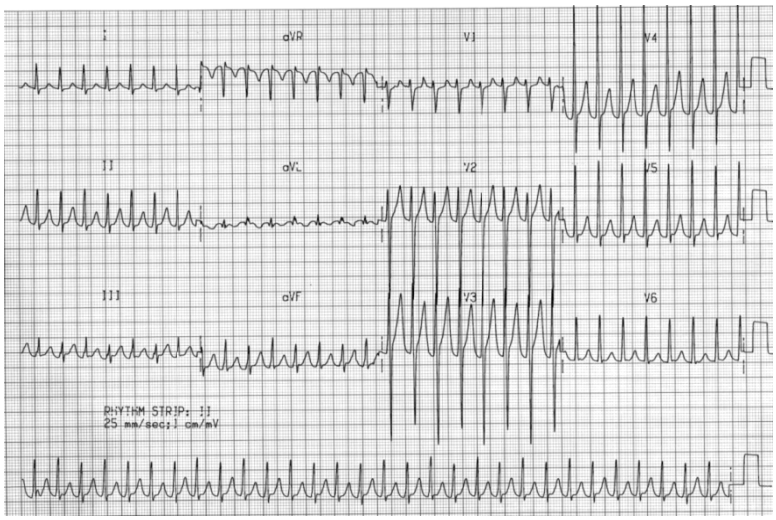

Fig 4 Orthodromic Tachycardia in a patient with concealed accessory pathway

these patients can potentially track high atrial rates as in atrial fibrillation to cause ventricular tachycardia which can degenerate to ventricular fibrillation.

Embroyologically the accessory pathways occur in patients as a remnant of the heart tube muscle fibres from which the fully formed heart at birth is developed. The heart tube bends upon itself, to form the valves that separate the atria from the ventricles and simultaneously disconnects the muscle fibers at the atrio-ventricular junction, that earlier was a continuum between the atria and the ventricles. The junction between the atria and the ventricles develops into a fibrous skeleton that incorporates the atrio-ventricular valves which will later be the tricuspid valve on the right and the mitral valve on the left. This fibrous skeleton called the Atrio-ventricular ring is an electrically inert structure that insulates the atria from the ventricles and prevents electrical activity of the atria from invading the ventricles except through a natural normal check-post - the penetrating bundle of HIS which remains as the sole electrical pathway[5] from the atria to the ventricles. Occassionaly in 0.1-0.3% of people there is an abnormal persistence of the muscle fibers that later gets modified to form the electrically conducting accessory pathways that act as an additional connection between the atria and the ventricles and this forms the substrate for a circuit that goes in a circus like pathway.

Acessory pathways can be manifest when they actively conduct impulses from the atrium by-passing the Atrioventricular node to pre excite the ventricle and cause a delta wave which is seen as a gradual sloping curve at the beginning of the QRS in a normal 12 lead surface ECG taken when the patient is in sinus rhythm. On the other hand they can be concealed when they are physically present as a connection but is able to conduct the impulses only in the opposite direction from the ventricles to the atrium. Here the surface ECG has a normal QRS as in any normal ECG and there is no pre-excitation. The accessory pathway is revealed during electrophysiological testing in the electrophysiology laboratory[6].

Generally speaking the tachycardias caused in WPW syndrome are usually benign causing mild symptoms if at all they do and many are asymptomatic recognized on an ECG just by chance. This is because the ventricular activation occurs through the normal conduction system and the patients feel only the tachycardia at the most. 65% of Adolescents and 40% over the age of 40 years with a WPW ECG are asymptomatic. About 40% lose anterograde conduction(conduction from atria to the ventricles over the accessory pathway) in the first year of life and

15% develop symptoms later while the majority remain asymptomatic. Sudden death can occur in a small minority (Incidence 0.15-o.39%). Sudden death as the first presentation is unusual.[7,8,]

How are we to identify those patients with an accessory pathway who are at danger of sudden cardiac death?

1. A simple clinical test in a patient who has a WPW is to perform a Holter or a treadmill stress test and look for the shortest pre excited RR interval. If this is seen to be less than 250 milliseconds then it is a marker of Sudden cardiac arrest[9]
2. Tachycardiomyopathy. This is a dilated cardiomyopathy where the cardiac chambers particularly the left ventricle and the lef atrium are dilated and there is reduction in the pumping capacity of the ventricle called the ejection fraction to levels below 35%. It is caused by the sustained tachycardia that is not felt by the patient or felt and ignored and persists for a few weeks at a stretch. The cardiomyopathy begins to cause exertional breathlessness. A persistent left ventricular dysfunction causes fibrosis within the myocardium and this can lead to ventricular arrhythmias and even sudden cardiac death.[10]
3. Antidromic tachycardia that activates the ventricle over the accessory pathway can attain the high rates that is present in the atrium during episodes of atrial fibrillation and this can potentially lead to dangerous ventricular fibrillation if the interval between two consecutive R waves (the RR interval) is less than 250 msecs. This is represented in a measurement called SPERRI (Shortest pre-excited RR Interval)[11]
4. Multiple accessory pathways are a risk factor for sudden cardiac arrest.[7,9]
5. Other risk factors for sudden cardiac death include Ebsteins Anomaly and familial WPW[7,9,12]

