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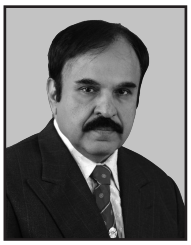

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Editor's Note

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 third issue of TAPIJ 2018, which has several informative articles, that covers various aspects of Medical sciences including unusual case reports.

This issue consists of an original article entitled 'The effect of counselling on the knowledge, practices and eating habits of individuals in community with diabetes' and three interesting case reports on 'Surgical cyclone and post-operative storm in a case of ulcerative colitis', 'Massive tuberculous pericardial effusion', and 'An unusual case of easy fatiguability'. This issue also contains valuable articles in the areas of ECG, Dermatology and Toxicology which enrich our knowledge and helps remain updated in the field.

In this edition, we have included an article on 'Improving nutrition in women with Diabetes Mellitus' that enhance our understanding on the various risk factors and nutritional requirements of women with Type 2 Diabetes Mellitus. Besides these, we have two review articles on 'Offloading techniques for diabetic foot ulcers' and 'Zika Virus'.

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)

Contents

Original Article

TAPI Journal Vol. 12, Issue 3, September – December 2018

The Effect of Counselling on the Knowledge, Practices and Eating Habits of Individuals in Community with Diabetes

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Abstract

Background: Diabetes education, with improvement in knowledge, practice and eating habit skills, leads to better control of the condition. The objectives of the study were to assess knowledge, practice and eating habits of diabetic patients in the community. Literature evidence shows that appropriate health education program could improve the knowledge of diabetic patients and change their attitude. Therefore, this study aims to document the baseline knowledge about diabetes; followed by an intervention and change in knowledge level among Type-2 diabetes patients is evaluated in the community.

Methods: This is a prospective study with a phase of intervention among the diabetic patients in Panamarathotti, a fisherman's colony in Chennai. A pre-tested pilot validated questionnaire in vernacular language has been administered to fifty Type-2 diabetic patients. Baseline and end line data were collected.

Result: Baseline knowledge about diabetes causes, symptoms and complications found to be poor in this study. Change in knowledge and improved practices were revealed in endline survey because of an appropriate intervention program.

Conclusion: This study found that the knowledge and practices about management of diabetes could be changed with suitably designed community based intervention programs.

Keywords: Knowledge, Practice, eating habits, Intensive counselling.

Introduction

Diabetes is now emerging as an epidemic of the 21st Century. It threatens to overwhelm the health care system in the near future[1]. Diabetes mellitus is one of the most common chronic diseases in nearly all countries, and continues to increase in numbers and significance, as changing lifestyles lead to reduced physical activity and increased obesity. The management of diabetes mellitus (DM) largely depends on patient's ability to self-care in their daily lives and therefore, patient education is always considered an essential element of DM management[2] .

There exists a large gap between the knowledge, attitude and practice towards diabetes among diabetic patients[3,4]. Knowledge about diabetes mellitus appropriate attitude and practices are vital to reduce the prevalence and morbidity associated with DM. However, very few studies focused on this area and there is also lack of data on knowledge and practice among Indian diabetic patients.[5]. Recent surveys indicate the diabetes now affects a staggering 10-16% of urban population and 5- 8% of rural population in India[6,7]. There is very little data on the level of awareness about diabetes in developing countries like India.

The initial aim was to assess baseline levels of knowledge and attitudes in a representative sample of an urban, fisherman's population in Panamarathotty with Type 2 diabetes. Then we used the baseline results to help develop an appropriate (competent) educational intervention programme for these groups. We then investigated whether this intervention could produce an improvement, and finally whether any

improvement was greater than background changes in knowledge or not.

Methods

Study type

This is a prospective study with a phase of intervention among the diabetic patients in general community. This study has three phases i.e. baseline to gather knowledge about DM among diabetes patients, followed by an appropriate intervention and finally end line to document the changes in knowledge.

Study setting

This study has been conducted in Panamarathotty a fisherman's colony in Royapuram in Chennai.

Study sample

The sample subjects were selected by systematic random sampling technique from known cases of diabetes in study areas. The samples were taken from both male and female respondents. A total of 50 samples were selected

Study data collection method

Written consent was obtained from the respondents after explaining the purpose of the study. Structured questionnaire tool was used for data collection. Tools were targeted for obtaining household information, measuring knowledge, practice and eating habits among diabetic population.

An initial baseline information was collected in regard to their knowledge, practice and eating habits with the structured questionnaire. Intervention phase of education and then the end line data was collected

Study data variables

The questionnaire was divided into knowledge, practice and eating habits. Data collection were done by pre-constructed and pre-tested questioner that was covered as follows:

- Personal and socio-economic data (Name, sex, education level, and occupation BMI and addictions.
- Closed ended questions were asked about knowledge and practices of diabetes management and care. General awareness and knowledge about diabetes such as its causes, symptoms, complication and diet were included in the questionnaire.
- Practices towards exercise, diet, medication and health care accessibility for diabetes care.
- -Eating habits eating behaviour, use of refined cereals were included.

Scoring Procedure

The analysis of three components was done on the basis of scoring method. Overall, there were 30 questions in the questionnaire. Totally, 87 scoring points were awarded if a respondent answered all questions correctly. The total 87 scoring points were divided into three sections in which 28 points awarded for the first two sections and 31 points attributed for eating habits section. For each question, the correct answers obtained higher scoring points while wrong answer or the respondents who don't know anything receives a zero scoring points. The moderate scoring points were provided for the better answer which is relevant to the question. No scoring points were provided for the respondents who did not know. The total knowledge, practice and eating habits score were analysed. This analysis was conducted to rank high, medium and low scores

Intervention phase

Community level interventions were done such as, lecture demonstrations regarding diabetes prevention & early management, and health camp. Posters were used to educate dietary concepts about diabetes and improve knowledge. Physical activity and yoga were demonstrated to the respondents and the respondents were encouraged to do the exercises during the demonstration. A healthy recipe was demonstrated using millets to encourage the use of complex carbohydrates. Intervention counselling was carried out at two monthly intervals for a period of six months.

Data analysis

Data entered were analyzed by IBM SPSS 15 software. Descriptive statistics were done for continuous variables in the form of mean and standard deviation. The continuous variables including knowledge, practice and eating habit scores were analysed using ANOVA with Tukey procedure.

Results and Discussion

Socio – demographic profile:

All 100% belonged to the low income group. It has been observed that about one-fourth of study sample have the habit of tobacco consumption. It was also observed that 68.2% of the study population had a BMI above 26 and 100% of the population were not exercising regularly.

Table1: Distribution of age group for low income

Age Group	% Respondents
30 – 40	6 %
41 – 50	22 %
51 – 60	28 %
61– 70	44 %

44% of the respondents were above 61 years, 28% between 51 to 60 years and 22% between 41 to 50 years of age. Only 6% were between 30 to 40 yea

Table 2: Educational status

Educational Status	% Respondents
Illiterate	28 %
Schooling	60 %
Graduates	12 %

Most of the respondents could read and write as they had completed their school. Only 28% were illiterate. The base line data on their knowledge, practice and eating habits before counselling was scored and the results tabulated shown in Table 1

Table 3: Baseline of mean scores of knowledge, practice and eating habits between male and female respondents

Variable	Group	%	Mean Scores ± S. D	Std Err
Knowledge	Before counselling (Male)	36%	18.5 ± 3.1	0.738
	Before counselling (Female)	64%	18.0 ± 3.4	0.609
Practice	Before counselling (Male)	36%	16.2 ± 2.6	0.620
	Before counselling (Female)	64%	16.5 ± 1.9	0.351
Eating Habits	Before counselling (Male)	36%	18.7 ± 2.7	0.639
	Before counselling (Female)	64%	18.3 ± 2.5	0.448

Al-Adsani *et al.,* 2009 stated that the awareness of type 2 diabetes-related health-knowledge was very poor at baseline, especially for patients in rural areas and with lower education level which was in accordance with previous studies conducted in both developed and developing countries[8]. This is of particular concern given consistent findings across population based studies of diabetes in rural populations that patients belonging to higher socio-economic strata with higher family income had a greater knowledge of diabetes[9] yet also higher prevalence of diabetes compared to those with lower income[10]. Khurshid & Othman (2014) also found that respondents who had good level of education had satisfactory knowledge on diabetes mellitus[11].

In this current study, the knowledge was less because they had a low level of education and also less awareness. Malathy *et al.,* 2011

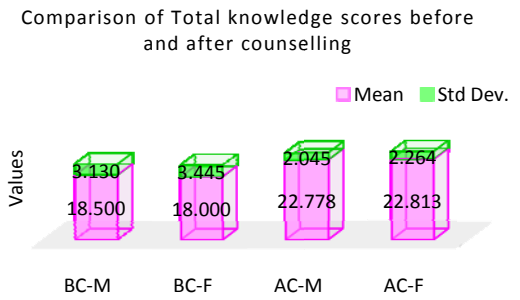
mentioned that the practice scores did not show any improvement ($p < 0.06$); the baseline practice scores had been relatively high[12]. No significant changes in KAP scores for the control group were observed. In 2014, Fakir M. Amirul Islam *et al* conducted a KAP survey among general community people, their practice in relation to checking blood sugar levels, there was no significant difference across the sociodemographic parameters, with the exception of low economic status, where those with insufficient funds most or all of the time checked their blood glucose less frequently compared to those with sufficient funds most or all of the time[13]. The low-income respondents do not check their blood glucose level regularly and also health related issues because of their low income. Another reason might be due to lack of knowledge, their practice level was poor before counselling.

Phyu Phyu Aung *et al.,* 2012 stated that no significant association between variables in the study and health eating practice indicates that for young adults and adolescents, eating behaviour mostly follows their desire, taste and preferences, rather than choosing food for health purpose[14]. The Da Qing, (1999) Study was undertaken over a longer intervention period (6 years). The cumulative incidence of diabetes after 6 years was 67.7% in the control group, 43.8% in the diet group, 41.1% in the exercise group and 46% in the diet plus exercise group. All the groups differed significantly from the control group (P 0.05).[15] The level of knowledge, practice and eating habits they gained from intervention counselling was evaluated and compared with the baseline values.

Knowledge Score

The knowledge, practice and eating habits of the individuals were evaluated again after a six period and the results are given below.

Figure 1: Comparison of mean scores of knowledge before and after counselling



BC-M Before counselling –Male
BC-F Before counselling- Female
AC-M After Counselling-Male
AC-Female After counselling-Female

Table 4: Post Hoc tests comparing Mean values of knowledge between before and after counselling. Tukey Test (Tukey statistics "q")

Variable	Before Counselling	After counselling	Q Value	P Value
Knowledge	Male	Male	6.746	0.000 (Sig)
	Female	Female	10.119	0.000 (Sig)

The comparison of before and after counselling male, it shows a significant difference ("p" < 0.01). The analysis variance of q value is 6.746. The comparison of mean value between before and after counselling females, their q value is 10.119 and it shows a significance ("p" < 0.01).

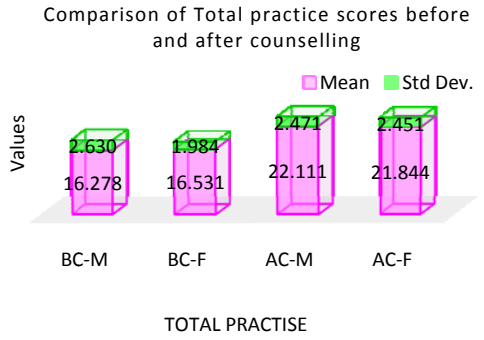
The authors of the study concluded that knowledge deficit was due to low educational levels, low family income and low self-care. We also found that educational status has a significant impact on the knowledge score of our study participants ($p = 0.01$), however no significant difference ($p = 0.44$) was observed in the knowledge of male and female participants which is in contrast to previous studies conducted elsewhere in the world where they found that men were significantly less knowledgeable about diabetes than women[16,17]. This statement suggests that educational intervention is able to improve knowledge level, such as dietary counselling. A study reported that 54%, 34%, and 13% had poor, fair and good knowledge respectively about DM[18].

It was found that there was a significant impact on the knowledge of the respondents after education as seen by the score given in Fig.1.

Practice score:

Figure 2 represents that the comparison of practice score for before and after counselling male and female among low income group. Their practice means score shows that better change when compared to before counselling male and female. The practice level has highly increased through intervention counselling. The practice mean score of before counselling male and female are 16.2 ± 2.6 and 16.5 ± 1.9 but after counselling male and female mean scores are increased i.e. 22.1 ± 2.4 and 21.8 ± 2.4.

Figure 2: Comparison of mean practice scores for before and after counselling



BC-M Before counselling –Male

BC-F Before counselling- Female

AC-M After Counselling-Male

AC-Female After counselling-Female

Table 5: Post Hoc tests comparing Mean values of practice between before and after counselling.

