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First Kerala Pharmaceutical Congress -2023

Theme : A self reliant Pharma Industry for the future of Pharmacy in Kerala

Venue: St. James College of Pharmaceutical Sciences, Chalakudy, Thrissur

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Prof. Dr. **Boby Johns G**

From The Chief Editor's Desk

It gives me immense pleasure to present before you this issue of Pharmline, Vol.22, January 2023.

On behalf of our editorial board, I would like to extend our gratitude to our readers, contributors, authors, editors, and anonymous reviewers, all of whom have voluntarily contributed to the success of the journal and to its mission of enhancing the quality of care and research through publication in the field of pharmacy. We are releasing our publication tri annually with a specific emphasis on quality, novelty and better outcomes of research. I am equally elated to inform you all that Pharmline has been contributing enormously to increase the quality of research and teaching in the field of pharmacy by releasing its issues periodically since 1981. In recent days, a tremendous amount of effort has gone into the establishment of this journal.

The mission of Pharmline is to rapidly disseminate high-quality research publications, reflection of ideas of the student community along with news and activities of KPGA. Only breakthroughs in pharmacy can help us face the problems of the 21st century and capitalize on the opportunities that lie ahead. We appreciate submissions that can demonstrate near-term practical utility, especially those that adopt a multidisciplinary/convergent approach, as many real-world situations are inherently complicated.

To maintain the longevity of a successful pharmaceutical publication, we also solicit contributions from the scientific community. Authors, reviewers and guest editors are always welcome. We also welcome comments and recommendations that could improve the quality of the journal.

We hope you will find Pharmline more informative in the future endeavor. Thank you.

Prof. Dr. **Boby Johns G**
Chief Editor, Pharmline

THE PRESIDENT SPEAKS

The seniors, beginners and other members of our Association, through their dedication and hard work have taken vital part to build in the minds of society the fact that we are an integral part of healthcare. In continuation of our ongoing efforts, we are organizing the Kerala Pharmaceutical Congress, on 25 th and 26 th February 2023 will be held at St. James College of Pharmaceutical Sciences, Chalakudy - the first of its kind in Kerala. The theme of the congress-A Self Reliant Pharma Industry for the Future of Pharmacy in Kerala is highly apt one in the present scenario.

It is a matter of great pride that Indian Pharma industry is acknowledged as third largest in the world and India supplies medicines to over 200 countries in the world. Kerala is a consumer state with respect to medicines and allied consumables. The consumption of medicines in Kerala is worth about Rs.15,000 crores as against a domestic production of Rs. 200 crores; i.e., 98% of medicines required in the state is imported from other states, though there is potential for immense resources, highly qualified man power and huge opportunities in the global market.

I am sure that This two-day congress will be a platform bringing together Government officials, experts from industry, academicians and students, eminent scholars, research scientists and all other segments of pharmaceutical sector to exchange innovative ideas and create practical plans, helping us to move a step closer to realizing the dream of Kerala-the Pharmacity in Kerala the concept of the Association has done wonderful beginning to make it a reality. . We hope that this congress will create a positive vibe for investors to establish industries in Kerala. The conference will also help attendees stay up-to-date with advancements and innovations in the Pharma sector. The scientific gathering navigates a way for a unique amalgamation of advancements in pharmaceutical sciences, pharmacological and pharmacy practice. There will be key note addresses, lectures, exhibitions, oral and e-poster presentations, in seven categories of pharmaceutical sciences. The presence of National leaders of prominent associations of India under the same platform and panel discussion on the industry will make the event colorful further. This congress will provide a beneficial opportunity to learn about the current scenario by offering exemplary practices, services, and potential employment opportunities for students, academicians, and individuals from various backgrounds.

I take this opportunity to sincerely wish and thank the entire organizing committee team members for their relentless efforts and the entire pharma fraternity who have directly or indirectly to make this congress a resounding success.

I also appreciate and thank the editorial board, Pharmline for their effort to make the projects and programmes organized by KPGA to be great success.

Dr. **PK Sreekumar**,
President- KPGA

AN INSIGHT INTO POST - COVID SYNDROME

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ABSTRACT

Severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) infection is associated with a range of persistent symptoms impacting everyday functioning, known as post-COVID-19 condition/ syndrome or long COVID. Post-COVID conditions can include a wide range of ongoing health problems; these conditions can last weeks, months, or years. Post-COVID conditions are found more often in people who had severe COVID-19 illness, but anyone who has been infected with the virus that causes COVID-19 can experience post-COVID conditions. People not vaccinated against COVID-19 and who become infected may have a higher risk of developing post-COVID conditions compared to people previously vaccinated.

Keywords: Cystic fibrosis, CFTR, Chloride ion channels, Sodium epithelial channels.

Introduction

More than 2 years after the declaration of the coronavirus disease 2019 (COVID-19) pandemic, the world continues to face its devastating impact, not only on morbidity, mortality, and healthcare services, but also its tremendous societal and economic consequences, globally[1]. Most patients with COVID-19 return to baseline after acute infection with SARS-CoV-2, but a proportion report ongoing health issue. Post-COVID syndrome is increasingly recognized as a new clinical entity in the context of SARS-CoV-2 infection[2]. Symptoms persisting for more than three weeks after the diagnosis of COVID-19 characterize the post-COVID syndrome. Its incidence ranges from 10% to 35%, however, rates as high as 85% have been reported among patients with a history of hospitalization[3].

