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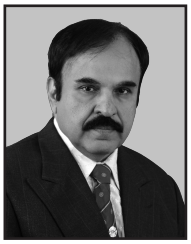

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Editor's Note

Dear colleagues,

Greetings of the season!

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

This issue consists of three interesting case reports on 'Descending Thoracic Aortic Aneurysm', 'Ectopic Right Thoracic Kidney' and 'Rare cause of chest pain- Spondyloarthritis'. This issue also contains valuable articles in the areas of ECG, Dermatology and Toxicology which enrich our knowledge and helps to remain updated in the field.

In addition, we have included an article on 'Fruits for People with Diabetes' in the section 'Getting Back to Basic Medical Science' that enhance our understanding on the Glycemic index and Glycemic load of various fruits. Besides these, we also have an interesting review article entitled "Yoga as a tool in the management of type2 diabetes mellitus" which highlights the impact of yoga in the self-management of diabetes.

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
MD., Ph.D., FRCP (London & Glasgow)

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Review Article

TAPI Journal Vol. 11, Issue 2, May – August 2018

Yoga as a Tool in the Management of Type2 Diabetes Mellitus

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Abstract

Diabetes has earned the status of an epidemic in India, with over sixty two million individuals presently afflicted with this disease. Because of the chronic nature of the disease and the socio-economic burden it causes to the families and nation, adjuvant therapies/life style modifications for correct regulation of polygenic disease is gaining prominence. Yoga includes follow of a number of physical exercises and postures, regulation of respiration with a range of breathing exercises that improve muscle strength, flexibility, blood circulation and oxygen uptake. Yoga is commonly combined with nutrition and practice of meditation that may be an aware process that induces relaxation and stress management. Yoga has been shown to own helpful effects on regulation of blood pressure, glycemic management, sleep quality and weight reduction. Yoga has been established to have beneficial effects on conditions like hypoglycemic agent resistance, diabetes. more constrained information recommend that yoga could likewise bring down aerobic stress and blood pressure and it additionally decreases drug use in grown-ups with type 2 DM. Notwithstanding, given the method impediments of existing examinations, additional high notch examinations are needed to affirm and to boot illustrate the potential benefits of yoga programs in populations with type 2 DM. The aim of this review was to grasp the impact of yoga on the self-management of type2 DM.

Keywords: Diabetes, Yoga, Complementary therapies, Self – Management

Introduction

Diabetes mellitus is a chronic, progressive metabolic disorder wherein the body does not produce enough insulin or does not use the insulin effectively leads to high blood glucose levels, uncontrolled blood sugar may lead to various complications such as retinopathy, neuropathy nephropathy and cardiovascular disease in the long run. Diabetes is on the ascent. No longer a disorder of prevalently rich countries, the predominance of diabetes is consistently expanding all over the place, our country is about to reach the capital in the world soon which rises socioeconomic burden. Estimates suggest that 422 million adults were living with diabetes in 2014, compared to 108 million in 1980.Over the past decade, diabetes prevalence has risen faster in low and middle-income countries than in high-income countries (WHO Report)[1].

While there are many medications that are available to treat this disorder lifestyle intervention acts as an adjuvant in improving the adherence to medications. Yoga is a traditional Indian practice that combines physical activity with lifestyle advice and body awareness techniques such as breath control and meditation[2, 3]. Yoga keeps body and mind under control. Yoga is thought to relieve stress and mental symptoms by the increased balance of body, thoughts, and emotions[3]. There are several studies that have studied the effectiveness of yoga as a complementary therapy in the management of diabetes mellitus.

Therefore, the aim of this review was to assess the effectiveness of yoga in patients with Diabetes mellitus and also discuss the limitations in the available literature in directing for future research. This review focuses on published research articles indexed in the PubMed, MEDLINE, Google Scholar, Science Direct and Scopus. Search criteria included research articles written in English with the keywords "yoga", "diabetes mellitus (DM)", "Type 1 DM", "Type 2 DM", "Exercise". Only clinical or human studies

published in the English language were included. Exclusion criteria included articles that were not written in English. The articles included in the review were based on research in India (2000-2017). Studies in adults with diabetes mellitus, as per ADA guidelines which included any form of yoga with any duration in comparison with control, were considered for this review.

The yogasanas such as Vakrasana (strengthens the spine and activates nerves), Paschinottasan (relieves constipation and activates the nervous system), Mandukasana (the exocrine gland is aroused by hyperbolic secretion of insulin), Uttanapadasana (corrects exocrine gland malfunction and reduces further weight in abdominal areas), Naukasana (better digestion and reduction of blood sugar), Bhujangasan (strengthens the spine and improves circulation of blood) and Trikonasan (Stimulates abdominal organs). Kapalbadhi and Nadisudhi is the most important pranayamas[3] assigned to the diabetic subjects.

Effect of Yoga on blood glucose, Glycosylated hemoglobin, and lipid profile levels

Several studies [4-19] have reported the significant reduction in fasting, postprandialand glycosylated hemoglobin levels. Insulin resistance and type 2 diabetes are associated with dyslipidemia features which in turn is associated with the cardiovascular dysfunction studies has reported that yoga regulates this dyslipidemia condition by increasing the High-density lipoprotein and decrease in low-density lipoprotein, Total cholesterol also the triglyceride levels in comparison to usual care alone.

Effect of Yoga on hypertension

Hypertension is one of the co-morbidities associated with diabetes. Bernard et al. [21] (2012) reported that hypertension and diabetes might share the common metabolic pathway. Obesity; Inflammation and oxidative stress are common pathways in these two disorders. Physical activity plays a major role in controlling this condition. Hypertension also leads to atherosclerosis. In agreement with this, studies have shown the reduction in blood pressure with regular yogic practice[17-20].

Effect of Yoga on body weight

Ideal body mass index is strongly recommended in the health management especially in case of diabetes mellitus. Studies have shown that obesity leads to a poor response of insulin-producing cells and diabetes is also seen in people who are underweight. Studies were done by Sahay[17] (2007) has shown the regular practice of yoga has improved glycemic control by the reduction of body fat among lean diabetics. There are also several other studies which have reported similar findings.[6,8,19,20]

Effect of yoga on Oxidative stress

Oxidative stress is the condition when there is an imbalance in the antioxidants and pro-oxidants levels in the body due to several factors such as aging, toxicity, inflammation etc [22]. This oxidative stress causes damage to enzymes and increases in insulin resistance since there is less availability of antioxidants to neutralize the free radicals that are generated during the energy production processes such as glycolysis, Tricarboxylic acid cycle and various other pathways in the body. It is believed that oxidative stress plays important role in the development of vascular complications in diabetes particularly type 2diabetes [22]. A three month Study done by Hegde et al[23] (2011) has reported that yoga resulted in significant reduction in BMI, glycemic control, and malondialdehyde and increase in glutathione and vitamin C among type 2 diabetic subjects. Another study was done by Shree Lakshmi[24] et al (2016) also reported the similar findings among elderly type 2 diabetic subjects. A preliminary report done by Singh [13] et al (2001) has shown the reduction in malondialdehyde levels with regular practice of yoga for 40 days in addition to drug treatment and diet control among type 2 diabetic subjects.

Effect of Yoga on the psychosomatic aspect of type 2 diabetes

Diabetes is associated with psychological disturbances such as stress, anxiety, and depression. Diabetes and psychological factors are linearly correlated to each other. Stress induces changes in dietary intake. A review done by Vincent[24] et al (2015) has examined the extent to which the type 2 diabetes mellitus is associated with impairments in the executive function it was found that type 2 diabetes mellitus is associated with mild to moderate level of impairment in executive function stronger in those with short disease duration. Yoga is the form of physical activity that controls both mind and body. It is hypothesized that yoga combats stress levels by regulating the hypothalamus – pituitary – adrenal axis. Studies have reported the improvement in stress management which is linked to glycemic control among type 2 diabetics through regular yoga practice. Pranayama involves breathe control improves the pulmonary function. It has been shown by Satish [25] et al (2016) and Sreedevi[26] et al (2017) that yoga improves the quality of life reduction in the frequency of depressive symptoms and intensity of depression concentration and attention span improved significantly. The discrepancy score also reduced. There was an improved mood and concentration. It also resulted in a sense of well-being.