Tukey Test (Tukey statistics "q")

Variable	Before Counselling	After counselling	Q Value	P Value
Practice	Male	Male	10.788	0.000 (Sig)
	Female	Female	13.100	0.000 (Sig)

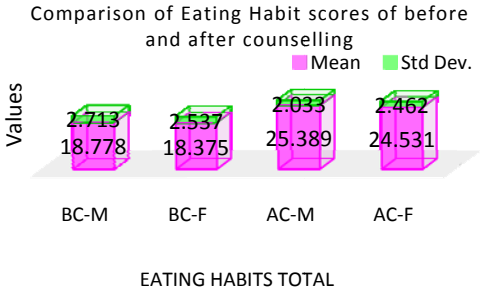
The practice mean values are compared between before and after counselling male shows that the significant difference i.e. ("p" < 0.01). The tukey statistics q value is 10.788. Among female, there was a significance difference and the statistical value of q is 13.100.

Improving knowledge of the people can improve their attitude towards diabetes and in the long run change their practices to embrace healthier lifestyles such as eating healthy foods, and engaging in physical activity[19] Such practices will minimize the risks for diabetes in the general public and delay the onset of complications in those already diabetic. The likelihood of good practice among female was more as compared to male. This finding remains contradict with the previous studies from Africa, Asia and middle east[20,21]. The increased duration of diabetes was associated with good practice in our study which was similar to Nigerian findings[19]. A multi-site, prospective study of randomly selected diabetic Asians living in Scotland, by Baradaran et al. (2006) evaluated an educational intervention tailored for South Asians, using a group comparison of changes in diabetes knowledge between the test group and the ethnic and the white control group. The test group had low baseline KAP scores. In the intervention group practice scores improved significantly (+20.0 %).[23]

To improving the knowledge can leads to better practice in diabetes. In this study, the practice among low income respondents were improved significantly through intervention counselling.

Eating habits:

Figure 3: Comparison of mean scores of eating habits for before and after counselling



BC-M Before counselling –Male

BC-F Before counselling- Female

AC-M After Counselling-Male

AC-Female After counselling-Female

The above figure represents that the comparison of eating habits before and after counselling male and female. The eating habits of before counselling male and female mean score was 18.7 ± 2.7 and 18.3 ± 2.5. The eating habits mean score was improved after counselling in both gender (For male - 25.3 ± 2.0 and for female - 24.5 ± 2.4).

Table 6: Post Hoc tests comparing Mean values of eating habits before and after counsellingTukey Test (Tukey statistics "q")

Variable	Before Counselling	After counselling	Q Value	P Value
Eating habits	Male	Male	12.138	0.000 (Sig)
	Female	Female	15.070	0.000 (Sig)

A comparison of eating habits before and after counselling, it was found that there was a significant difference in both gender. The statistical analysis of tukey q value for before and after counselling male is 12.138 and for female, it is 15.070.

Sharma et al. (2008) also reported that nutritional knowledge is significantly related to dietary habits (including consumption of meat, dairy, grains and water). The findings of these studies show that educational interference leads to an increase in nutrition knowledge and the enhancement of people's attitudes.[23] Among diabetic respondents, the eating habits is the most important thing and it helps to control their blood sugar level. Here, it shows the significance results after counselling.

In the present study, we have sought to determine the awareness level of urban, rural and tribal diabetic patients about the disease. The strategy of this study was to understand either the intervention was effective for improving the basic knowledge of diabetes mellitus, its causes, symptoms, complications, management, treatment and practices to their own diabetic management. The previous studies from various settings throughout India indicate that the knowledge level varies from region to region [5,24-26] .But the specialty of this study is to document the change in knowledge level among diabetic cases with Behavior Change Communication (BCC) intervention. Even the changes in practices have been documented with the change of knowledge.

Conclusions

This study found that the knowledge and practices about management of diabetes could be changed with suitable designed community based intervention programs. The background knowledge such as causes, symptoms, complications about type-2 diabetes found to be significantly changed during endline evaluation from baseline.

Conflict of interest

The authors have declared that no competing interests exist.

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Review Article TAPI Journal Vol. 12, Issue 3, September – December 2018

Offloading techniques for diabetic foot ulcers

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Introduction

Diabetes and its associated complications, including foot diseases, are increasing at an alarming pace in India and putting enormous burden on our limited health care resources. According to an estimate by International Diabetes Federation (IDF), 80% of people with diabetes live in low- to middle-income countries including India, a country with the second largest number of diabetic patients in the world after China[1]. An increasing number of patients with diabetes translate into an increased burden of diabetic complications including microvascular and lower extremity complications. Lower extremity diseases, including peripheral neuropathy, peripheral arterial disease (PAD), and foot ulceration, is twice as common in diabetic subjects as compared with nondiabetic persons and affects 30% of diabetic people older than 40 years[2].

Diabetic foot ulcer and DFI have long-term implications for persons living with diabetes in the form of morbidity and mortality. The annual incidence of diabetic foot ulcer (DFU) in population-based studies is 1.0 to 4.1% and prevalence of 4.5 to 10%, with an overall lifetime incidence of up to 25%[3, 4]. Foot wounds not only add to morbidity but also to health-care cost, and are attributed as the most frequent cause for diabetes-associated hospitalization. Early recognition, classification, diagnosis, and treatment of foot complications are needed to optimize outcomes in patients with diabetes.

Pathophysiology and risk factors

Ulcerations associated with diabetes are the most common cause of foot infections. This could be attributed to several socio-cultural practices, such as walking barefoot, inadequate facilities for diabetes care, poor education, and poor socioeconomic conditions[5]. It was reported earlier[6] that recurrence of foot infection was common among South Indian type 2 diabetic subjects and was related to the presence of peripheral vascular disease and neuropathy. Triad of peripheral neuropathy, foot deformity and minor trauma have been found as risk factors in majority of cases of diabetic foot ulcers[7].

Limited joint mobility and increased plantar pressure were appear to be important causative factors in the development of plantar foot ulcers in people with diabetes mellitus, irrespective of the duration of diabetes[8,9]. The pathogenesis of neuropathic diabetic foot ulcers involves mechanical trauma due to focal pressure and repetitive moderate stress[10]. Alterations in foot dynamics due to ulceration, joint deformity, or amputation can cause abnormal distribution of plantar pressures and result in the formation of a new ulcer[11]. This pressure causes maximum damage at the edge of the area of pressure application and has been termed as "edge effect"[12]. The combination of foot deformity, loss of protective sensation, and inadequate off-loading leads to tissue damage and ulceration. Once an ulcer has formed, unless the ulcerated area is off-loaded, healing may be chronically delayed, even in an adequately perfused limb[13].

Management of diabetic foot ulcers

Appropriate patient education to encourage them to regular foot care in order to prevent DFU and its complications is crucial in the management of DFU. Early effective management of diabetic foot ulcers can reduce the severity of complications such as preventable amputations and possible mortality, and also can improve overall quality of life. Optimal management entails a multidisciplinary approach for prevention, early evaluation and treatment

strategies. Blood sugar control, appropriate wound care (and debridement), infection control, advanced dressings and pressure reduction using offloading modalities should always be a part of DFU management. Furthermore, surgery to heal chronic ulcer and prevent recurrence should be considered as an essential component of management in some cases. Also, hyperbaric oxygen therapy, electrical stimulation, negative pressure wound therapy, bio-engineered skin and growth factors could be used as adjunct therapies for rapid healing of DFU.

Offloading techniques

In the treatment of diabetic foot ulcers, pressure modulation, commonly referred to as "offloading," is most successful when pressure is mitigated at an area of high vertical or shear stress[14]. It is the most ignored aspect of diabetic foot care, especially in developing world. The main aim of off-loading, or pressure modulation, is to redistribute plantar pressures so that there will be a resultant reduction foot pressures, shock and shear[15]. Even when the ulcer develops, proper evaluation and regulation of foot pressure may aid in faster healing[16].

A variety of offloading techniques are in use including surgical offloading. An ideal off-loading device must be patient compliant, easy to apply, cost-effective, effective in wound healing, comfortable for ambulation, accommodated in diabetic footwear with ease, and applicable at all levels of health care systems, including rural settings. Common methods to offload the foot include bed rest, wheel chair, crutch assisted gait, total contact casts, felted foam dressings, half shoes, therapeutic shoes, padded socks, shoe inserts, and removable cast walkers[17]. Bed rest and wheel chairs are considered to be an excellent option for offloading wounds that are ischemic in the developing countries[18].

Surgical offloading

Minimally invasive surgical offloading that includes correction of foot deformities has good short and long term results. The surgery alleviates the pressure under the bony prominence, thus enabling prompt ulcer healing, negating the patient's dependence on expensive shoes and orthotics, with a lower chance of recurrence[19]. Surgical offloading is usually reserved for chronic deformities or ulcers that are not amenable to conservative treatment especially for Charcot foot with rocker bottom feet or difficult to treat mid foot ulcers[16].

Total contact casts (TCC)

TCCs, which have been considered as the gold standard by academicians and consensus committees, is a method where a non-removable cast is fitted around and is in contact with the foot and part of the leg[20]. The main aim of TCC is to increase the weight-bearing surface area so the pressure is distributed more evenly in the sole of the foot[21]. TCC reduces plantar pressure by increasing weight bearing surface. The reduction of pressure peaks by providing special shoes turns out to be another effective tool for managing the neuropathic foot. TCC has several advantages such as forced compliance to offloading, protecting foot from infection and reduction of edema.

Disadvantages with TCC include unaffordable cost since it has to be changed every week or so and even more frequently if associated with oedema, needing specially trained technicians to apply and remove it; thus TCC application as well as removal is quite time consuming. TCC poses difficulty in sleeping, bathing and driving as also affects gait stability[22]. With TCC monitoring of the wound cannot be done and hence it is often not recommended for neuro ischemic diabetic foot.

Offloading foot wears

The higher recurrence rate of ulceration in the non therapeutic footwear group underscores the need to use specially designed footwear and include it as part of the overall diabetes care regimen for these patients. The incidence of recurrence of plantar foot ulcer in high-risk patients can be significantly reduced with

custom-made footwear that has a demonstrated pressure-relieving effect through guidance by plantar pressure measurements, under the condition that the footwear is worn. Heel Wedge heeling shoes are recommended in the treatment of plantar fasciitis, ulcerations, infections and trauma. They are used following the surgery of soft tissue or bony structure of the heel. Ortho Wedge healing shoes were used to protect forefoot by removing pressure from the metatarsal heads and digits. They often recommended for diabetic ulcerations, Hallux valgus, Tailor's bunions and osteotomies. Anterior Orthowedge and posterior orthowedge devices can be used in neuro-ischaemic wounds[23].

Removable cast walkers (RCW)

Removable cast walkers are cast-like devices that are removable to allow for self inspection of the wound and application of topical therapies that require frequent administration.

Charcot Restraint Orthotic Walker (CROW)/ Neuropathic walker is a rigid full foot enclosure ankle-foot orthosis where any residual deformity in the device can be accommodated with custom insoles and orthotics[24, 25]. The main aim of CROW is to uniformly distribute the pressure over the foot as a measure to protect the joints and skin, preventing deformity[26]. It is used as means to protect the foot after removal of a cast used for treatment of a foot ulcer[27]. The mechanism of action of this device is similar with the TCC, i.e. redistribution of pressure is more evenly in the sole of the foot by increasing the surface area which comes in contact with the ground when walking, thus relatively redirecting pressure away from the ulcer site[28]. The device helps in immobilisation of the affected limb and protects it to allow sufficient time for ulcer to heal[29]. The advantages of CROW include Patients being able to: bear full weight and ambulate[30], remove the equipment for inspection of the ulcers, and apply dressings or topical medication. In addition, CROW also effectively controls oedema of the limb[31]. Furthermore, its usage has good patient satisfaction. However, patients with CROW have the ability to remove their device and ambulate without protection, thus leading to an increased risk of trauma and development of ulcers. The device is also heavy and is not suitable for use in a frail individual. In addition, there is a likelihood of irritation to the skin and breakdown, requiring a support stocking to be used with this device[32].

Prefabricated walking braces are the devices that are relatively easy to use, cost effective and are removable, allowing access to the wound for certain procedures such as dressing, application of topical medication and bathing, which may improve patient compliance[33]. They have been shown to reduce forefoot pressure as effective as or more effective than TCCs[34]. They act in the same principle as TCCs, where redistribution of pressure over the sole is the main objective. However, as with the CROW, there is a potential for patient's non compliance. Aircast pneumatic walker is a type of prefabricated walking brace. It is a removable semi-rigid plastic off-loading device with internal air cells, a rocker sole for improved ambulation and off-loading and a dual-density insole[35]. It also reduces oedema and shearing forces by virtue of its air cells[36]. It reduces the peak plantar pressures to an equal or greater degree compared with TCCs in the forefoot, midfoot and hindfoot as well[37]. This device can be considered as a valid alternative to TCCs in view of its good healing rate and low rate of developing complications[38].

Conclusion

Noninfected, nonischemic neuropathic plantar forefoot ulcers should heal in 6 to 8 weeks with adequate offloading. Non-removable knee-high devices are found to be most effective in the management of nonischemic foot ulcers. This is probably because they eliminate the problem of non-adherence with the use of a removable device. Removable casts, pre –fabricated devices for offloading may be used in neuro-ischemic wounds so that daily inspection of the wound is possible during dressing.