The management of these patients depends on the presence of structural heart

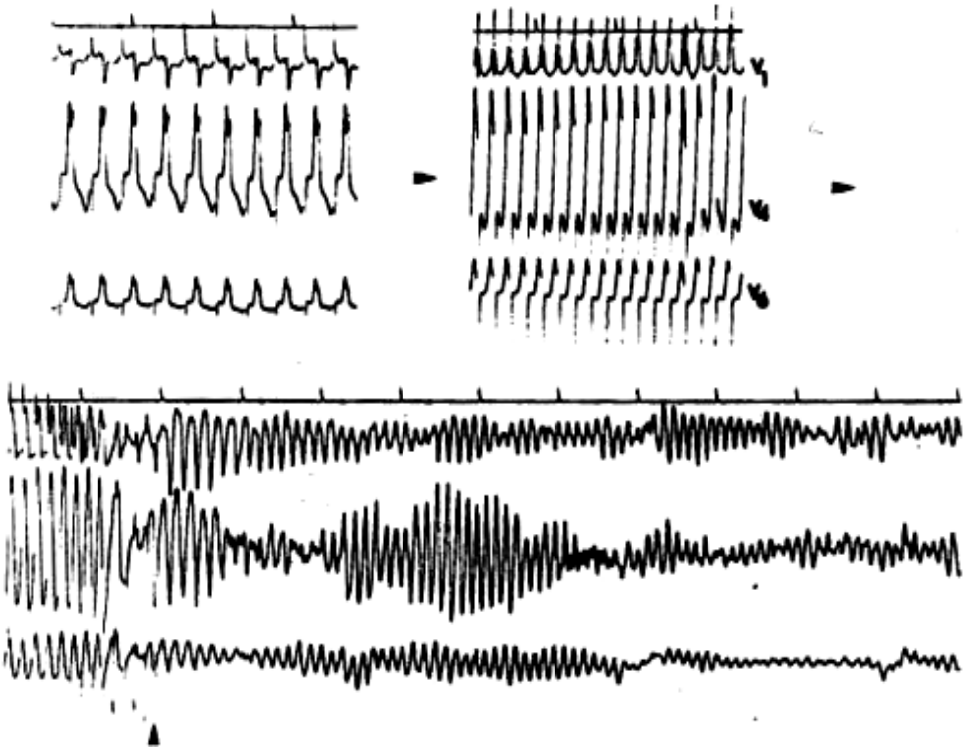

Fig 5. A pre-excited tachycardia degenerating into a Ventricular tachycardia and finally into ventricular fibrillation

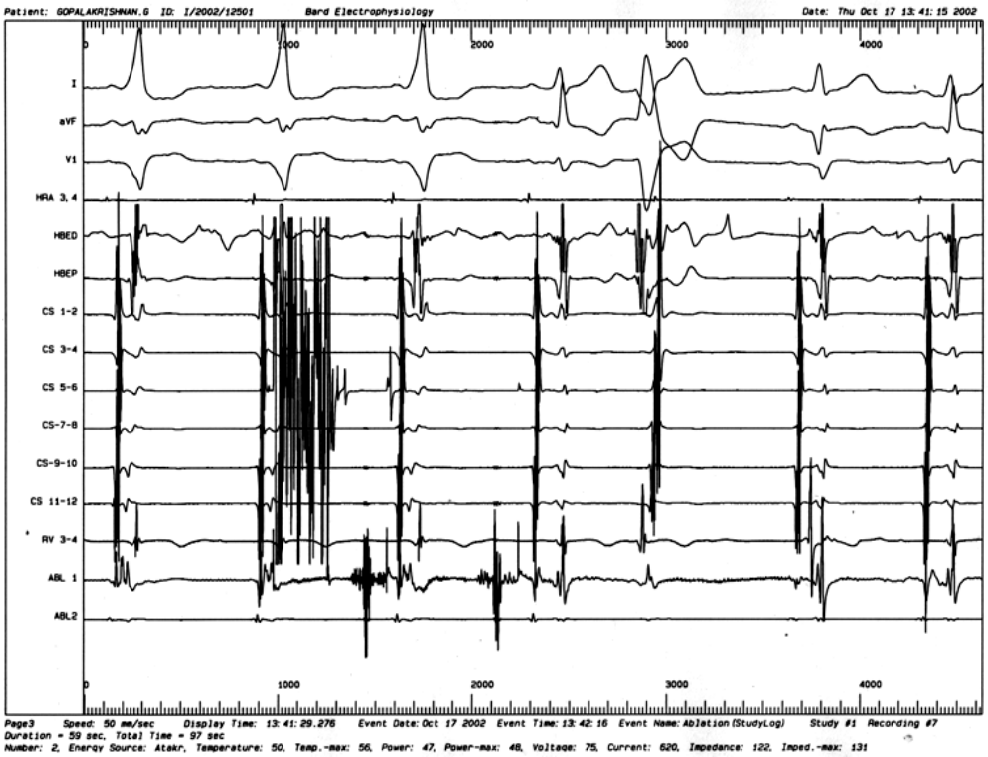

Fig 6: A snapshot during Radiofrequency of a patient of WPW syndrome. Notice the wide QRS ECG with corresponding short PR interval and AH interval on the left side panel. Almost instantaneously on applying Radiofrequency energy the delta wave disappears to yield a normal looking ECG on the right hand panel

disease such as Ebsteins Anomaly or left ventricular dysfunction. In such patients Radiofrequency Ablation is a Class II B indication

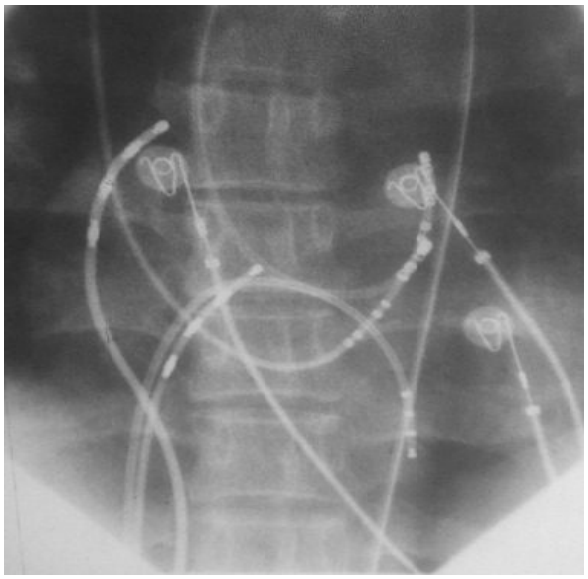

Fig 7: Postero-anterior Xray view of catheters during radiofrquency ablation of an accessory pathway in the left lateral region. The Radiofrquency delivery catheter is the one with a prominent white tip

with level of evidence C. If during a Treadmill stress test or during atrial fibrillation or any other tachycardia the shortest pre-excited RR interval (SPERRI) is less than 250 msecs then radiofrequency ablation is warranted (Class II A Level f evidence B or C). Young asymptomatic patients with WPW can undergo RFA if the risk of RFA is lesser than the SCD risk from the WPW[12'14].