Conclusion

Blood glucose monitoring is vital to avoid complications due to diabetes. Diabetes being the multi-facet disorder certain factors is responsible for the proper management of blood glucose levels. Proper diet control, regular physical exercise, calm mind along with medications will help us in the management of diabetes. Yoga is the form of physical activity which acts as a key for mind and body control. All the studies mentioned above have shown that yoga has the linear relationship with the stress levels as well as the executive function in the body. It is also a physical activity. It is evident that yoga acts as the adjuvant along with medication. Adherence to medication is much better with regular practice of yoga. Though studies included in this review have found the effect of yoga in direct and indirect control of diabetes the mechanism through which it does these wonders is yet a mystery. It is hypothesized that yoga might regulate all this through the hypothalamus- pituitary- adrenal axis. Long-term well-designed studies are required to unlock this mystery. However, studies showing the effect of yoga on diabetic complications are not available. Knowing that yoga has the positive role of oxidative stress which is associated with diabetic complications, future research on the effect of yoga on life-threatening vascular complications could be studied helping in the tertiary management of this disease.

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An Interesting Case of Descending Thoracic Aortic Aneurysm- A Rare Case Report

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Absract

Aneurysm is described as the irreversible dilation of the artery by way of greater than 50% of the everyday diameter. The incidence of thoracic aortic aneurysm (TAA) is 4.5 per lakh human beings. Frequently, atherosclerosis is a circumstance manifested with the aid of high blood pressure-brought on arterial wall weakening and rupture. Genetic illnesses (Ehler Danlos Syndrome-EDS, Marfan Syndrome and aneurysms osteoarthritis syndrome), temporal arteritis, cystic medial necrosis, infections (mycotic infections, tuberculosis, syphilis) and trauma play a position inside the aetiology. Thoracic aortic aneurysm is generally asymptomatic and is commonly diagnosed with various imaging techniques performed for unrelated problems. Patients with TAA have regularly had some existence-threatening headaches, which includes aortic rupture, dissection and mortality. We hereby excited to report a case of asymptomatic thoracic aortic aneurysm.

Case Report

57 years old non diabetic, non hypertensive male complaints of dull aching left sided chest pain, not radiating associated with difficulty in breathing on exertion for 3 days, chronic smoker and alcoholic. On examination he was conscious, oriented, afebrile, no pallor, icterus, cyanosis, clubbing, pedal edema, lymphadenopathy, PR - 88/min regular rhythm, normal volume, no radioradial, radiofemoral delay, BP - 120/80 mmHg in both upper limbs in supine position, BP - 110/70 mmHg in both lower limbs, RR - 18/min , abdomino thoracic, SPO2 - 99%,CVS: S1 S2 ⊕, no murmur, RS: NVBS ⊕, P/A: soft, CNS: NFND, Routine blood investigations and cardiac enzymes had been normal. Chest xray PA view confirmed mediastinal widening and prominent aortic knuckle. CT angiogram showed big saccular aneurysm in descending thoracic aorta for a length of 11.5cm in descending thoracic aorta. Diffuse irregular atherosclerotic wall thickening of distal arch, thoracic and in abdominal aorta. No evidence of coarctation of aorta, thrombosis or dissection.

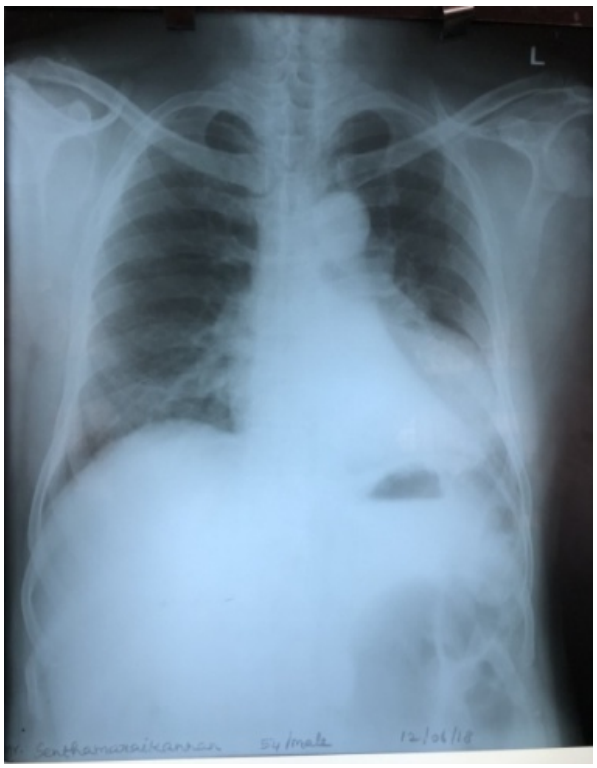

Figure1: Chest xray PA view showing mediastinal widening and prominent aortic knuckle

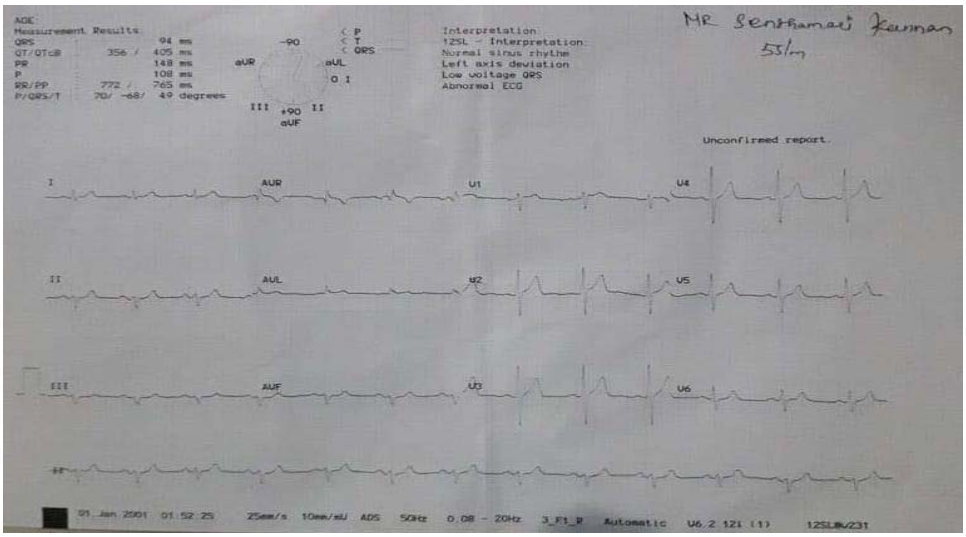

Figure 2: ECG- Within Normal Limits

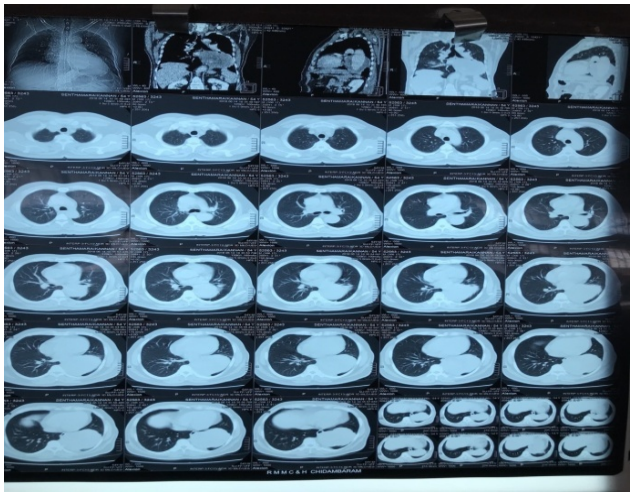

Figure 3: CT angiogram of Thorax

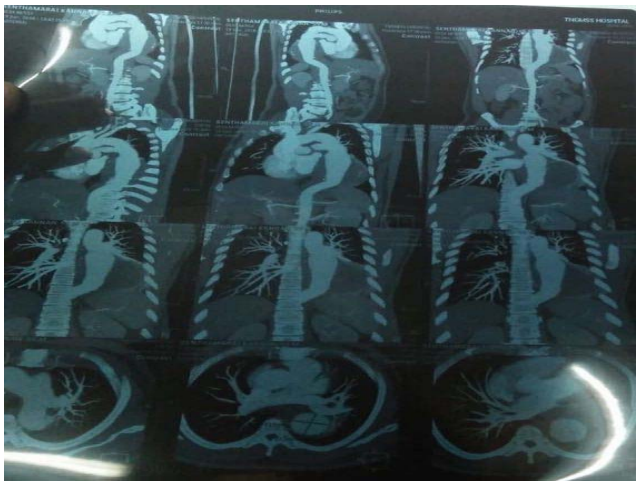

Figure 3: CT angiogram of Aorta

Discussion

Rupture of TAA and dissections are very uncommon, in spite of the very excessive morbidity and mortality costs. Therefore, early detection is vital. Thoracic aortic aneurysms are normally asymptomatic (about 75%), but pain is known as the fundamental referable symptom in approximately 17% of patients [7]. Although generally asymptomatic, chest pain, hoarseness due to recurrent laryngeal nerve compression, compression of the oesophagus causing difficulty in swallowing and bronchial compression causes shortness of breath may be seen [8]. In our case patient presented with chest pain and difficulty in breathing with Chest xray PA view showing mediastinal widening and prominent aortic knuckle and CT angiogram showed big saccular aneurysm in descending thoracic aorta for a length of 11.5cm in descending thoracic aorta.