Studies show a large discrepancy between evidence-based recommendations on offloading and what is used in clinical practice. Many clinics continue to use methods that are less effective or have not been proven to be effective, while ignoring evidence-based methods. Strategies are proposed to address this issue, notably the adoption and implementation of recent international guidelines by professional societies and a stronger focus of clinicians on expedited healing.

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Zika Virus (ZIKV)

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Zika virus belongs to family *Flaviviridae*, it was first isolated in 1947 from the Ziika Forest of Uganda. It mainly spread by daytime active *Aedes* mosquitoes, such as *A. aegypti* and *A. albopictus*. It is close to the dengue, yellow fever, Japanese encephalitis, and West Nile viruses and causes Zika fever or Zika virus disease, similar to a very mild form of dengue fever. Why we are bothered about Zika, because this is the 3rd time outbreak reported in India, in last 2 years. Recently few weeks back, the virus has been detected in 22 people in Jaipur, capital of the western state of Rajasthan. In Bihar a case diagnosed as zika (native of Jaipur).

Transmission

The vertebrate hosts of the virus were primarily monkeys in a so-called enzootic mosquito-monkey-mosquito cycle, with only occasional transmission to humans.

Mosquito

Zika is primarily spread by the female *Aedes aegypti* mosquito, which is active mostly in the daytime. The virus has also been isolated from a number of arboreal mosquito species in the genus *Aedes*, such as *A. africanus*, *A. apicoargenteus*, *A. furcifer*, *A. hensilli*, *A. luteocephalus*, and *A. vittatus*, with an extrinsic incubation period in mosquitoes around 10 days.

Sexual

Zika can be transmitted from men and women to their sexual partners; most known cases involve transmission from symptomatic men to women. Sexual transmission of Zika has been documented in many countries. The CDC has advised men who have traveled to an area with Zika should use condoms or not have sex for at least six months after their return as the virus is still transmissible even if symptoms never develop.

Pregnancy

Spread can be vertical transmission, during pregnancy or at the time of delivery. Zika infection during pregnancy mainly linked to changes in neuronal development. Severe progressions of infection lead to the development of microcephaly, while mild form may lead to neurocognitive disorders in adulthood.

Blood transfusion

Cases of Zika transmission through blood transfusions have been reported globally, the US Food and Drug Administration (FDA) recommended screening blood donors and deferring high-risk donors for 4 weeks.

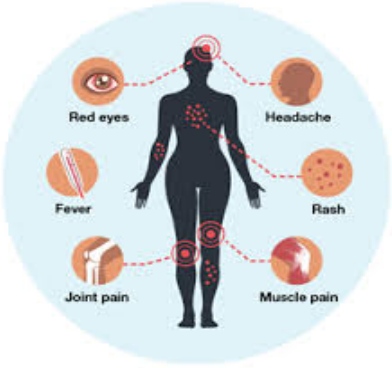
Pathogenesis

Virus replicates in the mosquito's midgut epithelial cells and then its salivary gland cells. 5–10 days later, the virus can be found in the mosquito's saliva. It is inoculated into human skin, the virus can infect epidermal keratinocytes, skin fibroblasts in the skin and the Langerhans cells. The pathogenesis of the virus is hypothesized to continue with a spread to lymph nodes and the bloodstream.

Clinical Features

Most cases have no symptoms, but when present they are usually mild and can resemble dengue fever. Symptoms may include fever, red eyes, joint pain, headache, and a maculopapular rash. Symptoms generally last in one week. No death reported during the initial infection. In pregnancy causes microcephaly and other brain malformations in unborn babies and neurocognitive disorders in adulthood. In adults it may lead to Guillain–Barré syndrome (GBS). Health officials recommended that women in

areas affected by the 2015–16 Zika outbreak consider putting off pregnancy and that pregnant women not travel to these areas.



Treatment

No specific treatment, paracetamol and rest may help in improving with the symptoms. Admission to a hospital is rarely necessary.

Vaccine

There is no vaccine available at present. It has suggested that priority should be to develop inactivated vaccines and other nonlive vaccines, which are safe to use in pregnant women. DNA vaccine was approved for phase-2 clinical trials.

Prevention

Avoid mosquito bites in areas where the disease occurs, use of insect repellent, covering much of the body with clothing, mosquito nets, getting rid of standing water where mosquitoes reproduce. Proper use of condoms up to six months from symptoms free. Pregnant women are strictly advised not to travel to those areas.

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Surgical Cyclone and Post-Operative Storm in A Case of Ulcerative Colitis

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Abstract

Ulcerative colitis is one of the chronic inflammatory disease of the colon and rectum presenting as diarrhea, rectal bleeding with acute exacerbation of symptoms with remissions and relapse with or without extra intestinal manifestations. Although common in Caucasians, it does occur in tropical countries like India. Medical management with anti-inflammatory drugs both steroidal and non-steroidal and immunosuppressant drugs are aimed to attain remission but total colectomy with Ileostomy or Ileo-Anal anastomosis offers a complete cure. In view of the disadvantages of ileostomy and frequency of defecation, incidentally an innovative colectomy with subtotal proctectomy and ileo-rectal anastomosis was done in this case in an emergency situation when the patient developed toxic megacolon and colonic perforation.

Introduction

In this case report it is presented because of the ulcerative colitis with its cyclonic complication, toxic megacolon and multiple perforations in colon, surgical, post-surgical storm experienced in a rural setup with limited available facility. However modern management methods were followed to tide over the emergency crisis and in the course of post-operative storm. During the post-operative period, the patient developed pneumonic consolidation on the 3rd day, Stitch abscess on 7th day. Post-operative Ileus due to hypokalemia managed in intensive care unit with limited resources. Intravenous alimentation, total parenteral nutrition (TPN) therapy given in pre-operative and post-operative phase. Total Colectomy, subtotal proctectomy with Ileo- rectal anastomosis done in this case resulted in better quality of life postoperatively, like free from frequent stools and almost near normal consistency of stool.

This case is a living testimony to enlighten the fact that anatomical and functional asplenia is prone to develop pneumonic consolidation. Since the rectum was not completely resected, sulphasalazine was continued to take care of the remaining rectal mucosa and this drug has precipitated frank bleeding per rectum and patient went in for hypotensive shock. He was aggressively treated appropriately and managed successfully. Patient was discharged in good condition with restoration of almost normal bowel habits.

Case Presentation

David 44/male admitted on 5th June 2013, operated on 23rd June 2013 and discharged on 25th July 2013, presented with fever, loose stools associated with bleeding per rectum for 6 days.

Past history

Bleeding per rectum and frequent diarrhea and lower abdominal pain on and off for the past 2 years.

Treatment history

He had undergone banding under the misdiagnosis as piles rectum twice in the last 2 years. He is a known hypertensive for which he has been on an anti-hypertensive drug Amlodipine 2.5mg daily for the past 2 years.

Personal history

He is not a smoker, alcoholic or a substance abuser

Clinical Examination

On examination, the patient was not anaemic, febrile temperature of 100 degree F, no jaundice, clubbing or lymphadenopathy and no pedal edema were observed

Examination on abdomen

Soft, non-tender, non distended, bowel sounds heard, no hepatosplenomegaly. Thus, clinical diagnosis of colitis was made and treated with ofloxacin and metronidazole. There was no clinical improvement and symptoms continue to remain the same.

Diagnosis

Clinical diagnosis was revised after inspecting the stool which was stained with mucus and blood. After the revised clinical diagnosis of ulcerative colitis, the treatment protocol was resorted to anti-inflammatory drugs, methylprednisolone 500 mg BD and sulphasalazine were given along with supportive measures and IV fluids. Since patient complained of insomnia, a benzodiazepine was empirically given. Subsequently patient developed progressive abdominal distension whereby it reached a state of toxic megacolon and increased frequency of stool with mucus and blood. Since the patient developed toxic megacolon and to prevent further complications, oral feeds were stopped and was subjected to total parenteral nutrition therapy. Colonoscopy was contemplated and not carried out in view unforeseen event of colonic perforation in the acute phase. Patient was all along conscious, conversible and was advised for surgery but he was not willing for a surgical procedure and requested for a non-surgical conservative management. Unfortunately, patient developed progressive abdominal distension associated with pain which clinically brought upon a suspicion of perforation which was then confirmed with an X-ray.

Investigation:

Laboratory values during the course of management in the hospital is as follows :

Hb: 11.8→9.1→ 10.2→ 9.9→10. 9→8→ 10.4→ 9.8→2.9→6 .8→9→ 8.6→12.6 gm%

TC: 5,600→ 4,900→ 13,800→ 22,500→ 25,100→ 22,000→ 14,800 /cu. Mm

DC: Polymorphs: 52→ 79→ 82→ 85→ 68

Lymphocytes: 40→16→ 10→ 09→ 25

Eosinophil: 08→ 04→ 08→ 06→ 07

ESR : 77→ 96→ 62 mm in 1 hr

Platelet count: 5.06→4.6 lakh /cumm

Urea: 44→ 45→ 48→ 36→ 34→ 57→ 68→ 66→ 24→ 25→ 19 mg%

Creatinine: 2.2→1.9→ 1.4→1.4→1.2→1.3→ 1→ 0.8→0.6→ 0.8→ 0.7 mg%

RBS : 63→ 81→ 93→ 191→165 mg%

Liver function test - WNL

Serum albumin : 2.8→2.6

Urine routine: 4-5 puscells

Stool routine: Plenty of pus cells and RBC's seen

Serum electrolyte: Sodium: 136 → 133→ 132→ 137→ 142→ 139→132

Potassium: 2.9→ 3.5→ 3.6→ 2.7→ 2.1→2.9→ 4.5→ 4.6

Chloride: 98→98→98→ 104→104→ 106→100→ 109

Uric Acid: 2.7

HIV - non reactive **HBsAg -** non reactive **HCV -** non reactive

Peripheral smear: Neutrophilic leucocytosis

MP smear - negative

P ANCA - negative

Scrub typhus - negative

Brucella - negative

Leptospira - negative

USG abdomen - features suggestive of colitis

Post operative CT scan of abdomen (plain and rectal contrast):

- Large 8.3 x 3.9 x 11cm postoperative hematoma in the left hypochondrium
- Intermediate density sludge noted in the GB lumen
- Dilated fluid filled small bowel loops with multiple air fluid levels from the duodenum to ileoanal- anastomosis, represents small bowel obstruction due to paralytic ileus
- Post operative changes in the abdomen in the form of mesenteric fat stranding and minimal localized free fluid in pelvis

- No leakage of contrast noted through the Ileo-anal Anastomosis. No significant pelvic collection noted
- Bilateral pleural effusion noted (left more than right side)

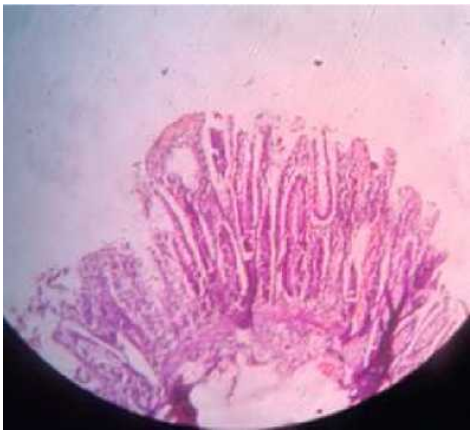
Blood culture and sensitivity: negative

Urine culture and sensitivity: negative

Pus culture and sensitivity: Sterile

ET tube tip culture and sensitivity: Candidial infection

Under this circumstances, there is no other alternative option available to the patient but to accept the surgical management. Emergency laparotomy was done on 23/6/2013 under general anesthesia.

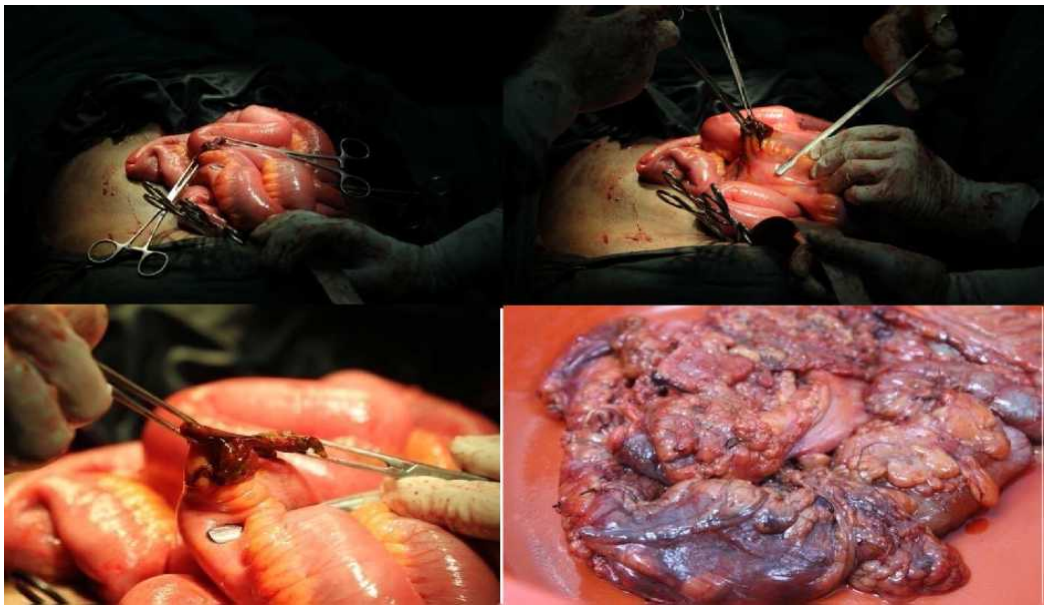


Colostomy Specimen Biopsy Showing:

Diffuse Inflammatory Cells, Crypt Atrophy, Irregular Superficial Erosions. Due to the technical difficulty in performing ileo-anal anastomosis deep in the pelvic cavity and non-availability of a stapler and lack of qualified surgeons to assist and trained theatre nurse, the procedure was modified to do Total Colectomy and Subtotal Proctectomy and Ileo-Rectal anastomosis was carried out.

