PUSHPAGIRI COLLEGE OF PHARMACY

We care God cures

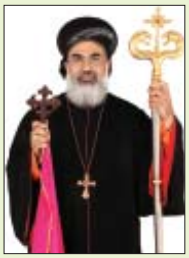
An official publication of the Department of Pharmacy Practice, Pushpagiri College of Pharmacy, Thiruvalla, Pathanamthitta (Dist.) Kerala
Ph: 0469-2645450 Email: pcppharmacypracticenewsletter@gmail.com

Vol. 2 Issue. 2
2016 November
2017 February

CLINICAL PHARMA PRACTICE NEWS ECHO

CONTENTS

Our Patron



H.G. Rev. Dr. Thomas Mar Koorilos
Metropolitan Archbishop of Tiruvalla
Catholic Archdiocese of Tiruvalla

Vision :

"We care.....
God cures....."

Mission :

To work towards a knowledge society with a life in abundance, through science and technology, improving health care of our immediate community, state, country and the world at large.

CEO's MESSAGE

I am extremely delighted to know that Pushpagiri College of Pharmacy is releasing the next issue of newsletter oriented on Clinical Pharmacy practice services. This newsletter is a product of an excellent team work with dedication, determination and discipline. Wishing you a grand success

Rev. Dr. Shaji Mathews Vazhayil
(Chairman & Chief Executive)

FROM THE DIRECTOR OF ACADEMICS

I take this opportunity to congratulate all the devoted hands who worked behind this endeavor and I wish all the success to Clinical Pharma Practice News Echo..

Rev. Dr. Mathew Mazhavancheril
Chief Advisor & Director- Academics & Research

FROM THE DIRECTOR

I am very happy to know that Pushpagiri College of pharmacy is bringing out a newsletter from the Department of Pharmacy Practice which highlights the clinical pharmacy oriented services.

Rev. Fr. Aby Vadakumthala
(Director Medicity)

FROM THE PRINCIPAL'S DESK

From the depth of my heart, I am very contended to declare that the Pharmacy practice department of our college is releasing the next issue of Clinical Pharma Practice News Echo. Heartfelt congratulations and appreciation for the team members behind this effort.

Prof. Dr. Mathew George,
Principal

FROM THE EDITORIAL ADVISORY BOARD

It's an immense pleasure to congratulate the editorial team members for releasing the next issue of Clinical Pharma Practice News Echo which reflects the Pharmacy practice activities of our institution in enhancing better pharmaceutical care to the society. Congratulations to all team members.

Prof. Dr. Lincy Joseph, HOD
(Department of Pharmaceutical Chemistry)

EDITOR'S DESK

We, the editorial committee have great privilege on releasing the next issue of our Clinical Pharmacy Practice newsletter of Pushpagiri College of Pharmacy. This newsletter covers the information related to the clinical practice activities and achievements in the department of Pharmacy Practice. Clinical pharmacy is concerned with the science and practice of rational medication use. The practice of clinical pharmacy embraces the philosophy of pharmaceutical care; it blends a caring orientation with specialized therapeutic knowledge, experience, and judgment for the purpose of ensuring optimal patient outcomes. The overall goal of clinical pharmacy activities is to promote the correct and appropriate use of medicinal products and devices. We extend our sincere thanks to all the devoted hands worked behind this venture. Please drop your valuable constructive suggestions and feedback at: pcppharmacypracticenewsletter@gmail.com.

BETA - LACTAMS

Dr. Sharma
Professor, Dept of General Medicine
Pushpagiri Medical College Hospital,
Thiruvalla

The Beta-lactam class of antibiotics consists of Penicillin, Cephalosporin, Carbapenem and Monobactams. The term ß-lactam refers to four-membered ß-lactam ring, which is their core structure. The differing side chains determine the spectrum of activity. They exert a bactericidal effect by inhibiting bacterial cell-wall synthesis. Their clinical efficacy is best correlated with the proportion of dosing interval during which drug levels remain above the MIC for the pathogenic organism.