In addition in the descending aorta, the aneurysms can be saccular or fuzzy form and might press onto the oesophagus and trachea. Moreover, in the actual aneurysms, all layers of the aorta wall can be affected. In our case, a thoracic saccular aneurysm was seen, which spanned all layers of the aorta, no dysphagia was observed .In our case, the patient had no genetic or systemic disease, no infection, and no history of trauma. Atheromatous plaque findings in CTT also suggested an aneurysm. It is unknown whether atherosclerosis is caused by deformities in vascular structure caused by aneurysms or atherosclerotic aneurysms are caused by atherosclerosis.

Conclusion

Thoracic aortic aneurysm is usually asymptomatic and is usually diagnosed with imaging techniques performed for unrelated reasons. Even though it is asymptomatic affected person may require regular follow up and in earlier intervention can be needed to prevent further complications.

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Ectopic Right Thoracic Kidney – A Rare Case Report

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Abstract

Intrathoracic kidney is an extraordinary congenital abnormality with the lowest frequency among all renal ectopics. Renal ectopia refers to a kidney situated in any region aside from the renal fossa. Ectopic kidneys are idea to arise in approximately 1 in 1000 births, but only 1 in 10 of those is ever identified. With a prevalence fee of less than 0.01%, intrathoracic kidneys represent much less than 5% of all renal ectopics; indeed, it has the lowest frequency rate among all renal ectopics. It has therefore a reported incidence of less than 5 per 1 million births. It is generally seen as an incidental finding detected on chest radiograph simulating a posterior mediastinal mass and mandating further evaluation. We present a similar case encountered in our routine clinical practice.

Case Report

83 years old male presented with complaints of breathlessness for 3 days duration. He was a known case of COPD and systemic hypertension. Patient was not a diabetic. He was a chronic smoker and alcoholic. On examination patient was conscious, oriented, no pallor / icterus / cyanosis/ clubbing/ pedal edema. PR- 102/min, regular, normal volume, no specific character BP- 150/100mmHg, RR- 18/ min. CVS-S1S2+ , RS- NVBS+ , BAE+, Occasional wheeze+, P/A- Soft, CNS- NFND. Routine blood research were normal. Chest X-Ray showed opacity over base of right lung. Ultrasonogram abdomen showed features suggestive of ectopic right thoracic kidney. The computed tomography (CT) scan elegantly demonstrated the presence of ectopic reniform structure in the intrathoracic location on the right side

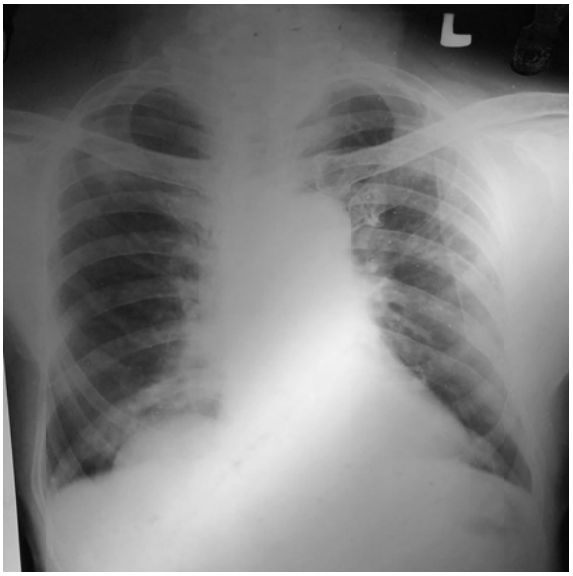
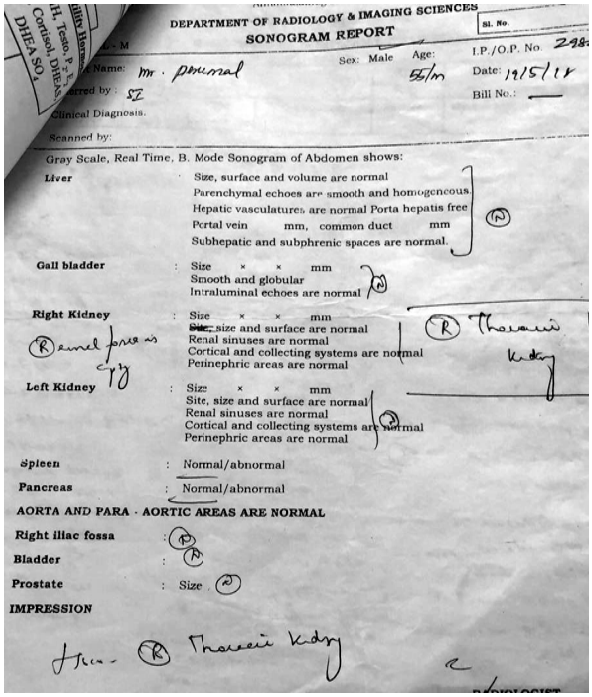


Figure 1. Chest X-Ray showed opacity over base of right lung


DEPARTMENT OF RADIOLOGY & IMAGING SCIENCES

SONOGRAM REPORT

Sl. No.

Name: Mr. Perumal Sex: Male Age: 55/m I.P./O.P. No. 298

Date: 19/5/18

Bill No.:

Clinical Diagnosis.

Scanned by:

Gray Scale, Real Time, B. Mode Sonogram of Abdomen shows:

Liver : Size, surface and volume are normal
Parenchymal echoes are smooth and homogeneous
Hepatic vasculatures are normal Porta hepatis free
Portal vein mm, common duct mm
Subhepatic and subphrenic spaces are normal. (N)

Gall bladder : Size × × mm
Smooth and globular
Intraluminal echoes are normal (N)

Right Kidney : Size × × mm
Site, size and surface are normal
Renal sinuses are normal
Cortical and collecting systems are normal
Perinephric areas are normal

Left Kidney : Size × × mm
Site, size and surface are normal
Renal sinuses are normal
Cortical and collecting systems are normal
Perinephric areas are normal

Spleen : Normal/abnormal

Pancreas : Normal/abnormal

AORTA AND PARA - AORTIC AREAS ARE NORMAL

Right iliac fossa : (N)

Bladder : (N)

Prostate : Size (N)

IMPRESSION

(R) Thoracic Kidney

Figure 2. USG Abdomen showed f/s/o Right Thoracic kidney

Discussion

Intrathoracic kidney is a partial or complete protrusion of the kidney above the

hemidiaphragm into the posterior mediastinal compartment of the thorax. The first case of thoracic kidney was diagnosed by Wolfromm in 1940 using retrograde pyelography. Since then, very few such cases have been reported. This condition shows male predominance and occurs usually at the left than on the right side. Ten percentage of cases are bilateral. It is noteworthy that in all cases, the kidney is located in the thoracic cavity not in the pleural space, with renal vessels and ureter normally exiting the thorax via the foramen of Bochdalek.

Various mechanisms have been thought to be responsible for intrathoracic kidneys such as accelerated ascent of the kidney, delayed closure or maldevelopment of the pleuroperitoneal membrane, effect of the developing liver and adrenal glands, and the persistence of the nephrogenic cord. During embryogenesis, the kidneys are initially situated within the pelvis; then, they ascend into the abdomen because the caudal portion of the embryo grows relative to cranial. Ascent stops whilst the kidneys reach the adrenals. In actuality, each kidneys are bodily hindered from higher ascension predominantly by superiorly positioned adrenals and, to some extent by the liver. Thus, under conditions affecting the development of adrenal glands and liver, the ascending developing kidney may rarely "overshoot" and ascend to a higher location than normal, ensuing in thoracic ectopia. However, none of these postulated mechanisms can totally explain all the reported cases.

Most patients with intrathoracic kidneys are asymptomatic and have a benign clinical course. However, anatomically, rotational anomalies (such as hilum facing posteriorly, lengthy ureter, high origin of renal vessels) and medial deviation of lower pole of kidney may be seen. Associated anomalies in other organs are extremely rare.

Several techniques were used to diagnose intrathoracic kidney. Plain radiographs are often indeterminate and might confuse this condition with other posterior mediastinal lesions along with Bochdalek hernia, pulmonary sequestration, or neurogenic masses. In the past, intravenous urography become the modality of choice for confirming the diagnosis, but it's been outmoded by ultrasonography and CT scan these days. In our case we incidentally found an ectopic thoracic kidney over right side in an 83 years old male.