Summary

The Wolff-Parkinson-White syndrome though by and large a benign arrhythmia with a nuisance value that can lead to hospitalization and expenses for the patient has a finite element of risk of sudden cardiac death. It is important to recognize this and make all efforts to identify these patients so that the dangerous element of WPW syndrome can be tackled efficiently.

References

1. Wolff l, Parkinson J, White PD. Bundle-branch block with short PR interval in healthy young people prone to paroxysmal tachycardia. Am Heart J. 1930 Aug. 5(6):685-704
2. Calkins H, Sousa J, el-Atassi R, et al. Diagnosis and cure of the Wolff-Parkinson-White syndrome or paroxysmal supraventricular tachycardia during a single electrophysiologic test. N Engl J Med. 1991 Jun 6. 324(23):1612-8
3. Durrer D, Schuilenburg RM, Wellens HJ, Pre-excitation revisited. Am J Cardiol. 1970 Jun.25(6):690-7.
4. Sethi KK, Dhall A, Chadha DS, Garg S, Malani SK, Mathew OP. WPW and Pre-excitation syndromes. J Assoc Physicians India 2007 Apr. 55 Suppl:10-5
5. Park DS, Fishman GI. Basic Science for Clinicians: The Cardiac Conduction System, Circulation. 2011; 123(8):904-915
6. Pappone C, Santinelli V, Manguso F, et al. A randomized study of prophylactic catheter ablation in asymptomatic patients with Wolff-Parkinson-White syndrome. N Engl J Med. 2003 Nov 6. 349(19):1803-11
7. Pappone c, Vicedomini G, Manguso F, et al. Risk of Malignant arrhythmias in initially symptomatic patients with Wolff-Parkinson-White syndrome: results of a prospective long-term electrophysiologocal follow-up study. Circulation. 2012 Feb 7. 125(5):661-8
8. James Kulig PA, Bruce A K. Life threatening event risk in children with Wolff-Parkinson-White Syndrome. JACC Clinical Electrophysiology. November 2017DOI:10.1016/JACEP.2017.10.009
9. Olen MM, Baysa SJ, Rossi A. et al. Wolff-Parkinson-White Syndrome: A stepwise deterioration to sudden death. Circulation 2016;133:105-106
10. Martin CA, Lambiase PD. Pathophysiology, diagnosis and treatment of tachycardiomyopathy. Heart 2017;103:1543-1552
11. Dreifus LS, Haiat R, Watanabe Y. et al. Ventricular Fibrillation: A possible Mechanism of Sudden Death in patients with Wolff-Parkinson-White Syndrome. Circulation. 1971;43:520-527
12. Khairy P, Van Hare GF, Balaji S. et al.PACES/HRS expert consensus statement on the recognition and management of arrhythmias in adult congenital heart disease: developed in partnership between the pediatric and congenital electrophysiology society (PACES) and the Heart Rhythm Society (HRS). Endorsed by the governing bodies of PACES, HRS, the American College of Cardiology (ACC), the Americam Heart Association (AHA), the European Heart Rhythm Association (EHRA), the Canadian Heart Rhythm Society (CHRS), and the international Society of Heart Rhythm. Heart Rhythm 2014 Oct.11(10):e 102-65
13. Cohen MI, Triedman JK, CannonBC, et al. PACES/HRS expert consensus statement on the mnangement of the asymptomatic young patient with a Wolff-Parkinson-White (WPW, ventricular preexcitation) electrocardiographic pattern: developedin partnership between the Pediatric and CongenitalElectrophysiology Society (PACES) and the Heart Rhythm Society (HRS). Endorsed by the governing bodies of PACES, HRS, the American College of Cardiology Foundation (ACCF), the American Heart Associuation (AHA), the Americam Academy of Pediatrics (AAP), et al. Heart Rhythm 2012. June9(6):1006-24
14. Pappone C, Santinelli V. Electrophysiology testing and catheter ablation are helpful when evaluationg asymptomatic patients with Wolff-Parkinson-White pattern: the pro perspective. Card Electrophysiol Clin. 2015 Sep. 7(3):371-6

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Toxicology clinics-bench to bed side
Allergy, Anaphylaxis and Angioedema -II

Senthil Kumaran S*, Balamurugan N

Manian Medical Center, Erode

**Corresponding author: maniansenthil@yahoo.co.in*

Allergy, Anaphylaxis and Angioedema -II

Angioedema in the absence of allergy continues to represent a medical paradox. This uncommon disorder may manifest as facial, laryngeal, genital, or intra-abdominal swelling or swelling of the extremities. Despite its often dramatic presentation, its rarity and its tendency to mimic other, dissimilar disease states often obscure its diagnosis.

1.What is angioedema?

Angioedema is a clinical diagnosis characterized by the abrupt onset of non-pitting, non-pruritic swelling that involves the reticular dermis, subcutaneous, and submucosal layers.

2.Why angioedema is important?

Angioedema may be life-threatening, depending on the underlying cause and the body location affected. Airway involvement is usually the immediate life-threat. The possibility of anaphylaxis must be considered.

3.What are the types for angioedema?

The angioedema may be Hereditary or Acquired.

4.Why hereditary angioedema?

Hereditary angioedema is a potentially life-threatening disorder caused by C1 esterase inhibitor deficiency due to genetic defect. The functionally abnormal C1-INH leads to bradykinin over-production. It affects 1/50,000 people and 50% present with recurrent episodes of angioedema by age 10 years.

5.What are the types of hereditary angioedema?

It is classified into 3 types based on the genetic defect

- Type 1 has low antigen and functional levels of C1-INH
- Type 2 has normal antigen levels but low functional levels of c1-INH
- Type 3 has normal functioning C1-INH

6.What are the causes for acquired angioedema?

- Medications
- underlying B-cell lymphoproliferative disease.
- antibody directed against C1-INH
- Food
- Latex
- Local trauma
- *Hymenoptera* envenomations
- Idiopathic

7.What medications cause angioedema?

Medications that can cause angioedema includes angiotensin-converting enzyme Inhibitors, angiotensin-2 receptor blockers, Opiates, Dextrans and NSAIDS.

8.What is the Incidence of ACE inhibitor induced angioedema?