A spreading Haematoma was noticed during separation of colonic adhesions near the Splenic Hilum towards the end of the surgery, hence splenectomy was done to attain haemostasis. A single layer ileo-rectal anastomosis with 3-0 vicryl was done and peritoneal toileting was carried out with 10 Ltrs ofwarm saline and abdomen was closed in layers with a pelvic drain.

During the surgery, patient developed hypotensive crisis and was managed successfully by the anesthetist by inserting a central venous catheter for rapid transfusion of blood and crystalloids. The BP was maintained to normal with low dose ionotropes. Patient was transferred to ICU and was connected to a ventilator for a day. He was breathing spontaneously with normal



1.The entire colon was distended 2.Multiple perforations were noted in the sigmoid colon 3.Greater omentum was adherent to the colon at the sites of perforation 4.Pelvic cavity was soiled with spillage of faecal matter

breathing and normal bowel sounds were also restored on the 2nd day

Pre -Operative Medical Management :-

- Sulphasalazine, Cyclophosphamide, Methylprednisolone, Diclofenac, Paracetamol, Multivitamins, and Supportive Care

Post -Operative Medical Management :-

- **Antibiotics :** Meropenem, Linezolid, Metronidazole, Netramycin, Fluconazole
- **IV Total Parenteral Nutrition**
- **Human Albumin**
- **Fluid And Electrolyte Maintenance Theraphy**

Post-Operative Complications

Patient developed breathlessness on 3rd post-operative day and he was found to have developed Pneumonic consolidation of left lung and he was managed effectively with appropriate measures. On the 7th post-operative day, patient developed fever with rigor and focal sepsis over the suture was discovered in the lower part of the abdomen and stitch abscess was made out. Simultaneously, he developed progressive abdominal distension and abdominal girth was 97 cm. Electrolyte imbalances was evaluated and Hypokalemic Ileus was established and was treated accordingly and it was normalized. He was then advised for oral fluids and oral sulphasalazine for ulcerative collitis. He was shifted to the special ward after 2 weeks of being in the ICU. He then developed fever and the central vein catheter tip and blood culture was done which showed candidiasis for which he was treated accordingly.

He was supported with IV TPN, multivitamins, sulphasalazine along with oral feeds. He became free from symptoms, he was started on oral feeds and his bowel habits were established and stool habits became almost normal and he was planned for discharge. Unexpected bleeding per rectum, prior to discharge of the patient due to overzealous use of sulphasalazine in the post-operative period caused us heavily. Blood loss was estimated to be 2 liters within 6 hours. Patient became hypotensive, BP 80/60 mmHg and tachycardia. He was aggressively treated with 6 units of whole blood, 5 units of plasma and single donor platelet to avoid further aggravation of Haematochezia and Disseminated Intravascular Coagulation (DIC). Sulphasalazine and other oral drugs were stopped.

Hematochezia stopped and vital signs restored to normal, BP 130/70 mmHg and heart rate at 84 beats/min. He was then kept for observation for a week. While under observation, the patient attained a stable state in which he had a normal diet and was symptom free. He was discharged in a good condition and was advised for follow up.

Discussion

Ulcerative colitis has now been recognized as a distinct disease entity for nearly 150 years. The first medical account of colitis by Sir Samuel Wilks of London in 1859 described a 42 year old woman who died after several months of diarrhea and fever, postmortem examination revealed a transmural ulcerative inflammation of the colon and terminal ileum that was originally designated as "simple ulcerative colitis" may in fact have been Crohn's disease. A subsequent case report in 1875, again by Wilks and Walter Moxon, that described ulceration and inflammation of the entire colon in a young woman who had succumbed to severe bloody diarrhea, was more likely the first detailed account of ulcerative colitis.

Ulcerative colitis is mainly seen in young people, it occurs between the age of 15-40 years and rarely seen at the age of 60 years. It is characterized by repetitious inflammatory episodes of intestinal inflammation followed by partial healing which leads to chronically disrupted bowel function. The recruitment and activation of numerous immune and inflammatory cells coupled with the release of proinflammatory mediators, perpetuates inflammation and facilitates damage to intestinal tissues. Immune-mediated inflammatory events include the

dysregulation of humoral and cell-mediated immunity and enhanced reactivity against intestinal bacterial antigens.

On gross inspection, the colonic mucosa appears swollen and congested; mucosa begins to erode leaving only a small island of mucosa that resembles polyps -pseudo-polyps. The mucosa, erosions often coalesces to form linear ulcers and superficial fissures, which becomes friable and erythematous with reduced haustral folds. The recurrent nature of the disease frequently leaves healed granular superficial ulcers superimposed on a friable and thickened mucosa with increased vascularity.

Histologically, the typical early lesion consists of an infiltration of inflammatory cells, primarily polymorphonuclear leukocytes, into the crypts at the base of the mucosa, forming crypt abscesses. In the most severe forms of the disease, such as fulminant colitis or toxic megacolon, the disease process may extend to the deeper muscular layer of the colon and even to the serosa.

Clinical Features

Patients with relatively mild episodes of ulcerative colitis typically present with bloody diarrhea, abdominal pain and fever. Rectosigmoid, it eventually progresses proximally in most cases. A smaller percentage (25%) present with a moderate attack in which bloody diarrhea is the major symptom. In a small number of patients (15 %), ulcerative colitis can present rapidly with a fulminating course. These patients develop the relatively sudden onset of frequent, bloody bowel movements, high fever, weight loss, and diffuse abdominal tenderness.

Extra intestinal manifestations of ulcerative colitis are observed in a number of organ systems. Many extra-intestinal manifestations of ulcerative colitis are closely related to disease activity and respond to therapy with steroids, immunosuppressive agents or surgical treatment. Liver and biliary tract disorders also commonly afflict patients with ulcerative colitis. Upto 80% of patients, especially those with pancolitis show some hepatic involvement. Sclerosing cholangitis, one of the most difficult extra-intestinal complications associated with ulcerative colitis is observed in 1-4% patients.

Diagnostic Modality

Endoscopy

Colonoscopy with biopsy plays an important role in diagnosis, management and surveillance of ulcerative colitis. It helps in differentiating ulcerative colitis from Crohn's disease. Since ulcerative colitis involves rectum, flexible sigmoidoscopy is the first step in the diagnosis. It shows loss of normal vascular pattern, a granular texture, micro ulceration and bleeding, purulent exudates and pseudopolyps in chronic cases.

Radiographic Studies

Plain radiograph of erect abdomen may demonstrate colonic dilatation and toxic megacolon and in fulminant case it shows free air in peritoneal cavity- diagnostic of perforation. Barium enema demonstrates colonic changes during acute and chronic ulcerative colitis.

Anagement of Ulcerative Colitis

Medical Management

Medical management of UC is not curative. It is mainly used to control patient's symptoms or managing the acute inflammatory process to induce remission. Choice of agents commonly used to induce remission may include oral and topical regimens alone or in combination. Drugs commonly used are sulfasalazine, corticosteroids, immunomodulators, suppressive antimetabolites, anti-tumor necrosis factor alpha, and biologics including Infliximab. Symptomatic anti-diarrheal and antispasmodic agents can be used in combination therapy.

Surgical Management

Indication for surgery

1. Emergency : Massive hemorrhage, Toxic Megacolon, Fulminant Colitis, Perforation, and Obstruction
2. Elective : Active disease with failed medical management, Dysplasia, Extra intestinal manifestation, Anorectal complications like Ischiorectal or Perirectal abscesses
3. Surgical option :
    - Proctocolectomy with Ileostomy: historically been the procedure of choice. This eliminates the risk of Malignant transformation.
    - Continent ileostomy: it differs from ileostomy in having ileal pouch that serves as reservoir for stool and an ileal conduit connecting the pouch to a cutaneous stoma. This operation was later modified to include an intestinal nipple valve between the pouch and stoma by intussuscepting the outflow tract from the pouch and then securing it with sutures or staples. Many complications are associated with this procedure are stagnant loop syndrome, enteritis, pouchitis small bowel obstruction, malabsorption.
    - Total proctocolectomy with ileal pouch anastomosis: it is total proctocolectomy, dissecting the rectal mucosa completely down to the dentate line of the anus and suturing the terminal ileum to anus circumferentially in an end to end fashion, which establishes a continuity of the intestinal tract. Due to increase stool frequency in patients after end to end anastomosis leads to modification of ileal pouching or reservoir of stool before ileoanal anastomosis-IPAA (Ileo Pouch Anal Anastomosis)
    - Subtotal Colectomy and Ileorectal Anastomosis: this can be opted as a procedure of choice in emergency background. It is less extensive procedure with more complication like impotence, bladder dysfunction, small bowel obstruction and chance of malignant transformation due to retained rectal mucosa. It eliminates the need of abdominal stoma.

Conclusion

Although UC is rare in Indians but not uncommon, awareness makes us to look for incidence of UC. This patient was presented as piles and colitis and was treated as piles but no improvement. UC was considered on clinical grounds and patient developed Toxic Megacolon with Colonic Perforation. Laparotomy was done with Total Colectomy, Subtotal Proctectomy along with Ileo-rectal anastomosis was carried out instead of Ileo-anal anastomosis and it was incidentally decided which has resulted in better quality of life in post-operative care, free from frequent stool habits and almost normal consistency of stools.

On follow up after discharge it is encouraging to note that patient is maintaining good health and attending his job as Marketing Officer in Chennai with good active ambulant career till now.

A case of massive tuberculous pericardial effusion

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Abstract

Tuberculosis is responsible for approximately 70% of cases of large pericardial effusion and most cases of constrictive pericarditis in developing countries, where most of the world's population live. Here we discuss about a 78 year old known case of Parkinsonism, hypertension and chronic hyponatremia who presented with breathlessness and chest pain. Chest X-Ray and 2D Echo showed evidence of massive pericardial effusion which was progressing to cardiac tamponade. Emergency pericardiocentesis was performed and the fluid was analysed. ADA was positive and Differential Count showed Lymphocytic predominance. The patient's LFT was elevated and hence modified ATT was started to which the patient responded well. Later, after the LFT normalized, regular ATT was started for the patient and over the subsequent reviews, the patient was found to have improved.

Introduction

Pericardial tuberculosis can be seen with 1 to 2% patients suffering from primary tuberculosis. Tuberculous involvement of the pericardium is well-known and can result in pericardial tamponade apart from other sequelae like constrictive pericarditis. Despite this, large pericardial effusions are uncommon, and manifestation as cardiac tamponade is rare. However, the mortality rate in untreated acute tuberculous pericardial effusion has been calculated to be around 85%. This kind of Pericardial Tuberculosis is also difficult to diagnose because a definitive diagnosis requires a biopsy of the tissue or pericardial fluid aspiration that needs to be observed under the microscope for mycobacterium tuberculosis.

Case Report

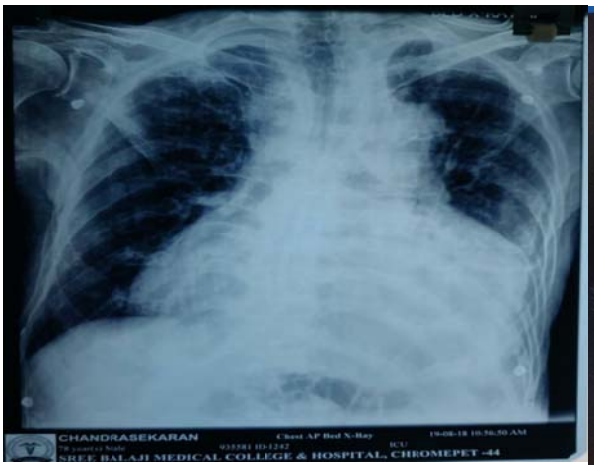
A 78 year old male patient who was a known case of Parkinson's Disease, Hypertension and Chronic Hyponatremia came with complaint of breathlessness for 2 ½ hours which was sudden in onset and occurs even at rest. Also, the patient complains of chest pain for 2 ½ hours which was central, non radiating, stabbing type. There was history of palpitations, generalised weakness and reduced activity. There was no history of syncope, sweating, cough or fever. He is a known case of hypertension – under regular treatment and Parkinsonism – under regular treatment (6 years). The patient was diagnosed with 'Low grade Papillary Urothelial Carcinoma Bladder' – on follow up since 5 to 6 years and currently not on any therapy. He is not a known case of diabetes, Bronchial Asthma, Tuberculosis, CVA or CAD. There was no history of similar episodes in the past. The patient had history of inhalation of snuff.