Conclusion

Intrathoracic renal ectopia is a rare clinical entity and is a diagnostic challenge for both clinicians and radiologists. Awareness of this abnormality together with a high index of suspicion may additionally obviate the need for unnecessary investigations and operative approaches. Treatment isn't always necessary in majority of ectopic thoracic kidney except in the ones associated with other anomalies which includes vesico-ureteric reflux and obstruction.

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Rare Cause of Chest Pain- Spondyloarthritis

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Case report

34 yrs old lady presented with back pain, neck pain and chest pain for 2 months on a background history of psoriasis for 8 years. She had no fever, cough or weight loss. She had inflammatory symptoms with morning stiffness, ankle synovitis and tender sternal joints.

Evaluation showed raised CRP, ESR, normal chest X ray and working diagnosis was spondyloarthritis with psoriasis. Her MRI showed features of sacroileitis and also Manubriosternal synovitis (Figure 1 and 2). Her psoriasis was limited to scalp, knees and nails. There was no evidence of bowel or eye involvement.

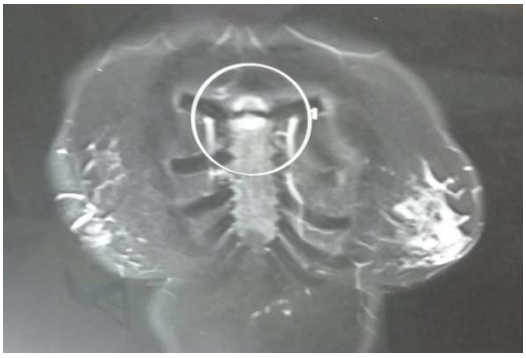

Figure 1 Bone marrow edema with synovitis in Manubrio sternal and sternoclavicular joint

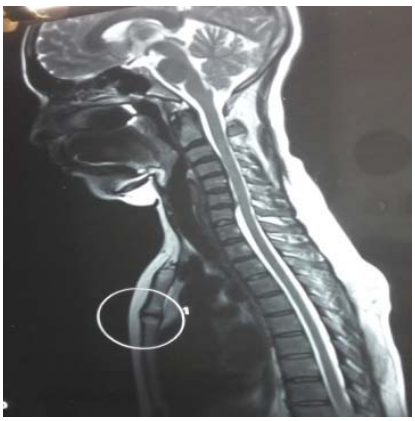

Figure 2. MRI lateral view with Manubrio sternal joint arthritis

Differential diagnosis of the Image

Spondylo arthritis- Unusual to have manubriosternal arthritis. Psoriatic arthritis, - not uncommon to have this arthritis. SAPHO syndrome- Rheumatologists think SAPHO first with this image. (Synovitis, Acne, Pustulosis, Hyperostosis, Osteitis.) Gout,- rare manifestation but need to keep in mind. Septic arthritis- if isolated arthritis- ALWAYS think Sepsis. Osteoarthritis- degenerative joint disease can occur. RA- few case reports but very rare manifestation.

Final diagnosis

Psoriatic Spondyloarthritis and she has been managed with methotrexate and Lornoxicam. She had TNF blocker- Adalimumab given 40mg every fortnight. She is in remission.

Conclusion

Manubriosternal joint arthritis can be difficult to diagnose unless the clinician has high index of suspicion and differential diagnosis should be considered as above.

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Cutaneous Hyperandrogenism

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Introduction

A 23 year old obese female presented with complaints of excessive hair growth over chin and abdomen and dark thickened skin over the neck and axillae since the last three years. She gave history of oligomennorhea. On examination coarse terminal hair was seen over the lower face (hirsutism). Velvety pigmentation and thickening of the skin was seen over the neck and axillae. She also had ice pick and box type of acne scars along with a few closed comedones on the malar area.

Diagnosis

Cutaneous Hyperandrogenism (CHA)

Pathophysiology

The pathophysiology of CHA is complex. Normally androgens are derived from cholesterol and in females they are synthesized by the ovaries and adrenal glands. In extra glandular sites like liver, muscles, skin and adipose tissue steroid conversion of androgen occur. In the pilosebaceous unit and skin, testosterone is converted into dihydrotestosterone (DHT) by the enzyme 5-alpha-reductase 1 or 2. Thereby pilosebaceous unit and skin represent the target structures for androgens which explains the cutaneous manifestations of hyperandrogenism such as hirsutism, acne, seborrhea and alopecia.

Description

CHA are seen in 5-10% of females of reproductive age group. The hallmarks of cutaneous hyperandrogenism are hirsutism, acanthosis nigricans, seborrhea, severe acne, telogen effluvium, and male or female androgenetic alopecia. Other signs of virilization like deepening of voice, male habitus in women and clitoral hypertrophy help to make a straightforward clinical diagnosis of hyperandrogenism.

The most common causes are polycystic ovary syndrome, congenital adrenal hyperplasia and anabolic steroid abuse. The rare causes are Cushing disease, acromegaly, hyperprolactinaemia and androgen secreting tumors of adrenal gland or ovary.

Investigations

The initial diagnosis of CHA is made with relevant menstrual and reproductive history and clinical examination. Blood investigations like fasting and postprandial blood sugar and insulin, follicle stimulating hormone, luteinizing hormone (LH /FSH ratio more than 2), testosterone, dehydroepiandrosterone sulfate and serum prolactin should be done. Ultra sonogram of abdomen and pelvis can be done to rule out polycystic ovaries.

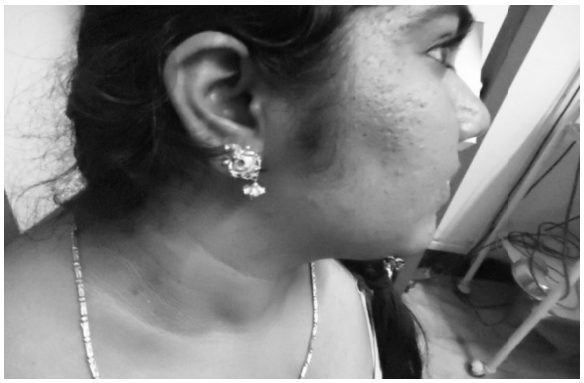

Figure 1

Treatment

Life style modification like diet and exercise is necessary. Cutaneous Hyperandrogenism can be treated with antiandrogens:

a) Cyproterone acetate (25mg to 50mg, in the first 10 days of the cycle): It is a potent progesterone with moderate antiandrogen action. Hirsutism improves after a 3-to-6-month treatment.

b) Spironolactone: It has a moderate antiandrogen effect when administered as a 100-200mg/day dose for 3 months. The duration of treatment can be extended and it can be combined with oral contraceptives.

c) Flutamide: It is a potent nonsteroidal antiandrogen very effective in treating hirsutism, but hepatocellular dysfunction has limited its use.

d) Finasteride: It is a 5-alpha reductase inhibitor, given in a dose of 5mg/day for three-months.

e) Eflornithine cream: It is an inhibitor of the enzyme decarboxylase ornithine which inhibits hair growth.

Diode and Nd-YAG laser hair removal can be tried.

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De Winter's 'T' wave with Precordial 'ST Depression' - An Under Recognised Anterior STEMI Equivalent

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Introduction

Acute left anterior descending (LAD) coronary artery occlusion leading to anterior myocardial infarction (MI) is usually characterized by ST-segment elevations. Rarely, acute anterior MI may present with ST-segment depressions in precordial leads, called deWinter T-wave ECG pattern. Early recognition of this unique ECG pattern is important for appropriate triage. This pattern (Fig.1) was first reported in a 2008 case series by de Winter and Wellens,[1] who observed this ECG pattern in 30 / 1532 patients with acute LAD occlusions (2% of cases). Key diagnostic features include ST depression (upsloping ST segment depression >1mm at the J-point) and tall, prominent, symmetric T waves in the precordial leads. In addition, lead aVR may show slight ST-segment elevation in most cases. "Normal" STEMI morphology may precede or follow the deWinter pattern.