Penicillin and Beta-Lactamase inhibitors:

Penicillin, the first ß-lactam, was discovered in 1928 by Alexander Fleming. Natural penicillin, such as penicillin G, are active against non ß-Lactamase producing gram-positive and gram-negative bacteria, some anaerobes, and some gram-negative cocci, and are used for penicillin-susceptible Streptococcal infections, Pneumococcal and Meningococcal meningitis, Enterococcal endocarditis, and Syphilis. The Anti-Staphyloccocal Penicilins, which have potent activity against Methicillin-susceptible *S.aureus* (MSSA), include Nafcillin, Oxacillin, Dicloxacillin, and Flucloxacillin. Aminopenicillins, such as Ampicillin and Amoxicillin, provide added coverage beyond penicillin against gram negative cocciÿsuch as *Haemophila influenza* and some Enterobacteriaceae, including *E. coli, Proteus mirabilis, Salmonella,* and *Shigella*, and are commonly used for otitis media, respiratory tract infections, intra-abdominal infections, endocarditis, meningitis, and urinary tract infections. The Anti-Pseudomonal Penicillins including Ticarcillin, Piperacillin, Azlocillin and Mezlocillin offer adequate anaerobic coverage with exception of Bacteroides species which produce ß-lactamases and are generally resistant. The rising prevalence of ß-lactamase producing bacteria has led to the increased use of ß-lactam/âlactamase inhibitor combinations such as Ampicillin-Sulbactam, Amoxicillin-Clavulanate, Ticarcillin-Clavulanate and Piperacillin-Tazobactam. The ß-lactam inhibitors themselves do not have antibacterial activity (with the exception of Sulbactam) but typically inhibit the S.aureus class A ß-lactamase, ß-lactamases of H. influenzae and Bacteroides species, and a number of plasmidencoded ß-lactamases. These combination agents are typically used when broader-spectrum coverage is needed, as in pneumonia, intra-abdominal infections and febrile neutropenia.

Cephalosporins: The cephalosporin drug class encompasses five generations determined by spectrum of antibacterial activity. The first generation (Cefazolin, Cefadroxil, Cephalexin) largely has activity against gram-positive bacteria, with some additional activity against *E. coli, P. mirabilis,* and *K. pneumonia*, and are commonly used for infections caused by MSSA and streptococci (eg: skin and soft tissue infections). The second generation (Cefamandole, Cefuroxime, Cefaclor, Cefprozil, Cefuroxime axetil, Cefoxitin, Cefotetan) has additional activity against *S. pneumonia, H. inluenzae* and *Moraxella catarrhalis*, and are used to treat community-acquired pneumonia as well as other mild or moderate infections, such as acute otitis media and sinusitis. The third-generation cephalosporins are characterized by greater potency against gram-negative bacilli and reduced potency against gram-positive cocci. These cephalosporins which include cefoperazoneÿ cefotaxime, ceftazidime, ceftriaxone, cefdinir, cefixime, and cefpodoxime are used for infections caused by Enterobacteriaceae, pulmonary infections in cystic fibrosis, febrile neutropenia, and meningitis. The fourth generation includes cefepime and cefpirome, broad-coverage agents that provide potent activity against both gram-negative bacilli, including *P. aeruginosa* and gram-positive cocci, and can be used in bacteremia, pneumonia, skin and soft tissue infections and urinary tract infections. Ceftaroline, a fifth-generation cephalosporin, differs from the other cephalosporins in its added activity against MRSA, which is resistant to all other ß-lactams. It is efficacious in community acquired pneumonia and skin infections.

Carbapenems: Carbapenems including doripenem, imipenem, meropenem and ertapenem, offer the most reliable coverage for strains containing ESBLs, having broad activity against gram-positive cocci, gram-negative bacilli, and anaerobes. None is active against MRSA, but all are active against MSSA, Streptococcus species, and Enterobacteriaceae. These are not active against Enterobacteriaceae containing carbapenemases.