It is documented up to 1%. The angioedema is a class effect and is not dose dependent – symptoms can occur any time from a few hours up to 10 years after the initial dose. More common in African Americans and patients on immunosuppressants. It is witnessed in patients switched to an angiotensin receptor blocker.

9.What is the Pathogenesis in angioedema?

The pathogenesis of angioedema has been studied extensively. It may be histamine-mediated or non-histamine-mediated.

- Histamine-mediated angioedema may co-exist with urticaria and is mast-cell mediated which

includes anaphylaxis, allergies, some drug reactions

- Non-histamine (bradykinin-mediated) angioedema tens to be more severe, more prolonged and less responsive to adrenaline which includes ACEI-related angioedema and both herediatry and acquired C1 esterase inhibitor deficiency.

10.What are the investigations can guide in management?

There are no point-of-care tests that can guide management in the emergency situation, but investigations may help guide long term management.

Laboratory

Identify underlying cause

- C1 esterase inhibitor (C1-INH) assays (low/ abnormal in HAE)
- C4 levels (low in HAE attacks, usually normal between attacks)
- Serial tryptase levels (may be elevated in anaphylaxis/ mast cell-mediated angioedema)

Imaging

CT abdomen may show evidence of angioedema in patients presenting with abdominal pain:

- May involve GI and GU tracts
- Angioedema of the visceral organs is often accompanied by adjacent fluid

11.What are the historical features of different causes of angioedema?

Characteristic	HAE	Acquired	ACE induced	Allergic	Idiopathic
Age of onset	2-13 yrs	Adult	Adult	Any age	Any age
Family history	75%	No	No	History of Atopy	No
Ethnicity	None	None	80% African-American	None	None
Gender predilection	No**	No	No	No	No
Location of attacks	Peripheral, abdominal, facial, laryngeal, genitourinary	Peripheral, abdominal, facial, laryngeal, genitourinary	Lips, tongue, facial	Lips, tongue, laryngeal	Lips, tongue, rarely laryngeal
Speed of attack onset	Gradual over a few hours*	Gradual over a few hours	Gradual over a few hours	Immediate within 1 hour	Variable
Duration of attacks	3-5 days Without treatment	3-5 days Without treatment	24-48 hours after drug discontinued	Several hours Without treatment	Several Hours Without treatment
Recurrent nature of attacks	Yes	Yes with or Without treatment	No if drug discontinued; attacks can persist for 4-6 weeks after drug discontinuation	Yes only if re-exposed to allergen avoided	Yes with or Without treatment
Associated with urticaria	No	No	No	Yes or No	Typically No but possible
Presents with abdominal pain	Yes	Yes	Usually not	No	No
Response to H1 antagonists and oral corticosteroids	No	No	No	Yes	Variable
Response to epinephrine	No	No	No	Yes	Variable

*can have a rapid onset in less than an hour

**for hereditary angioedema with normal complement with or without a genetic mutations Female>>Male.

- Involvement may be multifocal or asymmetric (not always be diffuse or concentric)
- CT neck primarily has a role in excluding conditions that may mimic angioedema (e.g. soft tissue infection)
- Glossomegaly is common
- Shows the extent of airway involvement

12.What are the Criteria for diagnosis of angioedema caused by C1 inhibitor deficiency?

Clinical criteria

Major

(1) Self-limiting, noninflammatory subcutaneous angioedema without major urticarial rash, often recurrent and often lasting more than 12 hours

(2) Self-remitting abdominal pain without clear organic etiology, often recurrent and often lasting more than 6 hours

(3) Recurrent laryngeal edema

Minor

(4) Family history of recurrent angioedema and/or abdominal pain and/or laryngeal edema

Laboratory criteria

1. C1 inhibitor antigenic levels <50% of normal at 2 separate determinations with patient in basal condition and after the first year of age
2. C1 inhibitor functional levels <50% of normal at 2 separate determinations with patient in basal condition and after the first year of age
3. Mutation in C1 inhibitor gene altering protein synthesis and/or function

Diagnosis can be established in presence of 1 major (1-3) clinical criteria and 1 laboratory criteria

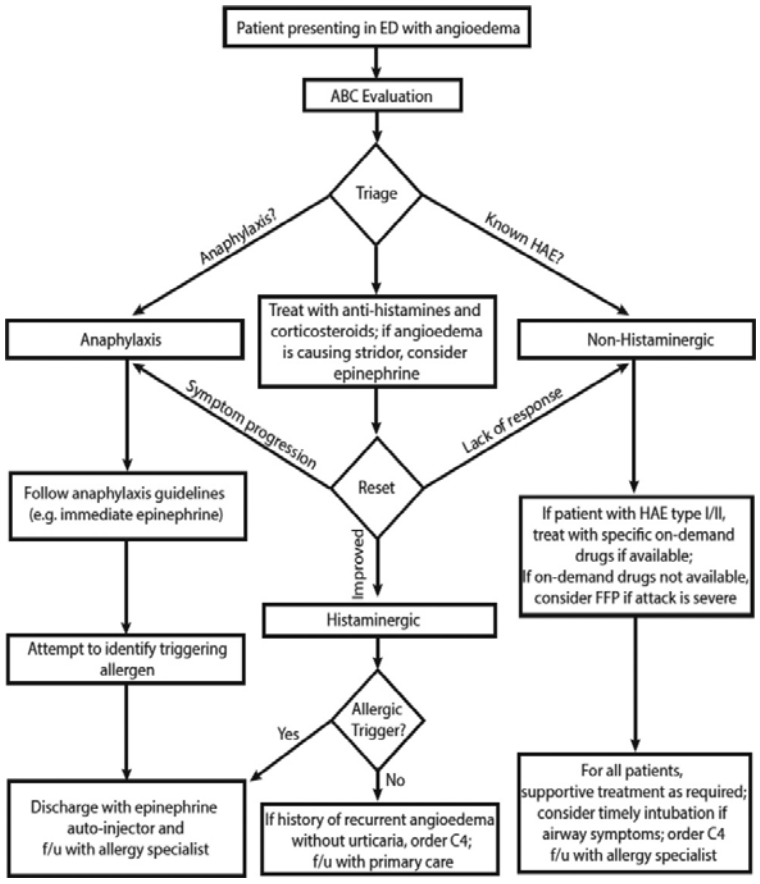
13.What are the Criteria for evaluation of disease severity?