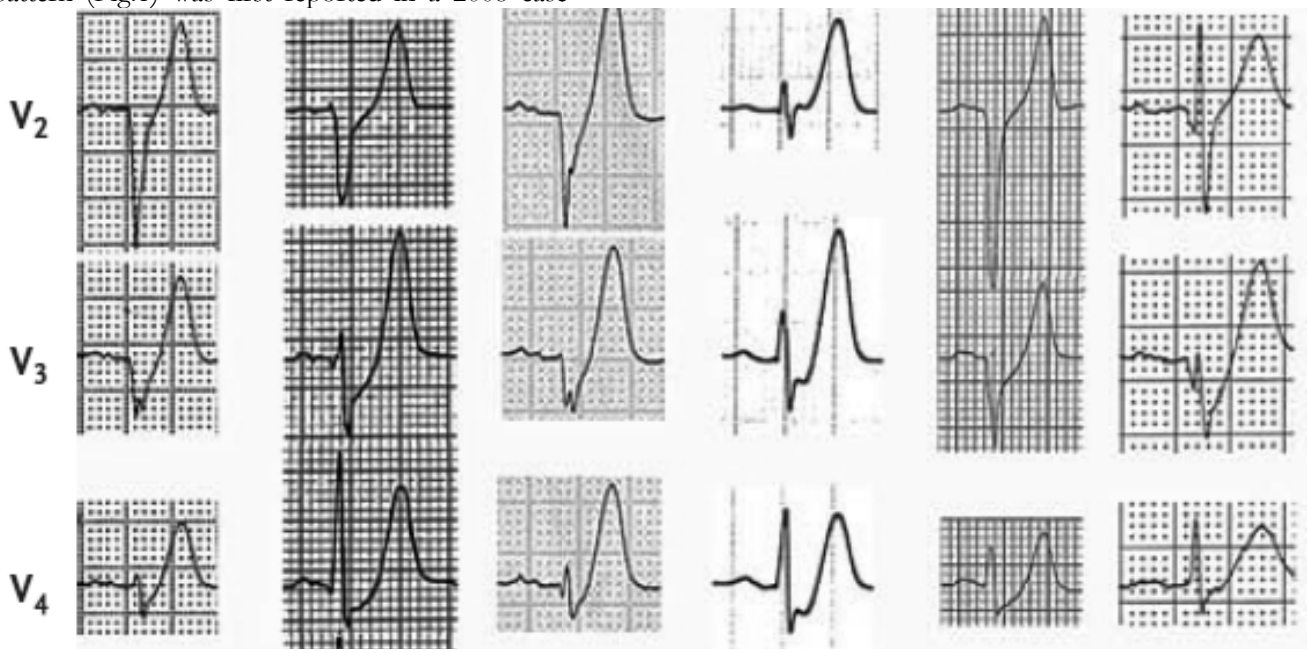

Fig.1 Precordial ST-segment depression at the J point followed by peaked, positive T waves in 6 patients (original case series)

Case report

A 41 year gentleman, non-diabetic, non-hypertensive, smoker had presented with retrosternal burning sensation for one hour, associated with sweating. Initial ECG is described below (Fig.2). Subsequently one hour later his ECG showed STEMI in anterior leads (Fig.3). Then he was thrombolysed with STK. He was DC verted for unstable VT. He had moderate LV dysfunction. CAG after stabilization showed double vessel disease, the culprit vessel being LAD. He subsequently underwent CABG with grafts to LAD/RCA.

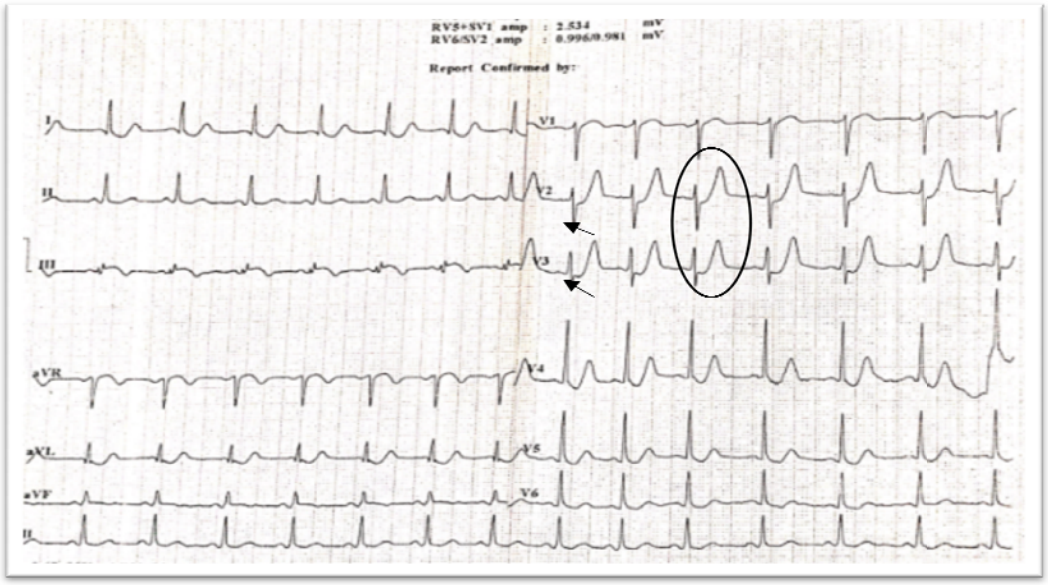

Fig.2 Initial ECG showing ST depression and peaked T waves in precordial leads.

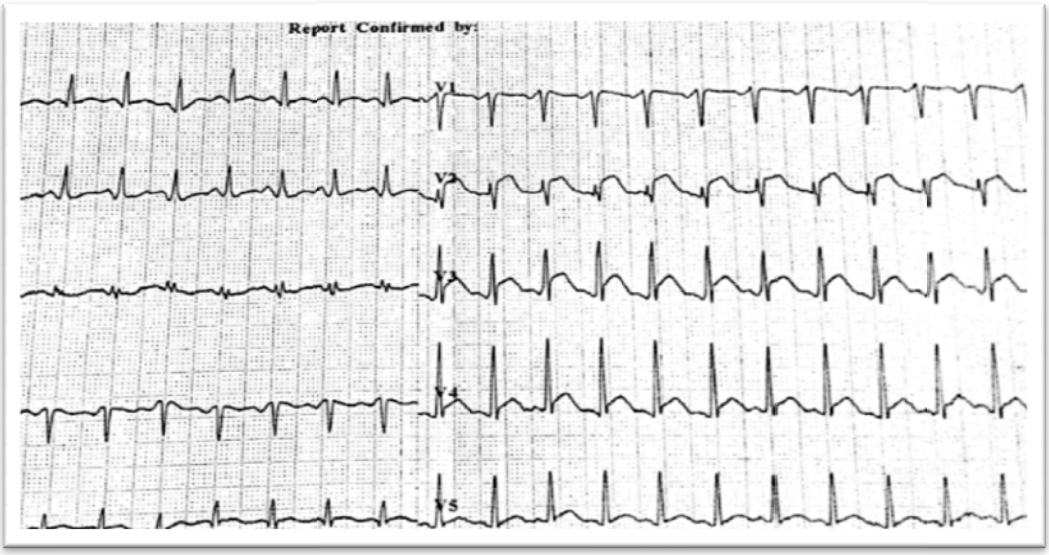

Fig.3 ECG taken 1 hour later showing typical anterior ST elevation.

Discussion

This unique ECG pattern is seen in ~2% of acute LAD occlusions and is under-recognized by clinicians.[2] Unfamiliarity with this high-risk ECG pattern may lead to under-treatment (e.g. failure of cath lab activation/ delay in thrombolytic therapy), with attendant negative effects on morbidity and mortality[3] (delayed thrombolysis, followed by unstable VT in our case). Theoretically the electrophysiological explanation could be an anatomical variant of the Purkinje fibers, with endocardial conduction delay. Alternatively, the absence of ST elevation may be related to the lack of activation of sarcolemmal ATP sensitive potassium (K_{ATP}) channels by ischemic ATP depletion. In the original case series, patients with the de Winter ECG pattern were younger, more likely to be male and with a higher incidence of hypercholesterolaemia compared to patients with a classic STEMI pattern.[4]

Conclusion

Awareness of this rare but highly characteristic deWinter ECG pattern is essential to avoid overlooking anterior MI patients who present with this ECG pattern and prevent undue delay in coronary intervention and revascularization causing morbidity and mortality. This also highlights the importance of serial ECGs in suspected angina.

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Mushroom Poisoning -Mysteries

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Poisoning from ingestion of mushrooms occurs worldwide usually when wild mushrooms containing toxins are misidentified as edible species, collected and eaten. However, severe toxicity from mushrooms is rare in humans.

1.What are the causes of mushroom poisoning?

The main causes of mushroom toxicity are I. Incorrect identification of a mushroom II. Intentional ingestion by a person with euphoric intent III. Intentional ingestion by a suicidal person IV. Foul play in which an individual is poisoned by someone else V. Mushroom might be contaminated with unknown toxic compounds (Internet purchase).

2.How to distinguishing edible mushrooms from poisonous mushrooms?

No simple rule exists for distinguishing edible mushrooms from poisonous mushrooms. In more than 95% of mushroom toxicity cases, poisoning occurs as a result of misidentification of the mushroom by an amateur mushroom hunter. In less than 5% of the cases, poisoning occurs after the mushroom is consumed for its mind-altering properties.