Monobactams: Aztreonam is the sole monobactam. Its activity is limited to gram-negative bacteria (including *P. aeruginosa* and most other Enterobacteriaceae), and is inactivated by ESBLs and carbapenemases. The principal use for aztreonam is as an alternative to penicillins, cephalosporins, or carbapenems in patients with serious ß-lactam allergy. It is commonly used in febrile neutropenia and intra-abdominal infections, but not for meningitis as it does not penetrate the CSF.

Adverse Reactions to ß-Lactam drugs:

Gastrointestinal side effects, mainly diarrhea are common, but hypersensitivity reactions constitute the most common adverse effect of ß-lactams. The reactions' severity can range from rash to anaphylaxis. An individual with an accelerated 19E mediated reaction to one ß-lactam agent may still receive another agent within the class, with a dissimilar side chain and a low level of cross-reactivity. In cases of severe allergy, desensitization (a graded challenge) to the indicated ß-lactam, with close monitoring, may be warranted if other antibacterial options are not suitable. ß-Lactams can rarely cause serum sickness, Stevens-Johnson syndrome, nephropathy, hematologic reactions, and neurotoxicity. Neutropenia (at high doses or prolonged use) and interstitial nephritis caused by ß-lactams generally resolve upon discontinuation of the drug. Imipenem and Cefipime are associated with an increased risk of seizure.

In general beta lactam antibacterial agents are very commonly used drugs which cater to many infections affecting human beings.

FDA APPROVED DRUGS 2016 (FROM MAY)

SYSTEMS	GENERIC NAME	BRAND NAME	INDICATION
Cardiology	Byvalson	Nebivolol and Valsartan	Hypertension
Dermatology	Ameluz	Aminolevulinic acid hydrochloride	Actinic keratoses
Endocrinology	Adlyxin	Lixisenatide	Type II diabetes
Hematology	Afstyla Opdivo	Antihemophilic Factor (Recombinant), Single Chain) Nivolumab	Hemophillia A Hodgkin lymphoma
Hepatology (Liver, Pancreatic, Gall Bladder)	Ocaliva	Obeticholic acid	Primary biliary cholangitis
Immunology	Epclusa Vaxchora	Sofosbuvir and Velpatasvir Cholera Vaccine, Live, Oral	Hepatitis C Active immunization against Cholera
Musculoskeletal	Zinbryta	Daclizumab	Relapsing multiple sclerosis
Nephrology	Lenvima Rayaldee	Lenvatinib Calcifediol	Renal cell carcinoma Secondary hyperparathyroidism
Neurology	Nuplazid Troxyca ER	Pimavanserin Oxycodone + Naltrexone	Hallucinations and delusions associated with Parkinson's disease psychosis. Severe pain
Oncology	Keytruda Sustol Tecentriq	Pembrolizumab Granisetron Atezolizumab	Head and neck squamous cell cancer Chemotherapy-induced nausea and vomiting Urothelial carcinoma
Ophthalmology	Humira Xiidra	Adalimumab Lifitegrast	Uveitis. Dry eye disease
Psychiatry/ Psychology	Nuplazid	Pimavanserin	Hallucinations and delusions associated with Parkinson's disease, psychosis.
Urology	Tecentriq	Azolizumab	Urothelial carcinoma

HYPERPHOSPHATEMIA IN CKD PATIENTS

Ms. Priya G. Udayan
Asst. Prof, Dept. of Pharmacy Practice
Pushpagiri College of Pharmacy

Hyperphosphatemia is defined as an abnormally high serum phosphate concentration of >1.46 mmol/L. It is principally observed in patients with reduced kidney function. However, other potential causes also exist. These mainly include hypoparathyroidism, pseudo-hyperphosphatemia, excessive intake of phosphate, excessive cellular injury (for example rhabdomyolysis and tumour lysis syndrome), intracellular shifts (metabolic or respiratory acidosis) and vitamin D toxicity.