Attack severity	**Score**
Mild attacks (discomfort noticed, but no disruption of normal daily activity)	0.5 for each 24 hours
Moderate attacks (discomfort sufficient to reduce or affect normal daily activity)	1 for each 24 hours
Severe attacks (inability to work or perform daily activity)	2 for each 24 hours
Need for treatment	
Emergency treatment: conservative, substitutive (C1-INH, FFP)	5 each
Emergency treatment: invasive (intubation, tracheotomy)	25 each
Long-term prophylaxis for more than 6 months	25
Long-term prophylaxis for 3-6 months	12.5

Score	**Class**	**Degree**
>30	1	Severe
21-30	2	Moderate
11-20	3	Mild
1-10	4	Minimal
0	5	Asymptomatic

14.What are the specific therapy for Angioedema?

- FFP
- Possible therapy for ACEI-related angioedema (case reports), thought to be beneficial in most cases
- FFP contains ACE (kininase II), which degrades bradykinin
- Risk of viral transmission, allergic reactions, and volume overload and a possibility of worsening symptoms in HAE (by providing additional substrate, kininogen)
- Therapies for HAE attacks
- Icatibant — a bradykinin 2 receptor inhibitor
- Ecallantide — a kallikrein inhibitor (kallikrein is the enzyme that produces bradykinin from high-molecular-weight kininogen (HMWK))
- C1-INH concentrate — C1 esterase inhibitor blocks the pathways that produceHMWK (the



Algorithmic Approach to the Management of Angioedema in the ED

C1-INH concentrate may be plasma-derived or recombinant)

- Limited data for role in non-HAE patients (e.g. ACEI-related angioedema)
- Several case reports describe the potential use of icatibant in the treatment of ACEI-related angioedema
- Role of adrenaline, steroids and antihistamines
- Unlikely to be be effective for ACEI-related angioedema
- This a bradykinin-mediated condition, not related to mast cell degranulation
- Many ACEI-related angioedema cases can be managed by observation alone, without pharmacotherapy or intubation
- Most ACEI-related angioedema cases resolve over 24-48h
- The apparent effectiveness of therapies may be confounded by spontaneous resolution of the angioedema
- Should be administered if the underlying cause of angioedema is uncertain (i.e. anaphylaxis is possible)

15. What is the Disposition in angioedema?

All angioedema patients with potential airway involvement should be observed in a high visibility area until marked resolution has occurred. This often requires admission to an HDU/ICU. The admission to hospital, rather than in ED, is preferred in the following situations

- Previous history of angioedema
- Tongue edema
- Pharyngeal edema (palate, uvula)
- Laryngeal edema (true vocal cords, false vocal cords, arytenoids, aryepiglottic folds,

epiglottis; the term upper airway oedema is probably more useful)

- Lack of improvement during the ED course

The patients with isolated angioedema of the face or lips and be usually be observed in ED for 4 to 8 hours for progression of symptoms, then discharged.

References:

1. Farkas H. Management of upper airway edema caused by hereditary angioedema. Allergy Asthma Clin Immunol. 2010 Jul 28;6(1):19. doi: 10.1186/1710-1492-6-19.
2. Moellman JJ, Bernstein JA, Lindsell C, Banerji A, Busse PJ, American College of Allergy, Asthma & Immunology (ACAAI); Society for Academic Emergency Medicine (SAEM). A consensus parameter for the evaluation and management of angioedema in the emergency department. Acad Emerg Med. 2014 Apr;21(4):469-84.
3. Lewis LM. Angioedema: etiology, pathophysiology, current and emerging therapies. J Emerg Med. 2013 Nov;45(5):789-96.
4. Senthilkumaran, S., Balamurugan, N., Karthikeyan, V., Ganapathysubramanian, J. Fresh-frozen plasma as a treatment for life-threatening ACE-inhibitors angioedema – case series. J Gene Med India. 2007;20:32–34.
5. Senthilkumaran, S., David, S.S., Menezes, R.G., Thirumalaikolundusubramanian, P. Role of fresh-frozen plasma in angioedema: progress and problems. Eur J Emerg Med. 2013;20:292–293.

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Adiponectin Level as A Powerful Risk Marker for Prediabetes

Udyama Juttada, Vijay Viswanathan[*]
M.V. Hospital for Diabetes & Prof. M. Viswanathan Diabetes Research Centre, Chennai
[*]*Corresponding author: drvijay@mvdiabetes.com*

Abstract:

Adiponectin consistently have inverse and stronger associations with risk of type 2 diabetes (T2DM) even after adjusting for adiposity and cardiovascular vascular risk factors (1). The predictive nature of adiponectin for development of diabetes was confirmed using univariate and multiple logistic regression analyses after the adiponectin values using biochemical tests are obtained. It is suggested by observations that many advanced drugs with beneficial effects on insulin resistance and glucose intolerance (e.g. peroxisome proliferator-activated receptor-g agonists and the selective cannabinoid-1 receptor blocker rimonabant) will increase adiponectin concentrations. In a number of case-control and cohort studies (2 - 5), adiponectin levels have been found to be inversely associated with insulin sensitivity, conversion to diabetes, and risk of myocardial infraction.These associations remain significant after adjustment for baseline measures of obesity, suggesting that adiponectin reflects components of metabolic and vascular risk beyond those encompassed in blood sugar level (6). The measurement of adiponectin may therefore carry additional prognostic value for diabetes and heart disease beyond the currently recognized set of risk factors.