3.How do we classify mushroom poisoning?

Mushroom poisoning is classified into the following 3 major categories on the basis of the time from ingestion to the development of symptoms.

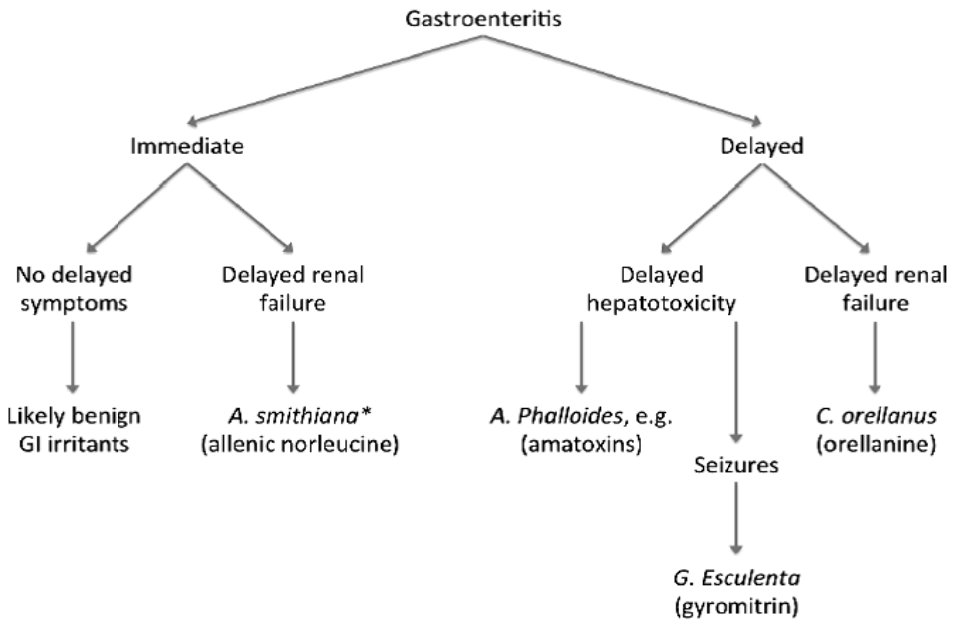
- **Early symptom category** – Symptoms generally appear within the first 6 hours of mushroom ingestion and include gastrointestinal (GI), allergic, and neurologic syndromes
- **Late symptom category** – Signs and symptoms begin to appear between 6 and 24 hours after ingestion and may include hepatotoxic, nephrotoxic, and erythromelalgic syndromes
- **Delayed symptom categories** – Symptoms appear more than 24 hours after ingestion and include mostly nephrotoxic syndromes.

4.What is the typical acute presentation of mushroom poisoning?

The most commonly ingested poisonous mushrooms are gastrointestinal irritants. These mushrooms cause self-limited nausea, vomiting, diarrhoea and stomach pain.

5.How long after exposure do symptoms develop?

The onset of symptoms is rather rapid (0.5 to 3 hours). In contrast, there is a characteristic delayed onset of symptoms (> 6 hours) for more severe types of poisoning, such as toxicity associated with cyclopeptide toxicity (amatoxin, phallotoxin, virotoxin). The timing of symptom onset has long been considered crucial for differentiating life-threatening or severe mushroom poisonings from less serious ones.



6.What is the Mushroom Poisoning Syndrome?

There are 12 groups of identified mushroom toxins with 14 described clinical syndromes as given below.

Mushroom poisoning syndrome	**Toxins**	**Onset of symptoms**	**Sites of toxicity**	**Specific mushroom examples**
Acute gastroenteritis without liver failure	GI irritants	<6 hours (most within 3 hours)	GI tract	*Chlorophyllum molybdites*
Hallucinogenic	Psilocybin, psilocin	30 minutes-2 hours	CNS (hallucinogenic effects)	*Psilocybe cubensis*
CNS excitation and depression	Ibotenic acid, muscimol	30 minutes-2 hours	CNS (depressant and excitatory effects)	*Amanita muscaria*
Cholinergic excess	Muscarine	30 minutes-2 hours	Autonomic nervous system (muscarinic receptors)	*Clitocybe dealbata*
Disulfuram-like reaction	Coprine	30 minutes-2 hours	Inhibition of aldehyde-dehydrogenase enzyme leading to increased blood aldehyde	*Coprinus atramentarius*
Gastroenteritis and delayed onset renal failure	Allenic norleucine	30 minutes-3 hours (GI toxicity) 12-24 hours (renal toxicity)	Kidney GI tract	*Amanita smithiana*

Delayed liver toxicity and delayed gastroenteritis	Cyclopeptides: - Amatoxins - Phallotoxins	6-24 hours	GI tract Liver Kidney	*Amanita phalloides*
Seizures, delayed gastroenteritis and liver toxicity	Gyromitrin	4-10 hours	GI tract Central nervous system Liver Blood	*Gyromitra esculenta*
Delayed renal failure	Orellanine, orellinine, cortinarin	3-20 days	Kidney	*Cortinarius orellanus*
Delayed rhabdomyolysis	Unknown	24-72 hours	Muscle	*Tricholoma equestre*
Erythromelalgia	Acromelic acid	>24 hours	P. Nerves Skin	*Clitocybe acromelalga*
Delayed Encephalopathy				
Patients with renal failure	Unknown	>24 hours to days	Encephalopathy	*Pleurocybella porrigens*
Normal healthy patients	Polyporic acid (causes violet colored urine)	>12 hours	Encephalopathy, liver and renal toxicity	*Hapalopilus rustilans*
Immune-mediate hemolytic anemia	Antibodies to *Paxillus involutus*	Repeated ingestion of cooked mushroom	Blood	*Paxillus involutus*
Allergic bronchioalveolitis	Allergic reaction to spores of Lycoperdon species	<6 hours	Lungs	*Lycoperdon* species

7.How to manage Mushroom Poisoning?

In the absence of a definitive identification of the mushroom, all ingestions should be considered serious and possibly lethal. Once mushroom toxicity is diagnosed, treatment is largely supportive. Early volume resuscitation is important for liver and renal toxic syndromes. Gut decontamination, including whole-bowel irrigation, may be necessary for amatoxins. Consider activated charcoal 50g if onset of GI symptoms occurs >6 hours of post-ingestion and multi-dose activated charcoal if suspected cyclopeptide hepatoxicity as alpha-amantin undergoes enterohepatic circulation.

8.What are the antidotes used to treat Mushroom Poisoning?

Currently, there are no FDA-approved antidotes. However, specific therapy depends on the presumed toxin ingested.

- Cyclopeptide hepatoxicity (e.g. GI symptoms onset >6 hours or increasing transaminases)
  - N-acetylcysteine
  - Cimetidine
  - penicilin 1 MU/kg/day
  - silibinin 5 mg/kg IB over 1 hour then 20 mg/kg/day for up to 3 days
- Consider pyridoxine for seizures, methylene blue for methemoglobinemia due to monomethylhydrazine (esculenta) poisoning

- Cholinergic syndrome — atropine or glycopyrrolate
- Anticholinergic poisoning may be treated with benzodiazepines; in rare cases, physostigmine may be required.
- Patients with severe poisoning from disulfiram-containing mushrooms may benefit from fomepizole (4-methylpyrazole)

9.What are Myths vs. Facts on Poisonous Mushrooms?

People pass along misinformation about how to distinguish edible mushrooms from poisonous species, like the following:

Myth: Poisonous mushrooms always have bright, flashy colors.

Fact: Toxic species can be pure white or plain brown.

Myth: Snails, insects or other animals won't eat poisonous mushrooms.

Fact: Just because a mushroom doesn't hurt another animal doesn't mean it won't hurt you.

Myth: Silver or onions blacken toxic mushrooms on touch.

Fact: All mushrooms darken or bruise if damaged.

Myth: Toxic mushrooms smell and taste horrible.

Fact: Some say toxic mushrooms actually taste very good.

Myth: Any mushroom becomes safe if you cook it.

Fact: You can't make a toxic mushroom safe by cooking, canning, freezing or drying.

Pearls

- No simple rule exists for distinguishing edible mushrooms from poisonous mushrooms.
- Mushroom identification is very difficult and often impossible (e.g. unavailable, decomposing, cooked, partially digested)
- Delayed toxicity after mushroom ingestion is potentially life-threatening; provide aggressive supportive care and consider antidote therapy when appropriate

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Fruits for People with Diabetes

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Fruit has been recognized as a good source of vitamins and minerals, and for their role in preventing vitamin C and vitamin A deficiencies. People who eat fruit as part of an overall healthy diet generally have a reduced risk of chronic diseases. Fruit are important sources of many nutrients, including potassium, fiber, vitamin C and folate (folic acid).