Pathogenesis, consequences and importance of controlling hyperphosphatemia

In chronic kidney disease, hyperphosphatemia is prevented until the later stages by two major forces that control phosphate homeostasis, parathyroid hormone (PTH) and fibroblast growth factor-23 (FGF-23). In the early stages of CKD, phosphorus retention stimulates FGF-23 and PTH secretion, which in turn suppress renal phosphate reabsorption and augment renal phosphate excretion. FGF-23 also suppresses 1, 25- dihydroxyvitamin D (1,25D) production which inturn causes a reduction of calcium absorption and stimulation of PTH secretion leading to secondary hyperparathyroidism (SHPT). PTH increases phosphate loss through the kidney by reducing the number of NaPi co-transporters. FGF-23 suppresses PTH secretion in normal parathyroids, and resistance to the effect of FGF-23 appears as kidney function declines because of decreased Klotho expression in the parathyroid and kidney.These compensatory mechanisms attempt to normalise serum phosphate and calcium concentrations in CKD patients.

However, as GFR continues to decline and falls below 25 ml/min, the renal phosphate reaches its maximum and excess dietary phosphate accumulates leading to persistent hyperphosphatemia. In addition, SHPT and low circulating 1,25D stimulate bone resorption to a greater extent than bone formation leading to the loss of bone mineral density (BMD) and further contributing to hyperphosphatemia in CKD patients. If the skeletal system does not accommodate the increased serum phosphate concentration, serum phosphate can interact with calcium to precipitate calcium phosphate salts (hydroxyapatite) in non-skeletal tissues. Calcification generally occurs in the blood vessels, heart valves, myocardium, and other soft tissues. Cardiovascular calcification is probably the main reason for the high prevalence of cardiovascular diseases (CVD) in CKD patients. Calcification risk is usually assessed by the Calcium–Phosphate product (Ca • P).A value of 5–6 mmol/L (70 mg/dl) is usually regarded as the 'threshold' value above which calcification is more likely.

Management of hyperphosphatemia in CKD patients

In clinical practice, the management of hyperphosphatemia is focused on controlling factors that are responsible for the intake and removal of phosphate from the body.There are three main strategies for correcting hyperphosphatemia:

I. Diet: restricting dietary phosphate intake

Reducing the daily phosphate intake in diet can be challenging as it is usually incompatible with the recommended daily protein intake of 1.0–1.2 g/kg/day. In addition, the required intensive patient education, the complexity of dietary tables and booklets as well as the substantial variability of phosphate contents in food from the same category are added challenges for dietary phosphate control.This makes diet control alone insufficient, and hence combination with other strategies, can assist in management of hyperphosphatemia.

Enhancing elimination: removing phosphate with adequate dialysis

Dialysis techniques are used in removing phosphates but they are usually ineffective in removing excess phosphate to the degree of normalising phosphate concentration since the rate of transfer of phosphate from the intracellular pool to the extracellular pool is relatively slow.

II. Minimising phosphate absorption: reducing intestinal absorption (phosphate binders)

Phosphate binders work by binding dietary phosphate and forming insoluble complexes that are excreted by the gut.

The most commonly utilised phosphate binders are:.

a. Calcium-based phosphate binders (Calcium Carbonate and Calcium Acetate).
b. Non-absorbable polymers (Sevelamer).
c. Heavy metal salts (Lanthanum Carbonate and Aluminium Hydroxide).

Hyperphosphatemia in CKD patients is an important complication of reduced kidney function. It is associated with severe clinical consequences including cardiovascular tissues calcification, bone diseases, and secondary hyperparathyroidism leading to increased cardiovascular diseases and mortality rates. Giving the significant clinical consequences of hyperphosphatemia in CKD it is recommended to manage serum phosphate, serum calcium, Ca • P product and PTH to strict and specific targets to reduce the potential impact of such consequences, improve patient's quality of life and probably improve survival.