Keywords: T2DM, AdipoQ Adiponectin, Prediabetes, MetS, metabolic syndromes

Introduction

Adiponectin is an adipocyte-specific factor, first described in 1995. Over the past two decades, numerous studies have elucidated the physiological functions of adiponectin in obesity, diabetes, inflammation, atherosclerosis, and cardiovascular disease (7). Adiponectin, elicited through cognate receptors, suppresses glucose production in the liver and enhances fatty acid oxidation in skeletal muscle, which together contributes to a beneficial metabolic action in whole body energy homeostasis. Beyond its role in metabolism, adiponectin also protects cells from apoptosis and reduces inflammation in various cell types via receptor-dependent mechanisms. Adiponectin, as a fat-derived hormone, therefore fulfills a critical role as an important messenger to communicate between adipose tissue and other organs. A better understanding of adiponectin actions, including the pros and cons, will advance our insights into basic mechanisms of metabolism and inflammation, and potentially pave the way towards novel means of pharmacological intervention to address path physiological changes associated with diabetes, atherosclerosis, and cardiometabolic disease (8).

In 2001, two key papers demonstrated for the first time the physiological role of adiponectin and highlighted the adiponectin axis as a potential therapeutic area for diabetes treatment (4, 9). Thus, the well-known association of adiponectin with diabetes risk is evident at a much earlier stage in pathogenesis during transition from normoglycemia to prediabetes. Adiponectin levels are positively associated with insulin sensitivity, and inversely associated with the development of diabetes and progression from prediabetes to type 2 diabetes (T2DM). Although the evidence linking adiponectin to diabetes risk is strong, the chronology of the association between adiponectin status and glucose homeostasis has not been fully determined. Specifically, the potential role of adiponectin in modulating early glucose abnormalities during transition from normoglycemia to prediabetes is a subject of significant interest. In a study, Jiang etal. (10) predicted that interventions that boost adiponectin levels may offer protection against the

risk of dysglycemia, regardless of gender or ethnicity.

History of adiponectin

Over the past 20 years, adiponectin has gained considerable attention as it plays an important role in many organs and it does so mainly via interaction with its two receptors. In the liver, adiponectin strongly suppresses hepatic gluconeogenesis, while enhancing nutrient utilization in skeletal muscle (11).The unique features of adiponectin make the pathway a unique candidate to look for therapeutic strategies for diabetes. The progress made in the adiponectin field greatly helped us to better understand the role of adipose tissue. The many additional adipokines secreted from adipocytes carry metabolic clues from fat to other tissues to communicate the state of the adipocyte under various conditions.

The predictive nature of low concentrations of adiponectin for diabetes has been demonstrated in Pima Indians (12) and in the European Prospective Investigation into Cancer and Nutrition (EPIC) study in Germany (13). Lower concentrations of adiponectin found in the above studies, when compared with the values in Indian study group (2), may be partly related to the differences in BMI and also there may be population-based differences in the adiponectin concentrations. *AdipoQ* gene can be used as the biomarkers for early diagnosis and clinic prediction of diabetes, obesity, diabetic complications and other metabolic disorders (14).

Hypoadiponectinaemia is associated with obesity, insulin resistance and type 2 diabetes (15), as well as atherosclerosis, hypertension and coronary artery disease (16). The ability of adiponectin to reduce insulin resistance in conjunction with its anti-inflammatory and anti-atherogenic properties makes this novel adipocytokine a promising therapeutic target, and agents that enhance adiponectin secretion or action have potential for treatment of diabetes. Low circulating levels, particularly of the HMW component (17), are also a strong risk marker for the development of the type 2 diabetes.

Choi et al. (18) in a prospective study of 372 elderly Koreans found adiponectin levels to

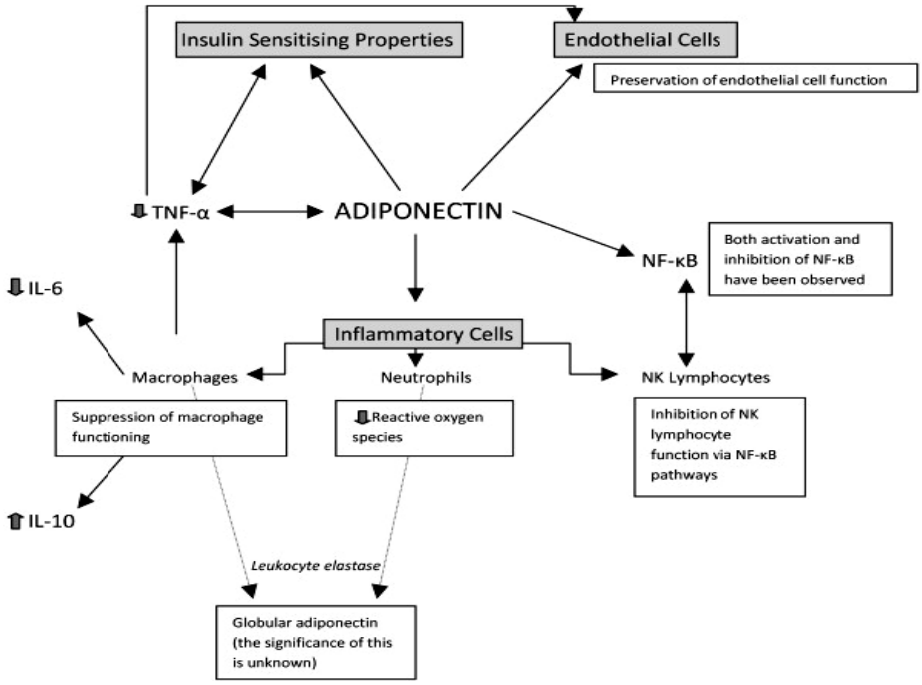

Fig1: Schematic representation of adiponectin regulation and currently known mechanisms of metabolic and vascular effects (22)

be strongly associated with developing diabetes. Mohan et al. (19) in a small case –control study of Asian Indians showed that lower adiponectin levels were associated with prevalent MetS. Matsushita et al. (20) performed a cross-sectional study of 624 Japanese middle-aged men and found that adiponectin level was a more significant predictor than TNFa, IL-6 or CRP for prevalent Metabolic Syndromes, although age and smoking were the only covariates adjusted for in their analysis. However, Wannamethee et al (21) in the British Regional Heart Study of 3640 nondiabetic men aged 60–79 years did adjust for BMI among other covariates and found that the likelihood of MetS decreased significantly with increasing adiponectin. Nevertheless, measures of insulin resistance and inflammation were not included as covariates in the model.