Benefits of fruits

1. Fruits are low in calories and fat and are a source of simple sugars, fiber, and vitamins, which are essential for optimizing our health.
2. Fruits provide plenty of soluble dietary fiber, which helps to ward off cholesterol and fats from the body and to help in smooth bowel movements as well as offer relief from constipation ailments.
3. Fruits compose of many **antioxidants** such as **polyphenolic flavonoids, vitamin-C, and anthocyanins.** These compounds, firstly help human body protected from oxidant stress, diseases, and cancers, and secondly; assist the body in developing the capacity to fight against these ailments by boosting our immunity level. Many fruits, when compared to vegetables and cereals, have very high anti-oxidant values, which measures their **"Oxygen Radical Absorbent Capacity" or (ORAC).**
4. Anthocyanins are flavonoid category of polyphenolic compounds found in some **"blue-fruits"** like blue-black grapes, mulberries, blueberries, blackberries, and in many vegetables featuring blue or deep purple pigments. Consumption of fruits rich in blue pigments offers many health benefits. These compounds have potent antioxidant properties that help remove free radicals from the body, and thus provide protection against cancers, aging, and infections. A majority of these pigments in the fruits tend to concentrate just underneath their skin.
5. Fruit's health benefiting properties are because of their richness in vitamins, minerals, micro-nutrients, pigment and anti-oxidants. Altogether, these compounds help the body prevent or at least prolong the natural changes of aging by protecting from damage and rejuvenating cells, tissues, and organs.

Carbohydrates and Fiber

The main nutrient you get from fruits and vegetables is carbohydrates, which you need for providing energy to your body, including your nervous system and your brain. These foods also provide significant amounts of dietary fiber, which is a type of carbohydrate your body can't digest that helps improve digestive function and lower your risk for high cholesterol, heart disease and Type 2 diabetes. Fiber may also help you maintain a healthy weight since it slows the emptying of the stomach and adds bulk to your food so you feel full for longer after eating.

Potassium

Many fruits and vegetables are high in potassium, which you need for counteracting the adverse effects of sodium on blood pressure, nerve and muscle function and regulating electrolytes, which are minerals in your body that carry an electrical charge. Fruits and vegetables that are particularly good sources of potassium include apricots, bananas, sweet potatoes, cantaloupe, celery, potatoes, beans, tomatoes, spinach and winter squash.

Folate

Fruits and vegetables often contain folate, a B-vitamin you need for forming DNA and limiting the risk of neural tube birth defects. Consuming vegetables like Brussels sprouts, asparagus, black-eyed peas, spinach, broccoli, avocado, peas, lettuce and kidney beans will help you increase your folate intake. Fruits tend to be a bit lower in folate than vegetables, but oranges, papaya, cantaloupes and bananas all contain this essential nutrient.

Vitamin C

Vitamin C is an antioxidant that helps limit cell damage from free radicals, heal wounds, keep your gums and teeth healthy and repair body tissue. Some of the best sources of this vitamin include grapefruit, oranges, cantaloupes, kiwi fruit, papaya, mango, pineapple, watermelon, berries, broccoli, tomatoes, winter squash, green leafy vegetables, cauliflower, Brussels sprouts, potatoes, sweet potatoes and bell peppers.

Vitamin A

Vitamin A is essential for proper immune function, reproduction, healthy vision and cell growth. Fruits and vegetables that contain significant amounts of this vitamin include sweet potatoes, carrots, spinach, cantaloupe, mangoes, bell peppers, apricots, black-eyed peas and broccoli. Serving these foods with something that contains a small amount of fat will help you better absorb the vitamin A they contain since it is a fat-soluble vitamin.

People With Diabetes Can Eat Fruit

The American Diabetes Association (ADA) advises that any fruit is fine to eat for a person with diabetes, so long as that person is not allergic to a particular fruit. A meta-analysis published in 2014 in the British Medical Journal found higher fruit intake was significantly associated with a lower risk of type 2 diabetes. The preparation of fruit, however, can affect blood sugar. Fresh fruits are better than processed fruits straight from a can or jar, such as applesauce and canned fruit. Processed fruits also include dried fruit and fruit juices.

What is the glycemic index?

For a person with diabetes, one way to select safe and suitable fruits and other high-carbohydrate foods is to check the glycemic index (GI). GI is a rating of foods on a scale from 1 to 100. The score indicates how quickly the food item may raise blood sugar levels. High GI foods are absorbed faster than medium or low GI foods.

Glycemic load (GL) takes into account the GI of a food plus the amount of carbohydrates in a serving. GL may be a more accurate of assessing how food affects blood sugar management over time. Low-GI and low-GL foods are better for helping control blood sugar levels. It may be a surprise to learn that many fruits have a low glycemic index. People digest starchy vegetables, such as potatoes and grains, more rapidly, so these have a higher GI index. The longer a carbohydrate-rich food is cooked, the higher the GI value. Fat, fiber content, and cooling carbohydrates after they have been transformed into resistant starches via cooking can all dramatically lower GI values.

People with diabetes should eat a balanced diet that provides enough energy and helps to maintain a healthy weight. Some fruits are high in sugar, such as mangoes, but can be part of a healthy diet in moderate amounts. Fruits can also satisfy a sweet tooth without resorting to candy and other foods with low nutritional value. Most fruits are high in nutrients and low in fat and sodium. Fruits also often contain nutrients not found in other foods. Bananas contain potassium and tryptophan, an important amino acid. Citrus fruits like oranges and grapefruits are high in vitamins A and C, which are powerful antioxidants. Table 1 gives the composition of some common fruits.

Table 1: The Glycemic Index and Load of some common fruits with its sugar content

	Glycemic Index	Glycemic Load	Fruit	Fructose	Sucrose
Apple	38	6	1.03	8.36	0.14
Oranges	44	4	1.21	2.86	2.79
Grapes	46	11	2.15	7.35	0.1
Banana	55	11	6.12	5.43	3.1
Mango	56	8	0.73	2.38	4.66
Pineapple	66	6	3.12	1.21	4.23
Watermelon	72	4	0.3	1.91	1.44
Guava	78	4	3.6	4.01	0.18

From Indian Food Composition Tables, Longvah et al NIN

A recent study published in PLOS medicine tracked the health of 512,891 Chinese men and women between the ages of 30 and 79 for an average of 7 years, in order to understand the effect that their diet had on their overall health. For those who did not have diabetes at the beginning of the study, those who had a higher fruit consumption were 12% less likely to develop diabetes, compared with those who ate zero pieces of fruit per day. The researchers found a dose-response relationship, which means that the more frequently these nondiabetic individuals ate fruit, the lower the risk for developing diabetes. Amongst those living with diabetes at the beginning of the study, those who ate fruit 3 times per week reduced their risk of all-cause mortality (death from any cause) by 17%, compared with diabetic individuals who ate zero pieces of fruit per day. In addition, researchers uncovered that those who ate fresh fruit 3 days per week were 13-28% less likely to experience macrovascular complications (heart disease and stroke) and microvascular damage (kidney disease, retinopathy and neuropathy). Even though this study was observational, the results of the study have profound implications for people living with diabetes around the world.

The lead researcher, Dr. Huaidong Du, believes that the reason why eating more fruit reduces the risk for diabetes and its complications is actually quite simple: "The sugar in fruit is not the same as the sugar in manufactured foods and may be metabolized differently. And there are other nutrients in fruit that may benefit in other ways." In a study performed in 1971, researchers classified the effects of a "mainly fruit diet" to determine what happened to 17 people who ate 20 servings of fruit per day. On this regimen, people ate up to 200 grams of fructose per day, the equivalent of 8 cans of soda. Researchers found that after 3 to 6 months on a mainly fruit diet, there were no adverse effects, and that body weight, blood pressure and insulin levels began trending down.

Following up on this research, in 2001 researchers tested the effect of 20 fruits per day on blood lipids and colon function, and found that within the first 2 weeks, total cholesterol dropped significantly (-40.6 mg/dL), as did LDL cholesterol (-37.9 mg/dL) (10). Although in this case it was a short-term study, they also reported zero adverse side effects of a diet packed with fruit.

Take Home Messages

- Carbohydrates are NOT the enemy for people with diabetes
- The more fruit you eat, the lower your risk for diabetes and its complications
- According to the evidence-based research, you cannot eat too much fruit

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T A P I J

The Journal of the Association of Physicians of India (Tamil Nadu State Chapter)

Honorary Editor:

Dr. Vijay Viswanathan, MD, PhD, FICP, FRCP (London), FRCP (Glasgow)

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TAPIJ invites all the members of the Association of Physicians of India of the Tamil Nadu State Chapter and other academicians involved in scientific and clinical research to contribute their research in the form of original articles/review papers/case reports to this journal. TAPIJ is a quarterly journal and seeks original, insightful and thought-provoking articles and reviews on all aspects of clinical and academic research.