REPORT ON DIABETES MEDICAL CAMP, 2016 AT TIRUVALLA RAILWAY STATION

A medical camp- **CLINICAL MADHUMEH** was conducted at Thiruvalla railway station on 21st November, 2016, Monday, as a part of Pharmacy Week celebrations, under the Dept. of Pharmacy Practice, by the 5th year Pharm D and 2nd year M.Pharm students. The camp was officially inaugurated by Mr K.V.Varghese, Muncipal Chairperson of Thiruvalla & the presence of Mrs. Saramma Francis, Manjadi councillor, Rev. Fr. Mathew Vadakkekuttu, Director, Medicity Campus, Prof. Dr. Mathew George, Principal, Pushpagiri College of Pharmacy, and Prof. Dr. Lincy Joseph, H.O.D & P.G incharge made the inaugural function event more splendid.

The camp included arrangements made for free blood glucose level checkup using glucometers sponsored by the Accucheck and Abott companies. Free B.P. monitoring & individualized counselling regarding the disease condition and medications were provided to about 906 participants.

CLINICAL ACTIVITIES

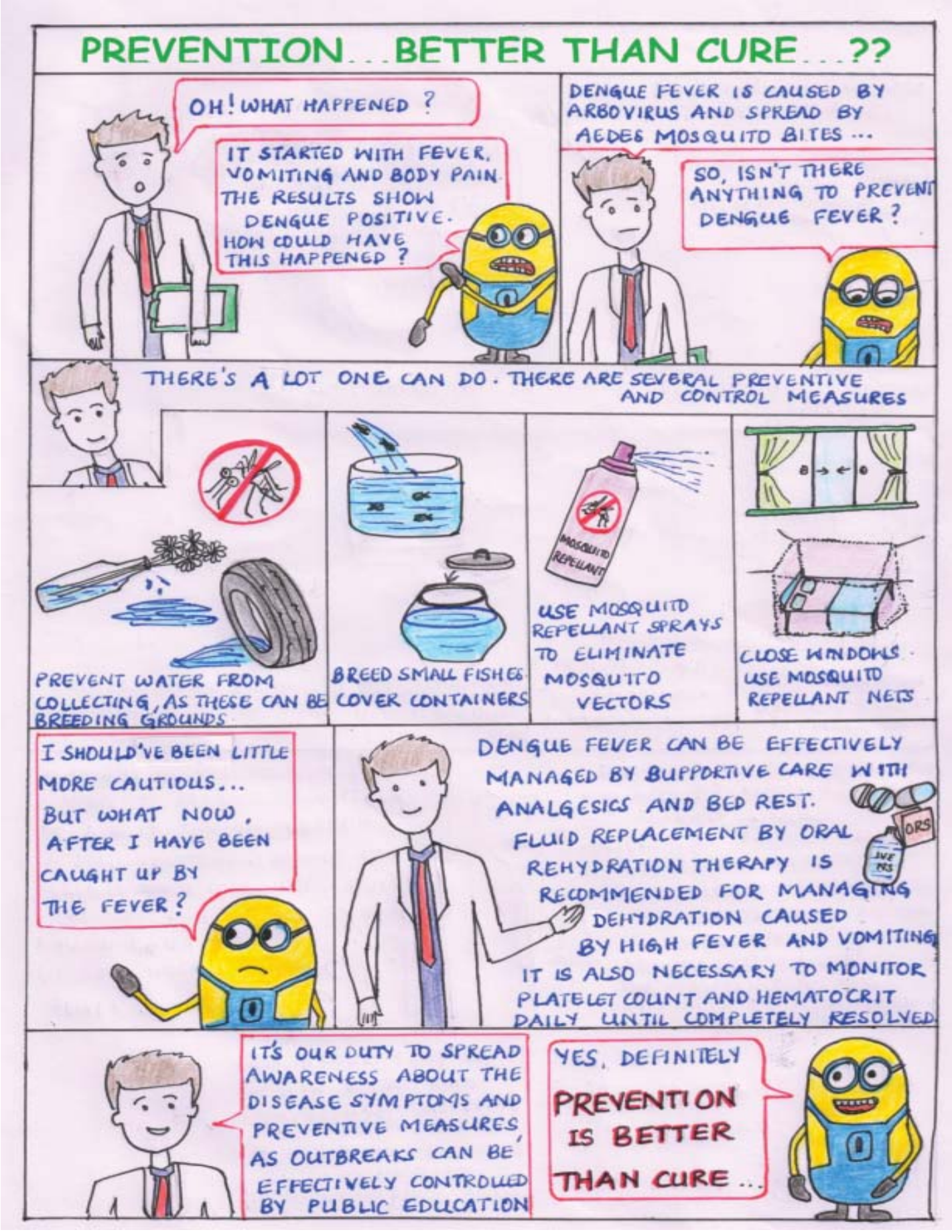
ACTIVITIES	NO.
ADR Reports	14
Pharmacist interventions	476
Patient Counselling	533
Drug Information Query	129