Level of adiponectin and its association with developing Type2-diabetes in different studies:

Plasma adiponectin levels are significantly decreased in T2D subjects compared to the healthy individuals. Genetic polymorphisms in linkage disequilibrium blocks of the AdipoQ gene, including the promoter region and the boundary of exon 2 & intron 2, are associated with T2D, obesity, diabetic nephropathy and insulin resistance. Therefore, plasma/serum adiponectin levels and genomic DNA polymorphisms in the AdipoQ gene can be used as the biomarkers for early diagnosis and clinic prediction of diabetes, obesity, diabetic complications and other metabolic disorders. Evaluation of adiponectin levels with the ratio of HMW (LMW and MMW) and consideration of different ethnic genetic backgrounds are of importance in the translation research of adiponectin (14). Plasma adiponectin levels in type 1 diabetes patients and in the patients with nephropathy are increased compared to non-diabetic individuals or the patients without nephropathy. The possible mechanism concerning different adiponectin levels in type 1 and type 2 diabetes is due to adiponectin modulation and action in related to C-peptide (23).Theoretical evidences suggest adiponectin plays an important role on insulin sensitivity, insulin ressistance and β-cell dysfunction. Reduction of plasma/serum adiponectin levels is significantly related to the development of diabetes (24).

In humans, a broad spectrum of deleterious consequences has been associated with lower adiponectinemia. It includes enhanced risks of T2D, but the association between circulating adiponectin concentrations have a strong relationship with T2D risk (25). Reduced adiponectin levels mainly stem from lower levels of its HMW form in patients with CVD or insulin resistance (26-27). Human adiponectin gene mutations, which specifically impair the formation of HMW (high molecular weight) adiponectin hexamers, have consistently been associated with T2D (28). Those observations suggest that the HMW - adiponectin form is the most pathologically relevant. Thus, it is clinically pertinent to identify a means of restoring normal adiponectin levels and, more specifically, the HMW form (29).

Conclusion

Adiponectin is a target for future research in reducing morbidity and mortality of type-2-diabetes. Diet, exercise, diabetic drugs, and insulin sensitizers improve endothelium-dependent vascular function, increase adiponectin levels, and reduce inflammation and insulin resistance by distinct mechanisms. This may help explain beneficial effects of combination therapies in recent clinical trials.The plasma/serum adiponectin levels and genomic DNA polymorphisms in the AdipoQ gene can be used as the biomarkers for early diagnosis and clinic prediction of diabetes, obesity, diabetic complications and other metabolic disorders Thus, there are a scientific rationale for recommending a combination of lifestyle modifications and multiple drugs from separate classes to prevent type-2-diabetes in prediabetes stage. Prospective studies are needed to examine the ability of increase in adiponectin levels and insulin sensitivity to improve primary end points

including incidence of diabetes and outcomes of cardiovascular events. It is possible that recombinant adiponectin may have a beneficial therapeutic role in the treatment and prevention of type-2-diabetes in the future**.**

References:

1. Sattar N (2008) why metabolic syndrome criteria have not made prime time: a view from the clinic. Int J Obes (Lond) 32(Suppl 2): S30–S34.
2. Snehalatha C, Mukesh B, Simon M, et al.: Plasma adiponectin is an independent predictor of type 2 diabetes in Asian Indians. Diabetes Care 26:3226 –3229, 2003.
3. Lindsay RS, Funahashi T, Hanson RL, Matsuzawa Y, Tanaka S, Tataranni PA, Knowler WC, Krakoff J: Adiponectin and development of type 2 diabetes in the Pima Indian population. Lancet Res Lett 360:57–58, 2002.
4. Yamauchi T, Kamon J, Minokoshi Y, Ito Y, Waki H, Uchida S et al. Adiponectin stimulates glucose utilization and fatty-acid oxidation by activating amp-activated protein kinase. Nat Med 2002; 8: 1288–1295.
5. Fumeron F, Aubert R, Siddiq A, et al.: Adiponectin gene polymorphisms and adiponectin levels are independently associated with the development of hyperglycemia during a 3-year period: the Epidemiologic Data on the Insulin Resistance Syndrome Prospective Study. Diabetes 53:1150 –1157, 2004.
6. Goldstein BJ, Scalia R. Adipokines and vascular disease in diabetes. Curr Diab Rep 2007; 7:25–33.
7. Knobler H, Benderly M, Boyko V, et al. Adiponectin and the development of diabetes in patients with coronary artery disease and impaired fasting glucose. Eur J Endocrinol 2006; 154:87–92.
8. Choi KM, Lee J, Lee KW, Seo JA, Oh JH, Kim SG et al. Serum adiponectin concentrations predict the developments of type 2 diabetes and the metabolic syndrome in elderly Koreans. Clin Endocrinol 2004; 61: 75–80.
9. Berg AH, Combs TP, Du X, Brownlee M & Scherer PE 2001 The adipocyte-secreted protein Acrp30 enhances hepatic insulin action. Nature Medicine 7 947–953.
10. Yunna Jiang1, Ibiye Owei1, Jim Wan2, Sotonte Ebenibo1, Samuel Dagogo-Jack1 Adiponectin levels predict prediabetes risk: the Pathobiology of Prediabetes in A Biracial Cohort (POP-ABC) study. http://dx.doi.org/10.1136/bmjdrc-2016-000194.
11. Galic S, Oakhill JS, Steinberg GR. Adipose tissue as an endocrine organ. Mol Cell Endocrinol. 2010; 316:129–139. doi: 10.1016/j.mce.2009.08.018.
12. Lindsay RS, Funahashi T, Hanson RL, et al. Adiponectin and development of type 2 diabetes in the Pima Indian population. Lancet 2002; 360:57–8.
13. Spranger J, Kroke A, Mohlig M, Bergmann MM, Ristow M, Boeing H, Pfeiffer AFH: Adiponectin and protection against type 2 diabetes mellitus. Lancet Res Lett 361: 226–228, 2003.
14. Harvest F. Gu, Biomarkers of Adiponectin: Plasma Protein Variation and Genomic DNA Polymorphisms. Biomarker Insights 2009:4 123–133,2009.
15. Hu E, Liang P, Spiegelman BM. AdipoQ is a novel adipose-specific gene dysregulated in obesity. J Biol Chem. 1996; 271:10697–10703. doi: 10.1074/jbc.271.18.10697.
16. Matsuzawa Y. Adiponectin: identification, physiology and clinical relevance in metabolic and vascular disease. Atherosclerosis Supplements 2005; 6: 7–14.
17. Hara K, Horikoshi M, Yamauchi T, Yago H, Miyazaki O, Ebinuma H, Imai Y, Nagai R, Kadowaki T. Measurement of the high-molecular weight form of adiponectin in plasma is useful for the prediction of insulin resistance and metabolic syndrome. Diabetes Care. 2006; 29:1357–1362. doi: 10.2337/dc05-1801.
18. Choi KM, Lee J, Lee KW, et al. Serum adiponectin concentrations predict the developments of type 2 diabetes and the metabolic syndrome in elderly Koreans. Clin Endocrinol, 2004;61:75–80.
19. Mohan V, Deepa R, Pradeepa R, Vimaleswaran KS, Mohan A, Velmurugan K et al. Association of low adiponectin levels with the metabolic syndromeFthe Chennai Urban Rural Epidemiology Study (CURES-4). Metabolism 2005; 54: 476–481.
20. Matsushita K, Yatsuya H, Tamakoshi K, Wada K, Otsuka R, Takefuji S et al. Comparison of circulating adiponectin and proinflammatory markers regarding their association with metabolic syndrome in Japanese men. Arterioscler Thromb Vasc Biol 2006; 26: 871–876.
21. Wannamethee et al., Association between adiponectin levels and coronary heart disease and mortality: a systematic review and meta-analysis.
22. M. Guerre-Millo / Diabetes & Metabolism 34 (2008) 12–18)) . Adiponectin: An update - 11/01/17 Doi: 10.1016 / j.diabet.2007.08.002.
23. Mather KJ, Funahashi T, Matsuzawa Y, et al. Adiponectin, change in adiponectin, and progression to diabetes in the Diabetes Prevention Program. Diabetes 2008;57:980–6.