Clinical Examination

On examination, the patient was conscious, oriented, afebrile and dyspneic. Pallor was noticed. The BP was 120/80 mm Hg and Pulse Rate was 148/min. SpO_2 was 94% at room air. His CBG was 129 mg/dl. The CVS examination showed muffled heart sounds. The rest of the systemic examination was unremarkable besides the presence of resting tremors.

Investigations

The basic investigations revealed the following results; Hb = 11.2g%, PCV = 33.7%, TC = 8740/cumm, Platelets = 3 lacs/cumm, RFT = Normal, Total Bilirubin = 0.5 mg/dl (Direct = 0.3), Total Protein = 5.7 g/dl (Albumin = 2.9 g/dl), SGOT = 84 IU/L, SGPT = 200 IU/L, ALP = 77 IU/L, GGT = 32 IU/L.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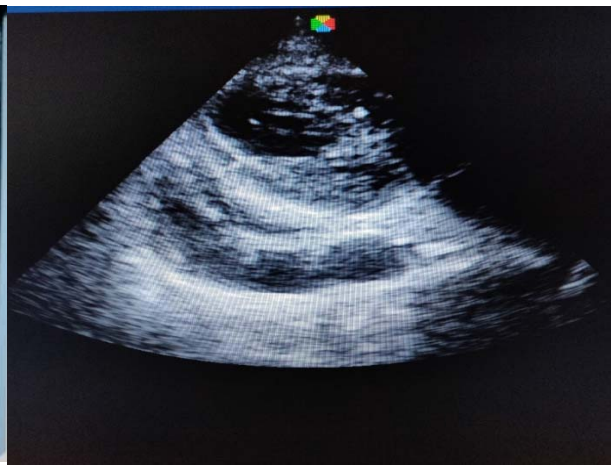

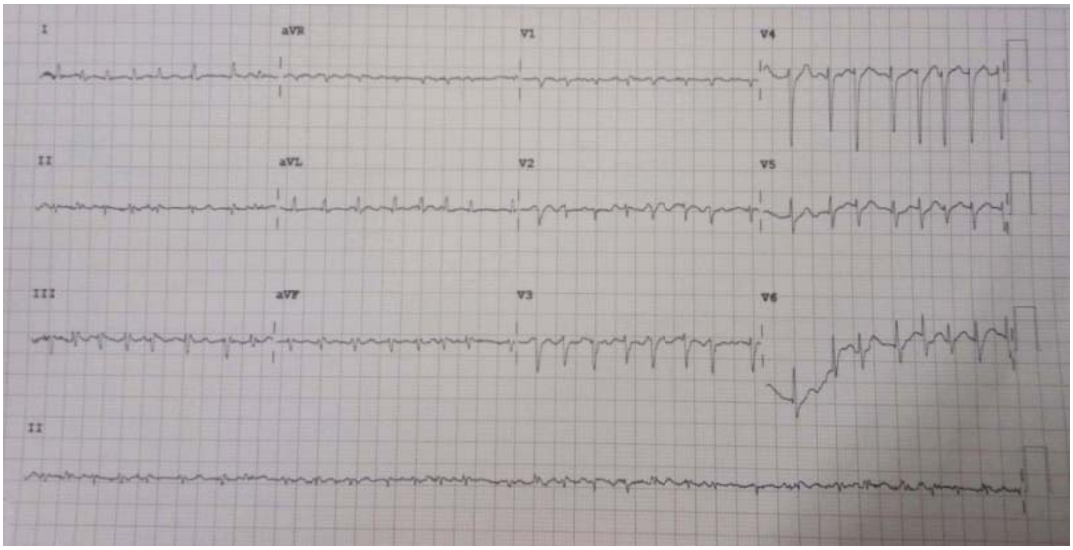
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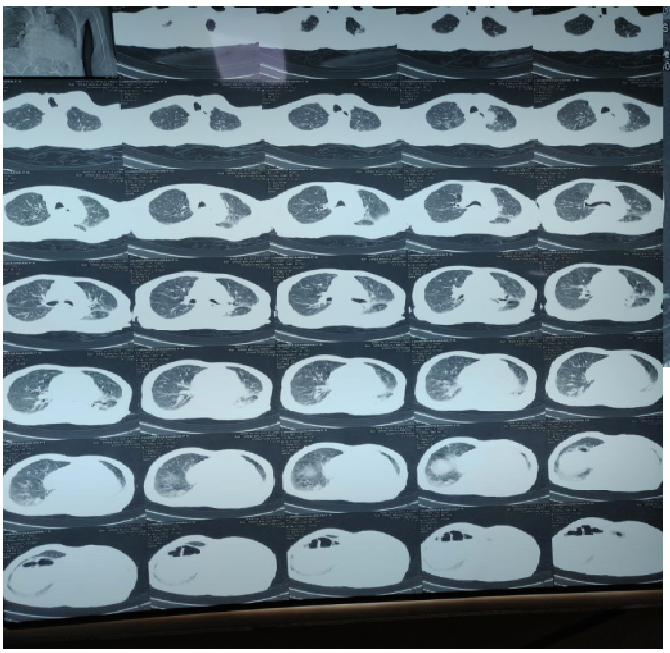

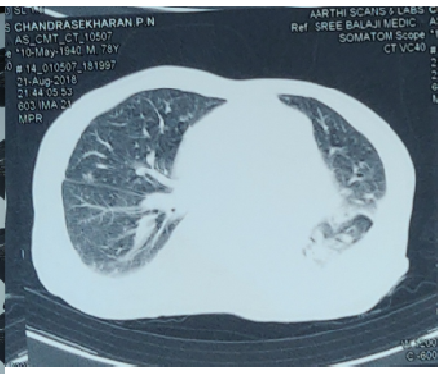
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Pericardial Fluid Analysis revealed; Sugar = 70 mg/dl, Protein = 4.4 g/dl, DC – (Lymphocytes = 79%, Neutrophils = 20%, Eosinophils = 1 %), LDH = 2953.2 U/L, ADA = 72.2 U/L, AFB –ve. Culture showed no growth and Cytological study showed 'acute and chronic inflammatory cells in proteinaceous fluid background'

Treatment given

Patient was admitted into ICU and emergency PERICARDIOCENTESIS was performed. Continuous drain with pigtail catheter was placed and 600 ml of hemorrhagic pericardial fluid was drained overnight. Patient developed atrial fibrillatory pattern in ECG following the procedure, which was reverted with Amiodarone infusion.

Inj. Heparin 5000 U IV 1 – 0 – 1
Inj. Ceftriaxone 1g IV 1 – 0 – 1
Inj. Streptomycin 750 mg IV 1 – 0 – 0
T. Levofloxacin 750 mg 1 – 0 – 0
T. Ethambutol 800 mg 1 – 0 – 0
T. Wysolone 40 mg 1 – 0 - 0

Discussion

The incidence of tuberculous pericarditis is increasing with the advent of the AIDS pandemic. Tuberculosis accounts for up to 4% of acute pericarditis and 7% of cardiac tamponade. The mortality rate of tuberculosis still ranges from 14-40%. Tuberculous pericarditis is a potentially lethal condition. The pericardial effusion is mainly due to hypersensitivity to tubercular protein. The route of spread of the organisms to pericardium is usually from mediastinal or hilar lymph nodes or from lungs or rarely as a part of miliary tuberculosis. Typically, the process begins as effusive constrictive pericarditis. In later stages, AFB are usually not detected but caseating granulomas involving the pericardium and epicardium may be present. Cardiac tamponade may present as a complication of pericardial effusion. In the present case, any such complications were averted due to the early diagnosis and prompt treatment. Prompt treatment of tuberculous pericarditis may be lifesaving. Effective treatment requires a rapid and accurate diagnosis, which is often difficult.

Definite tuberculous pericarditis can be diagnosed by one or more of the following criteria:

- Isolation of *M. tuberculosis* from pericardial effusion fluid or pericardial biopsy.
- Demonstration of granulomatous inflammation on histologic examination of pericardial biopsy sample.
- Isolation of *M. tuberculosis* from sputum or non pericardial effusion exudates in the presence of clinical and/or radiological evidence of tuberculosis, associated with a positive response to antitubercular therapy and in the absence of any other obvious cause for pericarditis.

ADA, a product of T-Lymphocytes is reviewed as an excellent, time-effective and cost-effective marker for the diagnosis of tuberculous pleural or pericardial effusion. Almost all research workers have shown sensitivity and specificity of 90% to 100% for the value of ADA using different cut off levels. ADA is also increased in conditions like Rheumatoid arthritis, Sarcoidosis, Psoriasis, etc. Therefore, these conditions should be ruled out clinically or through investigations.

Conclusion

Massive pericardial effusion, irrespective of the etiology is potentially lethal and therefore a medical emergency. Though it is a rare presentation of extrapulmonary tuberculosis, in countries like India where it is common, a differential of Tuberculosis must always be borne in mind whenever pericardial effusion is encountered and must be imperatively investigated for. With the advent of new tests, methods and technological advancements the diagnosis and prompt intervention can be achieved and therefore the rate of mortality can be significantly reduced.

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An Unsual Case of Easy Fatiguability

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Introduction

Wilsons disease is autosomal recessive disorder, mutation of atp7B gene. It impairs biliary copper excretion, accumulation and toxicity. Systemic involvement seen characterised by hepatolenticular degeneration. Diagnosis based on sr.ceruloplasmin, 24hr urine copper levels, slit lamp examination, gold standard being liver biopsy.

Diagnosis

45yrs old female was admitted with complaints of generalised weakness & easy fatiguability since 3 weeks. Preliminary investigations were done. USG abdomen showed Coarse echogenic echotexture, multiple nodular lesions in the liver, liver cirrhosis, presence of perihepatic fat layer and caudate lobe was found normal. Serum ceruloplasmin was low - 12mg/dL ; 24hr urine copper was high - 156mcg Kayser-fleischer rings: were negative. CT abdomen showed multiple nodular lesion in liver indicating the presence of perihepatic fat layer and a normal caudate lobe was noted. As the liver was already shrunken cirrhosis stage. In cirrhotic stage of wilsons disease copper content of individual regenerative nodules vary. Hence the procedure was not conducted. Patient diagnosed with Wilsons disease. Management of patient based on Nazer prognostic index, score < 7 medical management, > 9 liver transplant. Patient treated with anticopper drugs, penicillamine and zinc.

Discussion

Patient diagnosed with Wilson's disease which is an autosomal recessive disorder caused by mutations in the atp7b gene. The gene encodes a membrane-bound, copper transporting ATPase. Hepatic involvement Present as hepatitis, cirrhosis, or hepatic decompensation. Patient treated with anticopper drugs. Liver biopsy could not be done. Kayser Fleischer ring is positive in only 30-50% of patients with hepatic or presymptomatic state, the absence of ring do not exclude the diagnosis.

Tables : Nazer Prognostic Scoring

		Score (in Points)				
Laboratory Measurement	**Normal Value**	**0**	**1**	**2**	**3**	**4**
Serum bilirubin[a]	0.2–1.2 mg/dL	<5.8	5.8–8.8	8.8–11.7	11.7–17.5	>17.5
Serum aspartate aminotransferase	10–35 IU/L	<100	100–150	151–200	201–300	>300
Prolongation of prothrombin time (sec)	—	<4	4–8	9–12	13–20	>20

[a]If hemolysis is present, serum bilirubin cannot be used as a measure of liver function until the hemolysis subsides.

Source: Modified from H Nazer et al: Gut 27:1377, 1986; with permission from BMJ Publishing Group.

scores <7 : medical therapy

scores >9 : liver transplantation

scores 7 – 9: clinical judgment is required in deciding whether to recommend transplantation or medical therapy

Test	**Usefulness[a]**	**Normal Value**	**Heterozygous Carriers**	**Wilson's Disease**
Serum ceruloplasmin	+	180–350 mg/L (18–35 mg/dL)	Low in 20%	Low in 90%
Kayser- Fleischer rings	++	Absent	Absent	Present in >99% if neurologic or psychiatric symptoms are present Present in 30–50% in hepatic presentation and presymptomatic state
Urine copper (24-h)	+++	0.3–0.8 µmol (20–50 µg)	Normal to 1.3 µmol (80 µg)	>1.6 µmol (>100 µg) in symptomatic patients; 0.9 to >1.6 µmol (60 to >100 µg) in presymptomatic patients
Liver copper	++++	0.3–0.8 µmol/g (20–50 µg/g of tissue)	Normal to 2.0 µmol (125 µg)	>3.1 µmol (>200 µg) (Obstructive liver disease can cause false-positive results.)
Haplotype analysis	++++ (siblings only)	0 matches	1 match	2 matches

[a]Usefulness range: + (somewhat useful) to ++++ (very useful).