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1. EACH TABLE SHOULD BE ON A SEPARATE SHEET OF PAPER.

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3. With each diskette, a printout of manuscript must be sent in the event of CD damage/virus.

Typed manuscript on white bond paper, with margins of at least 2.5 cm. Number pages consecutively, beginning with the title page. The manuscript should be typed in double space and should include consecutively title page, abstract and key words, text, acknowledgements, references, tables and legends.

In the title page, the full names of all authors with their latest qualification, the name of the laboratory or the department/institution and its address should be mentioned clearly. Also indicate address for correspondence and reprints.

A running title not exceeding 45 spaces should be provided.

Abstract: It should be concise and should cover all the important aspects of the paper. The abstract format will be those used by Index Medicus/ Medicine headings of Index Medicus, should be 150-250 words for all articles, except case reports where it should be around 50 words only.

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Introduction: This should comprise of; (1) purpose of the study/article (2) brief references to pertinent literature only. The introduction should not be an extensive review of the subject.

Patients and methods: This should include the following: (1) Selection of observational or experi mental subjects and the controls, (2) Analytical/ therapeutic/surgical methods used. If these are in common use, identify them only by references. If not common, give a brief description, (3) Statistical methods used.

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Articles from a Book: Yokoyana K, Suyama T, Naito R Development of Fluosol D. A., And its perspective as a blood substitute. In: Oxygen and life, proceeding of the second Pristley conference. Royal Society of Chemistry, London, 1908; 142-52. (Note punctuations)

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ASSOCIATION OF PHYSICIANS OF INDIA
TAMIL NADU STATE CHAPTER

To
The Secretary
Association of Physicians of India – Tamil Nadu State Chapter
Chennai.

Dear Sir,

Kindly enroll me as a Member of API – Tamil Nadu State Chapter. My details are as follows

Name (Surname)

First Name Middle Name

Father / Husband's Name

Qualifications:

University:

Year of Passing

Tamil Nadu Medical Council Registration No:

API (Central) Life Membership No.

Address:

Please Stick one Stamp Size Photo here

Additional Stamp Size Photo to be attached to Application

City	Pincode	District
Telephone: Office	Clinic	Residence
E-mail		Mobile

I hereby declare the above particulars given by me are correct and agree to abide by the Rules and Regulations of the Association.

Signature
Date

Membership Fee	:	Rs.1000 (Rupees One Thousand only).
Details of Payment	:	Demand Draft to be drawn in favour of "**ASSOCIATION OF PHYSICIANS OF INDIA TAMIL NADU STATE CHAPTER**" payable at Chennai.
For Office Use	:	Application received on. Membership No.
Please Note	:	Members are requested to enclose the xerox copy of the Tamil Nadu Medical Council Registration Certificate and Post Graduation Certificate by a recognized university.
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Byelaw 2.3.3 which states that 'Persons who have completed MD can be enrolled as Associate Member, if they are not already member of the Central API. The period is for 5 years and within that stipulated time, He / She should get enrolled as Life Member of API Central Body. He / She fails to become a member of Association of Physicians of India Tamil Nadu State Chapter in case He / She fails to become a member of the API Central within 5 years.

The Association of Physicians of India, Turf Estate, No.6 & 7, Off: Dr.E.Moses Road, Opp. Shakti Mills Compound, Near Mahalaxmi Station (west), Mumbai - 400 011. Tel: 022-66663224 / 24912218, Fax:022-2492 0263, Email:api_ho@vsnl.com

For Office use only
R/No.
Date
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API Membership Application Form

PHOTOGRAPH

To,
The General Secretary
The Association of Physicians of India
Turf Estate # 6 & 7, Off Dr. E. Moses Road, Opp. Shakti Mills Compound, Nr. Mahalaxmi Station (West), Mumbai 400011
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We hereby propose the admission

(BLOCK LETTERS)

Name (Surname)

First name Middle Name

Qualifications :
(Mention the branch of Medicine in which Postgraduate qualification is obtained)

University :

Year of obtaining first Postgraduate qualification :

Address :

City District

State PIN

Tel. (Office) : Tel. (Resi.) : Fax :

email : Mobile :

as a ☐ LIFE ☐ LIFE ASSOCIATE member of the Association
(Please ✓ appropriate)

MEMBERSHIP FEES : Life Member / Life Associate Member : ₹ 7,500 plus admission fees ₹ 1,000.
Total ₹ **8,500.**

Details of payment : In favour of "Association of Physicians of India" (Cheque* / DD / Cash). Applicant's from outside Mumbai are requested to send Cheque / Demand Draft payable at Mumbai. (*For **outstation cheques** add ₹ **100**)

- I hereby direct The Association of Physicians of India to transfer ₹ 7,500 to the Corpus Fund and the balance of ₹ 1,000 for admission fees.
- I hereby state that the above information given is true and correct.

Note for proposer / seconder : To the best of our knowledge and belief the above particulars are correct, and we consider him/her a fit proper person to be admitted as a member of the Association.

Signature of Proposer

Signature of Seconder

Name

Membership No.

Name

Membership No.

Subject to the approval of the Governing Body in an ordinary or a special meeting, I agree to become a member and if admitted, to abide by the Rules and Regulations of the Association.

Signature of Candidate

Note by Secretary

Xerox copies of registration with Medical Council and Postgraduation Certificate by a recognised university should accompany the application form

N.B. Kindly read carefully the rules and regulations printed overleaf before filling this form.

Rules & Regulations of the Association Regarding Admission of Life Members / Associate Members

LIFE MEMBERS : Life Members are required to posses a post-graduate degree such as MD/DNB, DM, or equivalent in internal medicine from any institution or university recognised by the Medical Council of India and/or approved by the Governing Body of the Association. MD General Medicine / Internal medicine includes specialities such as Cardiology, Gastroenterology, Diabetology, Nephrology, Neurology, Clinical Haematology, Chest & Tuberculosis, Endocrinology, Gerontology, Infectious Diseases, Allergy, Immunology, Rheumatology, Medical Oncology and others approved by the Governing Body notified by the General Body. Life membership shall be open to citizens of India only.

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Indian College of Physicians

Eligibility Criteria for the Award of Fellowship of Indian College of Physicians

5.2.1.1 Minimum experience of 10 years after Post Graduation.

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

5.2.1.3 Should have made a significant contribution to research / teaching / development in the field of medicine.

1.1.1.4 Should have contributed to API by way of scientific or Organizational works.

To make the selection objective, a point system has been followed in assessing the suitability of the applications.

The Criteria used by the Credentials Committee for the award of fellowship are:

1. Qualification
2. Experience in Medical Profession
3. Publications
4. Honours / Awards
5. Research work
6. Contribution to API
7. CME & Conference (API/ICP)
8. Social welfare/ community service

The Fellowship form should be proposed and seconded by Founder Fellow / Fellow of ICP only.

- The Proposer / Seconder should not propose / second more than 3 nominees for award of ICP in a particular year.
- It is responsibility of the Nominee / applicant to get the proposal completed by the proposer and seconder along with the citation.
- API Membership No. of the proposer / seconder should be entered by the proposer / seconder themselves.
- The proposer should satisfy the requirements for proposal as under:-
  - ❖ The Nominee is a life member of API
  - ❖ The Nominee has completed 10 years after post-graduation
- The Nominee should read the Form carefully before filling the columns, to project their achievements appropriately.
- The Nominee should list their achievements in appropriate columns.
- Proof of qualifications, publications, honours, awards, must be submitted as supporting data. The supporting data should be numbered parawise (eg 1., 2., 3. , etc), For more than one supporting documents, the numbering should be in alphabets (eg 1 (a), (b), (c), etc).
- No hand written applications will be accepted.
- One original and seven Xerox copies to be submitted
- Last date for receiving application form is **31st May** of the year.

Dr. Milind Y. Nadkar
Hon. General Secretary

Dr. B. R. Bansode
Jt. Secretary

Format for Submission of Bio - Data of The Nominee for Consideration for Award of Fellowship of Indian College of Physicians.

1.	Name in Full (Surname First) (in Block Letters)			
2.	A. P. I. Membership No. and date of joining			
3.	Date of Birth			
	Address Residence		Address Office	
4.	Tel.:	Fax : Mobile	E-mail:	
5.	Postgraduate degree in Medicine	Year of passing	Institute	University
	Other Professional Qualifications	Year	Speciality / Subjects	University / Institute
a.				
b.				
c.				
d.				
	Certificates Attached			
6.	Experience in Medical Profession after Postgraduation in Medicine			

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
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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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