24. Daimon M, Oizumi T, Saitoh T, et al.: Decreased serum levels of adiponectin are a risk factor for the progression to type 2 diabetes in the Japanese population: the Funagata Study. Diabetes Care 26:2015–2020, 2003.

25. Hung J, McQuillan BM, Thompson PL, Beilby JP. Circulating adiponectin levels associate with inflammatory markers, insulin resistance and metabolic syndrome independent of obesity. Int J Obes (Lond) 2008; 32:772–779. doi: 10.1038/sj.ijo.0803793.

26. Kobayashi H, Ouchi N, Kihara S, Walsh K, Kumada M, Abe Y, et al.Selective suppression of endothelial cell apoptosis by the high molecular weight form of adiponectin. Circ Res 2004; 94:e27–31.

27. Lara-Castro C, Luo N, Wallace P, Klein RL, Garvey WT. Adiponectin multimeric complexes and the metabolic syndrome trait cluster. Diabetes 2006; 55:249–59.

28. Waki H, Yamauchi T, Kamon J, Ito Y, Uchida S, Kita S, et al. Impaired multimerization of human adiponectin mutants associated with diabetes. Molecular structure and multimer formation of adiponectin. J Biol Chem 2003;278:40352–63.

29. Han SH, Sakuma I, Shin EK, Koh KK. Antiatherosclerotic and anti-insulin resistance effects of adiponectin: basic and clinical studies. Prog Cardiovasc Dis. pp. 126–140.

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Indian College of Physicians

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5.2.1.2 Continuous membership of the Association of Physicians of India for not less than 7 yrs.

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b.				
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Name of Hospital / Clinic / Organisation & Location	Number of Beds (if applicable)	Period Served year wise (From-To)

7.	Publications: List below. (If number of publications in Journals exceeds 8, publications which can qualify as research papers may be listed under Research section 9.)

a) Number of Publications in Indexed National / International Journals.	Attach title page / Abstract as Appendix
b) Number of Chapter in Books / monograms	
c) Editorship of National level or State level: Book /Monogram/Update Series	

8.	Honours And Awards (list below with photocopy of proof)

(a) Oration in National / State Association Meeting

Title of Oration	Organisation	Year

8 (b) Award National / International / or State level		
Title of Award	Organisation	Year

9.	Research work (list below)	
(a) Research sanctioned & funded by Research Agency		Attach Letter of sanction.
(b) Departmental Research. (To qualify, the findings should be published in National/International Journal) Do not include papers already listed under Publications		Attach title page / Abstract

10.	Contribution to API (list below and attach proof)		
Post held in Organisation / Meeting	Name of Organisation / Meeting / CME	National / Zonal / Under API/ICP	Year

11.	Participation in CME or Scientific Sessions of API or ICP as Faculty		
Speaker / Chairperson / Other	Title of Talk / Session	Name of Meeting	Year

12.	Social welfare / Community service. (Include under the headings given below, with documentary evidence) (a) Emergency services during National calamities (Quakes/ Floods/Cyclones, etc) (b) Public education Programme (Radio), TV talk/ writing in news papers . (c) Service in Rural Areas	
Service		Evidence

N.B : No handwritten application will be accepted. * To be typed on separate page
*One original and seven Xerox copies of sets to be submitted
Last date for receiving the application form is 31st May 2011.
Address : Turf Estate, No. 006 & 007, Dr. E. Moses Road, Opp. Shakti Mill Compound, Mahalaxmi (West), Mumbai – 400 011.

Indian College of Physicians

Citation

The Fellows proposing and seconding the nomination for Fellowship of Indian College of Physicians should highlight the professional / scientific achievements of the candidate and the contribution to A. P. I. from personal knowledge in 200 words, in the format given below :

Name _____ Name _____

Membership No._____ Membership No. _____

Signature Proposer _____ Signature Seconder _____

Note:- The Fellowship form should be proposed and seconded by Founder Fellow / Fellow of ICP only. In case there are more than 3 nominations by any proposer/seconder, the first three nominations in order of receipt in API Office and complete in all respects will be considered for award of Fellowship of ICP and the others rejected for consideration.

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#ADA: American Diabetes Association
^ICMR: Indian Council of Medical Research
1. Diabetes Care. 2014;37:S14-S80