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Folliculitis

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Introduction

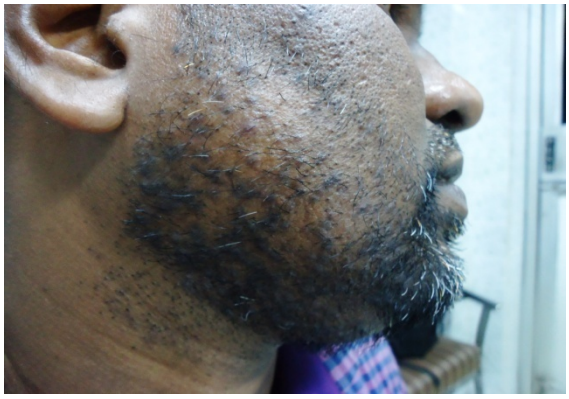
Folliculitis is an inflammation of the hair follicle affecting both children and adults. Clinically, it appears as follicular-based erythematous papules or pustules in a hair-bearing area. Sometimes a collarette of scale may be the only clinical clue. Although typically asymptomatic, patients may experience itching, tenderness, or pain. The most common affected areas include the head and neck (particularly the beard and scalp), upper trunk, buttocks, legs, axillae, and groin. Occluded areas and areas with terminal hair are also favored.

Diagnosis

Folliculitis

Description

A 22-year-old healthy male college student presented for management of 7 days' history of a pustular eruption on the face. There was no significant past medical history. Review of systems was non-contributory.



Pathogenesis

Folliculitis is typically categorized based on infectious or noninfectious etiologies.

Infectious folliculitis can be due to bacteria, fungi, viruses, or ectoparasites and can be exacerbated by shaving, occlusion, and rubbing. *Staphylococcus aureus*is the most common infectious cause. Gram-negative folliculitis due to *Klebsiella, Proteus, and Enterobacter*can develop in acne patients treated with a prolonged course of oral antibiotics, while pseudomonal folliculitis is associated with hot tub use. Fungal causes include dermatophytes, *Malassezia* spp (*Pityrosporum*) and *Candida* spp. The most common viral causes are the herpesviruses (varicella zoster virus more commonly than herpes simplex virus), while the ectoparasite, *Demodex spp*, can cause its own subtype of infectious folliculitis or demodicidosis.

The main causes of **noninfectious folliculitis** include irritation, occlusion, and medication use. Eosinophilic pustular folliculitis (EPF) and actinic folliculitis are 2 rare forms of folliculitis that occur secondary to eosinophilia and UV exposure, respectively. Folliculitis due to irritation and occlusion may occur with the use of certain topical products, friction from clothing, heat, or sweat. Medications associated with drug-induced folliculitis include the epidermal growth factor receptor inhibitors, corticosteroids, sirolimus, cyclosporin, lithium, lamotrigine, aripiprazole, chlorpromazine, dantrolene, dapsone, and halogens.

EPF or Ofuji disease is a rare form of chronic and recurrent folliculitis characterized by the presence of eosinophils within the follicular epithelium and perifollicular stroma on histology and associated peripheral eosinophilia. Three clinical variants have been described: classic EPF, immunosuppression-associated EPF, and infancy-associated EPF. EPF commonly occurs on the face, extremities, and upper trunk and is frequently associated with intense pruritus. EPF flares tend to recur every 3-4 weeks with a sudden eruption of crops of papulopustules lasting on

average for 7-10 days. Lesions characteristically will increase in size and merge to form plaques with central clearing.

Actinic superficial folliculitis is another very rare form of folliculitis that occurs secondary to UV exposure. Monomorphic papules and pustules occur in sun-exposed, sebaceous-rich areas of the shoulders, trunk, and arms typically within 24-36 hours. The face is typically spared. Only a few cases have been reported in the literature.

Clinical features

Folliculitis is a common inflammatory condition of the hair follicles that can affect any hair-bearing area of the body. It occurs in both children and adults and is characterized by the presence of follicular-based papules and pustules. Folliculitis can occur due to infectious (ie, bacterial, yeast, viral, ectoparasitic) and noninfectious (irritation, occlusion, and medications) causes. The diagnosis of folliculitis is clinical and rarely requires biopsy confirmation. When infection is suspected, swab cultures may be useful in guiding appropriate treatment. Treatment of folliculitis varies depending on the underlying cause. Eosinophilic pustular folliculitis (EPF) is a rare form of chronic and recurrent folliculitis that on histology is characterized by the presence of eosinophils within the follicular epithelium and perifollicular stroma and is associated peripheral eosinophilia seen in infants, adults, and the immunosuppressed.

Evaluation

When evaluating patients with folliculitis, a thorough history and examination should be performed to determine possible causes and to distinguish between infectious vs noninfectious etiology. While mostly a clinical diagnosis, dermoscopic and/or histological examination may rarely be indicated. When an infectious bacterial etiology is suspected, a Gram stain and bacterial culture can be helpful in guiding treatment, particularly in severe, recurrent, or resistant cases or when systemic treatment may be needed. If indicated, viral swabs and KOH preparation can be done to assess for viral or fungal/yeast etiologies, respectively.

Differential diagnosis

- Folliculitis may be confused with other common disorders of the hair follicle such as acne vulgaris. A good history and physical examination should help aid in the diagnosis of this condition.
- Infectious vs noninfectious etiology should be distinguished when determining treatment.
- In all cases of folliculitis, regardless of etiology, general good skin hygiene and avoidance of occlusive and irritating products are important steps in improving the condition.

Treatment

Identifying the underlying etiology is necessary in determining appropriate treatment. Infectious folliculitis due to bacteria, fungus, virus, or ectoparasite may be treated with use of targeted antibiotics, antifungals, antivirals, or antiparasitics, respectively. Noninfectious folliculitis should be treated by identifying and, where possible, eliminating the underlying cause. Patients should avoid tight-fitting clothing and may apply an astringent powder or an astringent solution (such as aluminum chloride) to decrease moisture from sweat. If patients are shaving, they should shave in the direction of hair growth to avoid irritation of the hair follicles. Patients should be instructed to avoid occlusive or irritating topical products or behaviors. In drug-induced folliculitis, the offending agent should be discontinued if and when possible. If not possible to discontinue the medication, supportive care should be recommended including gentle skin care and hygiene and avoidance of any occlusive or irritating factors.

Patients with actinic folliculitis should be instructed to avoid UV exposure; sunscreen may be of some benefit in these patients; topical steroids may improve pruritus. In severe cases, tretinoin 0.35-0.5 mg/kg/day for 3-6 months may

be of benefit. Antihistamines, topical antipruritics, and topical corticosteroids may help relieve pruritus in patients with EPF. Infantile EPF is self-limited and tends to resolve on its own. Patients who are immunocompromised (ie, due to AIDS or malignancies) may resolve with improvement of their immune system. Other reported second- and third-line treatments for refractory cases of EPF include UVB phototherapy, topical calcineuron inhibitors, topical permethrin, oral itraconazole, oral antibiotics, oral dapsone, oral steroids, oral isotretinoin, and interferon.

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Electrical Alternans

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Background

Electrical alternans is a broad term that describes alternate-beat variation in the direction, amplitude, and duration of any component of the ECG waveform (ie, P, PR, QRS, R-R, ST, T, U). It was first recognized by Hearing in 1909 and further characterized by Sir Thomas Lewis in 1910 as occurring "either when the heart muscle is normal but the heart rate is very fast or when there is serious heart disease and the rate is normal." Kalter and Schwartz first identified electrical alternans on surface ECG in 1948. [1] Electrical alternans must be distinguished from mechanical alternans (eg, pulsusalternans), although both may coexist.

Pathophysiology

The pathophysiologic mechanisms that cause electrical alternans can be divided into 3 categories: (1) repolarization alternans (ST, T, U alternans), (2) conduction and refractoriness alternans (P, PR, QRS alternans), and (3) alternans due to cardiac motion. True electrical alternans is a repolarization or conduction abnormality of the Purkinje fibers or myocardium. The cellular mechanism behind electrical alternans is thought to be due to abnormal calcium cycling, either impaired release or impaired reuptake of sarcoplasmic reticulum calcium. Electrical alternans due to cardiac motion is effectively artifact, as the heart swings in relation to the chest wall and electrodes, with a period twice that of the heart rate.

Repolarization alternans can be further sub classified as T-wave alternans and ST-segment alternans. T-wave alternans is associated with rapid changes in heart rate or prolongation of the QT interval. A long QT interval is associated with polymorphic ventricular tachycardia (ie, torsade de pointes); therefore, T-wave alternans is a possible precursor to torsade de pointes.

T-wave alternans has been reported with congenital long QT syndrome electrolyte imbalances (eg, hypocalcaemia, hypokalemia, hypomagnesaemia), treatment with quinidine or Amiodarone, hypertrophic cardiomyopathy, alcoholic cardiomyopathy, congestive heart failure, and acute pulmonary embolism. T-wave alternans has also been reported following cardiac resuscitation. Most importantly, the presence of T-wave alternans can be used as a predictor of ventricular tachyarrhythmic events, such as sudden cardiac death sustained ventricular tachycardia, ventricular fibrillation, implantable Cardioverted defibrillator (ICD) therapy for ventricular tachyarrhythmia, and cardiac arrest.

ST-segment alternans describes alternating levels of ST elevation, usually in the presence of myocardial ischemia. It has been reported with vasospastic angina pectoris, acute myocardial infarction, non-vasospastic angina pectoris, during exercise tests, during percutaneous transluminal coronary angioplasty (PTCA), and after subarachnoid hemorrhage. ST alternans during acute ischemia has been associated with appearance of ventricular arrhythmia, including ventricular tachycardia and ventricular fibrillation.

Conduction alternans is an alternation of impulse propagation along any of the anatomic structures involved in conveyance of electrical impulse and is usually precipitated by changes in heart rate or input from nervous, humoral, or pharmacologic components. Conduction alternans may be seen in the setting of myocardial ischemia, atrial fibrillation, Wolff-Parkinson-White syndrome, rheumatic heart disease, acute pulmonary embolism, myocardial contusion, and

left ventricular dysfunction. It may manifest on the surface ECG as alternation of the P wave, QRS complex, PR interval, R-R interval, or any combination of these. QRS alternans during narrow complex tachycardia has been suggested as a marker for orthodromic atrioventricular (AV) re-entrant tachycardia using an accessory pathway as a retrograde limb.

Electrical alternans associated with cardiac motion is due to alternation in the position of the heart with relation to recording electrodes. The most common underlying disorder is an enlarged pericardial sac; however, not all pericardial effusions cause electrical alternans. The presence of pericardial disease and total electrical alternans (P, QRS, and T wave) frequently suggests cardiac tamponade, but total electrical alternans is seen in only 5-10% of patients with cardiac tamponade. Heart movement in patients with hypertrophic cardiomyopathy also may result in electrical alternans of this type.

History

The presence of electrical alternans has no clinical manifestations outside those present from the underlying cause or association. A search for the underlying cause of electrical alternans is warranted. For example, the patient with ST-T alternans may complain of chest pain, shortness of breath, or profuse sweating caused by myocardial ischemia. The patient with QRS alternans may complain of syncope resulting from underlying hypertrophic cardiomyopathy.

Physical

Electrical alternans does not result in physical findings separate from the underlying cause of the alternans. A physical examination, searching for the underlying cause, is necessary. This may reveal, for example, jugular venous distension and hypotension from cardiac tamponade. Making the distinction between electrical alternans and mechanical alternans is important. Mechanical alternans may have physical findings (eg, pulsusalternans).

Causes

Repolarization (ST – T alternans)

- Vasospastic angina pectoris
- Acute myocardial infarction
- Nonvasospastic angina pectoris
- Congenital long QT syndrome
- Electrolyte imbalances, such as hypocalcaemia, hypokalemia and hypomagnesaemia
- Treatment with quinidine
- Treatment with Amiodarone
- Hypertrophic cardiomyopathy
- Alcoholic cardiomyopathy
- Congestive heart failure
- Acute pulmonary embolism
- Following cardiac resuscitation
- During exercise tests
- Acute mental stress
- During PTCA
- After subarachnoid haemorrhage

Conduction (QRS alternans)

- Myocardial ischemia
- Atrial fibrillation
- Wolff-Parkinson-White syndrome
- Rheumatic heart disease
- Acute pulmonary embolism
- Myocardial contusion
- Left ventricular dysfunction

Cardiac motion

- Large pericardial effusion
- Hypertrophic cardiomyopathy

Laboratory Studies

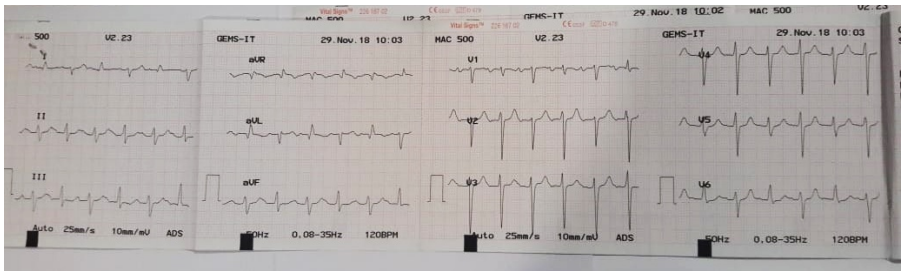
Direct laboratory investigations toward discovery of the primary underlying etiology of electrical alternans. Based on other clinical information, appropriate lab studies include cardiac enzymes for myocardial ischemia and infarction as well as serum calcium, potassium, and magnesium if electrolyte abnormalities are suspected. In the setting of a large pericardial

effusion, laboratory studies searching for a malignancy may be warranted.