NAAC Admin level celebrations with H.G. Most Rev. Dr.Thomas Mar Koorilos,
Honourable Revenue Minister, Sri. E. Chandrasekharan & Shri. B Rajan

Basic Life Support (BLS) Course by **Indian institute of Emergency Medical Service** with
American Heart Association conducted on march 20th 2017 at Pushpagiri college of Pharmacy

One day seminar on **"PROFESSIONAL ROUTE OF PHARMACEUTICAL SCIENCES"**
by **Dr. Mahesh D Burande**, EX-APTI President on 8th December 2016 and was inaugurated by Shri. Harishankar, IPS.


CLOTTING FACTORY
So, is everyone ready for today?!!
ermmm.... No !!
Yes!!
Yes!
Yes!
Why?!!
Vit-k is not here yet!!
Where is he?!
He was with Mr. Warfarin since yesterday!
Mr Warfarin, if you could send Vit-k out here, it could be great help!!
No, I am not sending him anywhere!
Vit K
WARFARIN
But our factory cannot run without him!!
Then, shut down the factory as long as I am here!!
WARFARIN
Hey, so no work today!!
Yeah!! That's a good news!
Ca2+


PREVENTION...BETTER THAN CURE...??
OH! WHAT HAPPENED ?
IT STARTED WITH FEVER, VOMITING AND BODY PAIN. THE RESULTS SHOW DENGUE POSITIVE. HOW COULD HAVE THIS HAPPENED ?
DENGUE FEVER IS CAUSED BY ARBOVIRUS AND SPREAD BY AEDES MOSQUITO BITES ...
SO, ISN'T THERE ANYTHING TO PREVENT DENGUE FEVER ?
THERE'S A LOT ONE CAN DO. THERE ARE SEVERAL PREVENTIVE AND CONTROL MEASURES
PREVENT WATER FROM COLLECTING, AS THESE CAN BE BREEDING GROUNDS
BREED SMALL FISHES COVER CONTAINERS
MOSQUITO REPELLANT
USE MOSQUITO REPELLANT SPRAYS TO ELIMINATE MOSQUITO VECTORS
CLOSE WINDOWS USE MOSQUITO REPELLANT NETS
I SHOULD'VE BEEN LITTLE MORE CAUTIOUS... BUT WHAT NOW, AFTER I HAVE BEEN CAUGHT UP BY THE FEVER ?
DENGUE FEVER CAN BE EFFECTIVELY MANAGED BY SUPPORTIVE CARE WITH ANALGESICS AND BED REST. FLUID REPLACEMENT BY ORAL REHYDRATION THERAPY IS RECOMMENDED FOR MANAGING DEHYDRATION CAUSED BY HIGH FEVER AND VOMITING. IT IS ALSO NECESSARY TO MONITOR PLATELET COUNT AND HEMATOCRIT DAILY UNTIL COMPLETELY RESOLVED
ORS
IT'S OUR DUTY TO SPREAD AWARENESS ABOUT THE DISEASE SYMPTOMS AND PREVENTIVE MEASURES, AS OUTBREAKS CAN BE EFFECTIVELY CONTROLLED BY PUBLIC EDUCATION
YES, DEFINITELY PREVENTION IS BETTER THAN CURE ...