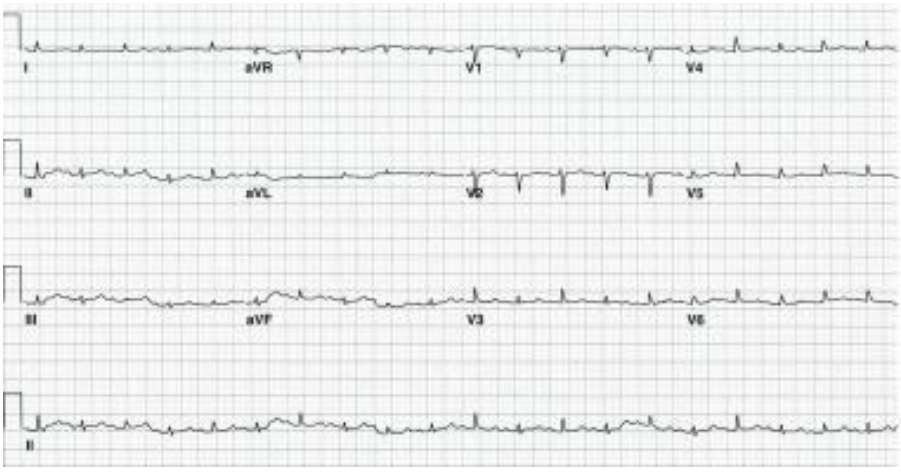
Imaging Studies

Chest radiography

- Chest radiograph may reveal an enlarged cardiac silhouette, possibly indicating cardiomyopathy or large pericardial effusion.
- Evidence of the Westermark sign or Hampton hump may suggest pulmonary embolism as the cause of electrical alternans.

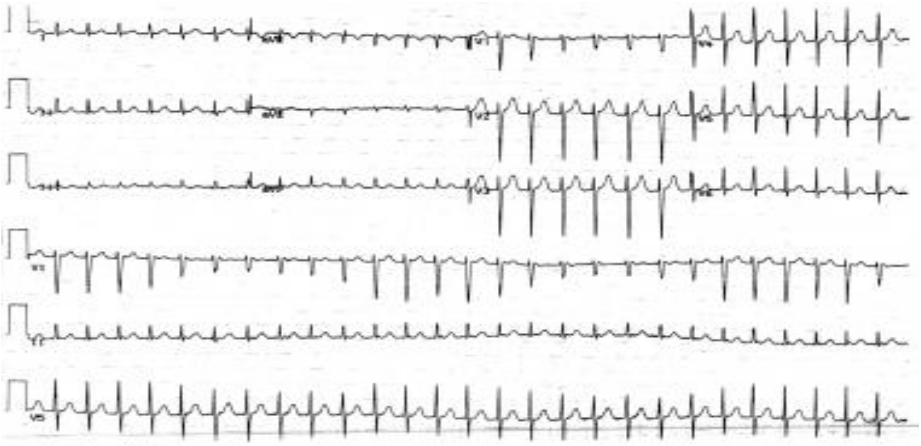


70y/m post CABG with severe LV dysfunction admitted with heart failure with QRS alternans



Acknowledgement: Emedicine; medscape

Typical alternate-beat QRS electrical alternans. Note that QRS voltage is low.



Acknowledgement: Emedicine; medscape

Echocardiography

- Echocardiography should be performed on those patients with total electrical alternans (P, QRS, and T wave) to evaluate for pericardial effusion.
- Echocardiography is also necessary for evaluation of patients with hypertrophic cardiomyopathy, alcoholic cardiomyopathy, or congestive heart failure.

Other Tests

Electrocardiogram

- ECG is the main study through which electrical alternans is discovered. Any or all components of the electrical waveforms may exhibit alternans (see following images).

Supraventricular tachycardia with alternans. Note the phasic nature to the QRS morphology, particularly in the rhythm strip in V1.

- High-resolution ECG with spectral analysis can detect alternans in the microvolt range of amplitude. This detailed study is appropriate when searching for T-wave alternans as a predictor of ventricular tachyarrhythmia events.
- Routine ambulatory ECG monitoring of T-wave alternans, using dynamic, nonspectral, modified moving average analysis, may be helpful for risk stratification for arrhythmias.
- T-wave alternans may be seen best in lead V_2.
- T-wave alternans can be detected by use of implantable cardio defibrillators (ICDs).

Treatment

Medical Care

Direct treatment toward correction of the underlying cause of electrical alternans. For example, myocardial infarction should be treated using standard measures (eg, consider thrombolytic administration or PTCA). Pericardiocentesis for cardiac tamponade. Long QT syndrome may be treated with removal of offending drugs or correction of metabolic abnormalities.

Surgical Care

Most diseases that cause true electrical alternans do not require surgical treatment. Pulmonary embolectomy may be required for unresolved large pulmonary emboli. Left-sided cervicothoracic sympathetic ganglionectomy may be required for patients with congenital long QT syndrome who continue to have episodes of syncope despite drug therapy. Recurrent pericardial effusions may benefit from pericardiectomy.

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Colchicine poisoning: the dark side of an old drug

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Colchicine is a widely prescribed and effective medication for the treatment of gouty arthritis. Plants such as autumn crocus or meadow saffron (Colchicum autumnale) and glory lily (gloriosa superba) contain colchicine alkaloids. Colchicine toxicity is rare but any intention overdose with colchicine is potentially lethal.

1. Is the plant is more toxic?

   No, less toxicity is reported from colchicine-containing plants than from the medication.

2. Is every part of the plants are toxic?

   Yes, however the toxicities vary. The seeds can contain up to 0.8% colchicine, the corm (stalk) up to 0.6%, and flowers 0.1%. The tubers contain approximately 6 mg colchicine per 10 grams of tuber.

3. What is the lethal dose?

   The lethal dose is generally considered to be above 0.5mg/kg. However, one-off ingestions as low as 7mg in adults have resulted in death.

4. What is the Toxic mechanism of colchicine?

   It binds tubulin, preventing microtubule formation thus inhibiting mitosis. Therefore any tissues with a high cellular turnover are affected (Gastrointestinal tract, bone marrow).

5. What are the pharmacokinetic properties of colchicine?

Absorption

- Rapidly orally absorbed, peak serum concentrations at 0.5-3 hours.
- Bioavailability is about 50% due to first-pass metabolism.

Distribution

- 50% protein bound, distributed to all tissues, high Vd (2-8L/kg, although may be higher in OD).

Metabolism

- Liver metabolism CYP3A4, but extensive enterohepatic recirculation.

Excretion

- Renal and fecal; elimination half-life 26-30 hours.

6. What initial signs, symptoms and history should raise a clinician's index of suspicion for colchicine toxicity?

   Persistent nausea, abdominal pain, vomiting and diarrhea in a patient with a history of arthritis should immediately direct the clinician to consider colchicine poisoning in the differential diagnosis.

7. What are the clinical manifestations of colchicine toxicity?

   There are early and late phases of colchicine toxicity. The typical time course and manifestations are as given below

2-24h

- GI symptoms — nausea, vomiting, diarrhoea, abdominal pain.
- Hemodynamic instability may result from GI fluid loss.
- Leukocytosis may be seen on FBC.

2-7 days

- Bone marrow suppression and pancytopenia
- Rhabdomyolysis
- Renal failure
- Metabolic acidosis
- Respiratory failure and ARDS
- Cardiac dysrhythmias — potentially lethal.

>7 days

- Rebound leucocytosis and transient alopecia in patients fortunate enough to survive to this stage.
- Sensory and motor neuromyopathies, parethesias and other neurologic toxicities have all been reported after recovery from the second phase of colchicine poisoning.
- In some cases these symptoms have persisted longer than one year.

8. Is there an antidote available for colchicine poisoning?

   Colchicine Fab has been described in animal models, and used successfully in a single human case (late presentation, 0.96mg/kg, France), but is not readily available. Aggressive supportive care is the treatment of colchicine poisoning.

9. How Activated charcoal is helpful in colchicine poisoning?

   The administration of activated charcoal 1 g/kg as soon as possible in any patient who has ingested > 0.5 mg/kg of colchicine as even a small decrease in absorption may be maybe lifesaving.

10. Is multi-dose activated charcoal is useful?

    Although charcoal is potentially beneficial in preventing colchicine absorption and significant enterohepatic recirculation, it is of limited utility due to initial symptoms of nausea, vomiting and hemorrhagic gastritis.

11. Does hemodialysis is recommended for enhanced elimination of colchicine?

    Due to colchicine's extensive volume of distribution and excellent tissue binding, hemodialysis and hemoperfusion do not effectively remove the drug and are not recommended.

12. What is the role of Granulocyte-colony stimulating factor in colchicine poisoning?

    Patients who develop bone marrow suppression should be placed on neutropenic precautions. Colony stimulating factors such as G-CSF have been used for the treatment of colchicine-induced leukopenia. However, this may be more useful to treat secondary sepsis rather than colchicine-induced leukopenia.

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Improving Nutrition In Women With Diabetes Mellitus

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A review of the literature shows that women with T2dm often have worse CVD risk profiles and outcomes compared with men. Women with diabetes have a 3 to 5-fold higher risk of developing CVD since, Obesity as a risk factor is more common in women. Gender differences arise from socio-cultural processes such as different behaviors of women and men, exposure to specific environmental influences, differences in nutrition, lifestyle or stress, or attitude towards treatments and prevention.

Risk Factors

1. Biological Risk Factors

- Body Mass Index (BMI)

  Diabetic women are more obese than diabetic men in most studies and show a stronger association between increase of BMI and diabetes risk. According to the Global Nutrition report (Lancet, 2016) 20 million Asian women are obese when compared to 9.8 million men and 4 million women are super obese with BMI > 40kg/m^2
- Body Fat Distribution

  The visceral obesity increases the risk of diabetes and cardio- metabolic risk in terms of glucose and lipid abnormalities more in women.
- Metabolic Syndrome (MetS)

  Diabetes appears to diminish the favorable cluster of MetS risk factors of females compared with males and increases the risk of CVD complications
- Adipokines (Leptins and Adiponectins)

  Women show an up-regulation of expression of adiponectin and its receptor in abdominal adipose tissue, possibly contributing to their lower cardio metabolic risk. Adiponectin and WC are important predictors of insulin resistance even in healthy non- diabetic women.
- Imbalance of Sex Hormones

  The PCOS describes a female specific state of excess androgen and hyperinsulinemia related to obesity, T2DM, and high cardiometabolic risk.
- Prediabetes

  Prediabetes in women is expressed as IGT which is primarily due to peripheral insulin resistance.

2. Psychosocial Risk Factors

Females appear to be more vulnerable to the so-called allostatic load, the imbalance between the ability to adapt to environmental demand and over exposure to environmental stress. Women at all ages show 40% higher risk for suffering from insomnia, which in turn was associated with insulin resistance and obesity

Hypothyroidism & Diabetes

In hypothyroidism, glucose induced insulin secretion by the cells is reduced; the rates of glucose oxidation and glycogen synthesis are also decreased. Excess thyroid hormone leads to hyperglycemia via multiple mechanisms increasing hepatic glucose output which leads to hyperinsulinemia, thus progressing towards glucose intolerance and further development of peripheral insulin resistance.

PCOS & Insulin Resistance

The South Asian population, in general, also exhibit higher prevalence of insulin resistance and T2DM which may increase long term morbidity among those with PCOS. Recent research indicated higher insulin concentrations and lower insulin sensitivity in South Asian women with PCOS.

- Abdominal obesity is an important component of PCOS that affects 30-70% of the PCOS population
- Insulin resistance is present in women with PCOS independent of body mass. However, obesity in PCOS is associated with greater insulin resistance, and a higher incidence of dyslipidemia, hypertension and T2DM.
- Fertility is decreased, and the rate of spontaneous abortion is increased

Nutrition & PCOS

Effective approaches to nutrition and exercise improves endocrine features, reproductive function and cardio metabolic risk factors. Diet and exercise need to be tailored to the individual's needs and preferences. Studies in patients with PCOS confirm that modest weight loss improves glucose tolerance, cardiovascular risk profile and reproductive function. Many studies in overweight and obese subjects shown beneficial effects of even modest (5%) weight loss on well-being, insulin sensitivity, and cardiovascular risk profile. A 5% to 8.6% weight loss results in reduction in HbA1c by 0.2% and 0.6% respectively. Calorie intake should be distributed between several meals per day. A daily calories deficit of 200 kcal/day will prevent weight gain; a deficit of 500 kcal/day is needed for the average person to lose 0.5 kg/week, while a 1000 kcal deficit is required for 1 kg weight loss/week. Fat should be restricted < 25 % of total calories with low proportion of saturated fat.

Dietary Carbohydrates

A recent study demonstrated that a reduced carbohydrates diet results in lower measure of beta cell responsiveness and circulating insulin (27%reduction in fasting insulin) when compared with a standard higher carbohydrate diet. Increased consumption of high GI carbohydrate contributes to dyslipidemia and weight gain, also stimulates hunger and craving.

Glycemic Index

It has been shown that eating foods with a low GI improves glucose control in women with PCOS and diabetes.

Dietary Protein

Protein should be high biological value that improves hormonal function.Diets that are either low in fat or low in carbohydrates almost inevitably deliver an increased proportion of calorie intake as protein. Adequate protein intake is important to protect lean body mass and to increase muscle in response to exercise. There is little evidence to suggest benefits of high protein diet on insulin resistance and several studies in women with PCOS have failed to show significant long-term benefits of a high protein diet on weight loss or insulin sensitivity. There are also concerns about the safety of high protein, low carbohydrates diets including the effect of kidney function and bone mineral density. General advice is that the diet should deliver up to 20% of its calories as protein, this may increase at the expense of the other dietary components for short term diets designed to help the patient lose weight or improve glucose tolerance.

Dietary Fat

Increased consumption of unsaturated fatty acids has been reported to improve insulin sensitivity in healthy, obese and type2 diabetic subjects.

Type of Fats

Type of Fat	Main Source
Monounsaturated	Canola, peanut, and olive oils; avocados; nuts such as pumpkin and sesame seeds.
Polyunsaturated	Sunflower, corn, soybean, and flaxseed oils, foods such as walnuts, flax seeds, and fish.
Saturated	Whole milk, butter, cheese, and ice cream; red meat; chocolate; coconuts, coconut milk, and coconut oil
Trans	Some margarines; vegetable shortening; partially hydrogenated vegetable oil; deep fried foods; many fast foods; some commercial baked goods (check labels)

The longer chain PUFAs, Eicosa pentaenoic acid and docosa hexaenoic acid which are found in fatty fish have beneficial effects on metabolic parameters in patients with diabetes, but specific evidence relating to PCOS is not available at this stage. While the Mediterranean diet rich in Mono Unsaturated Fatty Acids (MUFA), has been widely accepted as a gold standard for healthy diets, its potential benefits in patients with PCOS have not been documented, although decreased features of obesity and insulin resistance have been noted in patient with PCOS. Over all, dietary fat should account for no more than 30% of the calorie content of the diet, with <7% of calories coming from saturated fat. The remainder of the fat content should be as a balanced mixture of unsaturated fat including cooking oils and spreads. Cheese has been reported to be less insulinemic than other dairy products. Moderation is the key (Br J Nutr. 2005). Consumption of Trans fat has been linked with increased risk of anovulatory infertility.

Salt

The Dietary approach to stop hypertension (DASH) eating pattern resulted in the improvement of insulin resistance, serum hs-CRP levels, and abdominal fat accumulation in overweight women with PCOS. (Chavarro, 2007)

Antioxidants

Therapeutic strategy to reduce the oxidative stress includes diet rich in fruits and vegetables, weight reduction, physical exercise, smoking cessation, alcohol consumption reduction and adequate number of sleeping hours.

Vitamin D

Hypovitaminosis D was found in about 80% of PCOS women.Calcium has an important role in follicle development and both calcium and vitamin D deficiencies are considered as potential risk factors for insulin resistance and obesity.

Endocrine Disrupters

Bisphenol A (BPA) is found to have obesogenic properties, disrupting normal metabolic activity and making the body prone to overweight. (SeminReprod Med.2014). Environmental exposure to industrial products, particularly BPA, may also exacerbate the clinical course of PCOS. Eating plastic-packaged food, eating fruit with pericarp and drinking alcohol were found to be the independent risk factors for PCOS.

Gut Dysbiosis

Dysbiosis of Gut Microbiota (DOGMA) theory of PCOS can account for all three components of the syndrome an ovulation/ menstrual irregularity, hyper-androgenism and the development of multiple small ovarian cysts (Med Hypotheses. 2012).

Gestational Diabetes Mellitus (GDM)

Pregnancy per se, is a hyperinsulinemic state. Decreasing insulin sensitivity (increasing insulin resistance) with increasing gestational age is a physiological process designed to provide glucose to the fetus. Few who cannot cope up with this increasing insulin resistance manifest as case of GDM. Women who had a short stature are more likely to develop GDM than their counterparts (Diabetes Care, 2002). Women with GDM are at a greater risk of T2dm and cardiovascular disease in later life because of an increased susceptibility to hypertension, dyslipidemia and Metabolic Syndrome. The most important modifiable risk factor, being overweight or obese or severely obese increases the risk of developing GDM by a rate of 2.1, 3.6 and 8.6 respectively. PCOS is also a risk factor, although relevant evidence remains controversial. GDM is a significant risk factor for future maternal renal morbidity. The risk is more substantial for patients with recurrent episodes of GDM (JCEM, 2015).

Lifestyle Medicine

Counseling before pregnancy and multi-disciplinary management are important for good pregnancy outcomes. 90% women can manage their GDM with dietary changes and exercise.

Any diet needs to provide enough calorie for pregnancy with exemption for simple

carbohydrates. Carbohydrate restriction is no longer part of diabetes management. Both the quantity and the type (high/low glycemic index (GI)) or the source of carbohydrate (starch/sugar) found in foods influence postprandial glycaemia. The main goal of dietary modification is to avoid peaks in blood sugar level. This can be done by spreading carbohydrate intake over meals and snacks throughout the day. In using slow release carbohydrate sources known as the low GI diet since insulin resistance is highest in the morning, breakfast carbohydrate needs to be restricted more. Ingesting more fiber in foods with whole grains or fruits and vegetables can also reduce the risk of gestational diabetes. Carbohydrate must be consistently distributed throughout the day and incorporated in to each meal and snack to improve glycemic control and to minimize the risk of hypoglycemia and ketoacidosis. It is important to focus on carbohydrate counting and insulin dose adjustment to achieve Dose Adjusted for Normal Eating (DAFNE) specific to Type 1.

- Research suggests a possible benefit of breast feeding to reduce the risk of diabetes and related risk for both the mother and the child.
- Lose excess pounds prior to planning pregnancy. Weight loss is not recommended during pregnancy. It's important to focus on permanent changes to healthy eating habits.
- Although PCOS is thought to be mostly genetic in origin, your genes are strongly influenced by your environment. If you clean up your environment and reduce your exposure to chemicals, toxic metals and other contaminants, you will help your genes do the right thing (J Obstet Gynaecol Can, 2003).
- One trial has shown a reduction in the rate of GDM when women are randomized to probiotics early in pregnancy but more uncertain evidence of any effect on miscarriage/ IUFD/ stillbirth/ neonatal death (Barrett,2014).
- Keep active: Exercising before and during pregnancy can help protect you against developing GDM. Aim for 30 mins moderate activity on most days of your week. Take a brisk daily walk. Ride your bike, swim laps. If you cannot feat a single 30 mins work out in your busy day several shorter sessions can do much good. Get off the bus one stop before you reach your destination.

Dietary tips on carbohydrate consumption

- Start the day with some whole grains.
- Hot cereals porridges - when on a sick day diabetic diet choose on steel-cut oats or little millets.
- Cold cereals - Look for those that list whole wheat, whole oats, or other whole grain first on the ingredient list without added sugars. Check for gluten sensitivity when choosing on whole grains like wheat, oats and barley.
- Include a good source of fiber containing food with every meal or snack.
- Use whole grain breads for lunch or snacks. Check the label to make sure that whole wheat or another whole grain is the first ingredient listed.
- Eat less potatoes. Instead, try brown rice or less well-known grains like bulgur, millet, or hulled barley or quinoa.
- Include beans/legumes which are an excellent source of slowly digested carbohydrate as well as a great source of lean protein. Substitute for red meat.
- Strive to include a variety of fresh fruits and vegetables in meals every day. Avoid fruits along with meals to reduce the post prandial response.
- Fructose has been shown to produce a lower post prandial glucose response when it replaces sucrose or starch in the

diet than other carbohydrates, however it is thought to adversely affect plasma lipids. Therefore, the use of added fructose as a sweetening agent is not recommended. There is however no evidence to suggest avoiding naturally occurring fructose in fruits, vegetables and other foods.

- Prefer consumption of low glycemic index and low glycemic load fruits and vegetables
- Omega 3 and soluble fiber rich whole grams could be apt snacking options or half a handful of walnuts would also prove to be a cardiac and glycemic friendly snack

Summary

In the past couple of decades, evidence from prospective observational studies and clinical trials has converged to support the importance of individual nutrients, foods, and dietary patterns in the prevention and management of type 2 diabetes. The quality of dietary fats and carbohydrates consumed is more crucial than the quantity of these macronutrients.

Diets rich in whole grains, fruits, vegetables, legumes, nuts, moderate in alcohol consumption, and lower in refined grains, red/processed meats, and sugar-sweetened beverages have demonstrated to reduce diabetes risk and improve glycemic control and blood lipids in patients with diabetes.

Several healthful dietary patterns emphasizing the overall diet quality can be adapted to appropriate personal and cultural food preferences and calorie needs for weight control and diabetes prevention and management. Although considerable progress has been made in developing and implementing evidence-based nutrition recommendations in developed countries, concerted global efforts and policies are warranted to alleviate regional disparities.

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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.

Results: The results should be presented in the text, tables, and illustrations. Do not repeat in the text all the data in the tables and/or illustrations. Emphasize or summarize only important observations. Do not include discussion of your results and do not refer to observations of other

workers in this part of your text; these usually should be included in the Discussion.

Discussion: This should emphasize the new and important aspects of the study and conclusions that follow from them. Do not repeat in detail data given in the results section. Include in the discussion the implications of the findings and their limitations and relate briefly the observations to relevant studies.

Tables: Each table should be typed on a separate sheet and give a number and caption. Explain in footnotes all nonstandard abbreviations that are used in each Table. Cite each table in the text in consecutive order. If you use data from another published or unpublished source, obtain permission and acknowledge fully. The same data should not normally be presented in both tabular and graphical form.

Photographs should be of good quality and on glossy paper. Illustrations and graphs should be drawn on thick white paper with India ink. They should not be pasted on papers. The numbers should be marked at the back in pencil and the top should be marked by arrow. Legends should be typed on a separate sheet. Each should be brief but sufficiently descriptive to be complete by itself.

All the References given in the 'reference list' must be only those cited in the text. Reference should be arranged in the order of appearance in the text. Citation in the text should be as superscribed. Only those articles which have been read by the authors must be listed. The rest must be given as quotes. Each original article/review article requires at least 30 references whereas a short report or case report may suffice with 5. Also relevant Indian references on the subject must be quoted.

The pattern of References should be as follows.

Article from a Journal: List the first 3 authors with initials. The remaining authors may be given et al., e.g. Glogar D. H., Konar R. A., Muller J., et al; Fluorocarbons reduce myocardial ischaemic damage after coronary occlusion, Science. 1981; 211: 1439-41. (Note Punctuations)

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)

Authors' responsibility: The author is responsible for all statements in the work. Views expressed in the articles in interpreting conclusions from the data presented shall be the responsibility of the authors. The accuracy and completeness of the references is author's responsibility.

Acknowledgment of receipt

An acknowledgment, with a reference number for future inquiries, is despatched immediately (this does not apply to letters).

Authors should retain a copy of manuscript with them. Rejected articles are not returned.

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.
Website	:	www.apitnsc.org
Please send Application to	:	**Dr. V. Palaniyappen - Gen Secretary - API TNSC** Dr. V. Palaniyappen's Diabetes Specialities Center & Sri Sakthi Vinayagar Multispeciality Hospital, No.95, 95A, Near Bus Stand, Karur Main Road, Guziliamparai - 624703. Dindigul (Dt) Tamil Nadu Mob: 9965534483 / 9965534490 / 04551 234422 Email: drpalaniappen@yahoo.com / drpalaniappen1971@gmail.com

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
Membership No.

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
Tel : (022) 6666 3224 / 2491 2218 • Fax : (022) 2492 0263 • e-mail : api_ho@vsnl.com • www.apiindia.org

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.

LIFE ASSOCIATE MEMBERS : A person holding a post-graduate degree or diploma recognized by Medical Council of India in any branch of medical science who is not eligible for life membership shall be enrolled as a Life Associate Member. Life Associate Members shall have no voting rights, nor the rights to propose, second any one or contest for any office of the Governing Body. Life Associate Members of the Association are not eligible for any oration, lectureship or any other award of the Association.

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
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.

Available on API and JAPI websites: www. apiindia.org & www.japi.org

In uncontrolled diabetes patients with high PPBG

Azulix MV

Glimepiride 1 mg / 2 mg + Metformin SR 500 mg + Voglibose 0.2 mg / 0.3 mg

All Round Glycemic Control

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