The UK National Institute for Health and Care Excellence (NICE) makes a distinction between disease occurring from 4 to 12 weeks after infection (ongoing symptomatic COVID-19) and symptoms persisting beyond 12 weeks (post-acute COVID-19 syndrome)[4,5]. The World Health Organization (WHO) defines it as a condition characterized by symptoms impacting everyday life, such as fatigue, shortness of breath and cognitive dysfunction, which occur after a history of probable or confirmed SARS-CoV-2 infection6. Symptoms usually occur 3 months from the onset of acute COVID-19 symptoms, last for at least 2 months and cannot be explained by an alternative diagnosis. Previous studies suggested that higher risk of developing long COVID was observed with a gradient increase in age, female sex, hospital admission during acute COVID-19 (including the need for oxygen therapy), symptom load (including dyspnea at presentation and chest pain), abnormal auscultation findings and the presence of comorbidities such as asthma

Symptoms

People with post-COVID conditions can have a wide range of symptoms that can last weeks, months, or even years after infection. Sometimes the symptoms can even go away or come back again. Post-COVID conditions may not affect everyone the same way. People with post-COVID conditions may experience health problems from different types and combinations of symptoms happening over different lengths of time[7]. People who experience post-COVID conditions most commonly report:

1. General symptoms
- Tiredness or fatigue that interferes with daily life
- Symptoms that get worse after physical or mental effort (also known as “post-exertional malaise”)
- Fever

1. Respiratory and heart symptoms
- Difficulty breathing or shortness of breath
- Cough
- Chest pain
- Fast-beating or pounding heart (also known as heart palpitations)

2. Neurological symptoms
   - Difficulty thinking or concentrating (sometimes referred to as “brain fog”)
   - Headache
   - Sleep problems
   - Dizziness when you stand up (lightheadedness)
   - Change in smell or taste
   - Depression or anxiety

1. Digestive symptoms
   - Diarrhea
   - Stomach pain

2. Other symptoms
   - Joint or muscle pain
   - Rash
   - Changes in menstrual cycles

Some people, especially those who had severe COVID-19, experience multi-organ effects or autoimmune conditions with symptoms lasting weeks, months, or even years after COVID-19 illness. Multi-organ effects can involve many body systems, including the heart, lung, kidney, skin, and brain. As a result of these effects, people who have had COVID-19 may be more likely to develop new health conditions such as diabetes, heart conditions, blood clots, or neurological conditions compared with people who have not had COVID-19 [8].

Classifications Of Post-Covid Syndrome

According to Fernández-de-las-Penas et al,

Transition Phase: Symptoms potentially associated with acute COVID-19: symptoms up to 4–5 weeks

Phase 1: Acute post-COVID symptoms: symptoms from week 5 to week 12

Phase 2: Long post-COVID symptoms: symptoms from week 12 to week 24

Phase 3: Persistent post-COVID symptoms: symptoms lasting more than 24 weeks.

Mechanism Of Covid-19 Infection

COVID-19 is a multi-system infection. The cell surface angiotensin-converting enzyme 2 (ACE2) receptor, which is abundant in cells of most organs, is the main target for SARS-CoV-2 binding and infection. A monocyte-macrophage, CD4 and CD8 cellular response, and a controlled inflammatory response occur, which results in an uncomplicated recovery of most patients. A SARS-CoV-2 immune dysregulation, associated with elevated levels of cytokines interleukin-1β (IL-1β), IL-6, IL-2, and IL-10 (“cytokine storm”) and profound inflammation, is found in patients with severe life - threatening illnesses. The pathogenesis of post-COVID syndrome remains largely unknown. Evidence suggests that prolonged inflammation has a key role in the pathogenesis of most post-COVID manifestations.

These long-term symptoms are not only present in severe COVID-19, but also in mild and moderate patients. In addition, recent preliminary data also underlined the presence of long-term COVID-19 symptoms on children and adolescents. Some clinical studies and survey questionnaires also highlighted a potential high-risk factor for long-term COVID-19 in the female gender; women's risk of developing long-term COVID-19 seems to be double that of men among patients aged between 40 and 50. After the age of 60 the risk level of long-term COVID between male and female should become similar. This pattern appears to be like that of autoimmune diseases that are more common in female through menopause to become similar between male and female after age 60.

Conclusion

Necessary active and future research include the identification and characterization of key clinical, serological, imaging and epidemiologic features of COVID-19 in the acute, sub-acute and chronic phases of disease, which will help us to better understand the natural history and pathophysiology of this new disease entity. Active and future clinical studies, including prospective cohorts and clinical trials, along with frequent review of emerging evidence by working groups and task forces, are required to develop database and informing clinical practice in this area.

Conflict of interest

The author declared no conflict of interest with respect to the authorship, research or publication of the article.

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PREPARATION AND EVALUATION OF HAIR GEL FROM PIPER BETLE LEAVES EXTRACT

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ABSTRACT

Aim of our present study was to formulate the Hair gel from Piper betel leaves Extract. We extract Piper betel leaves and study the formulation parameters. Piper betel leaves was extracted by aqueous Extraction method and study of the parameters like homogeneity, transparency, PH, viscosity and spreadability.

Key words: Piper betel, Digital pH meter and Brookfield Viscometer.

Introduction

The betel (Piper betle) is a vine of the family Piperaceae, which encloses Pepper and Kava. The betel plant is native to Southeast Asia[1]. It is an evergreen, dioecious, perennial, with glossy heart-shaped leaves and white catkins. Betel plants are sophisticated for their leaves which are most frequently used as flavouring in chewing[2]. In India and Sri Lanka, a sheaf of betel leaves is conventionally obtainable as a smudge of admiration and auspicious beginnings in traditional Indian culture. It may likewise be reused in cooking;Chemistry of betel leaf varies geographically and is mostly chavibetol, Eugenol, Isoeugenol, and Germacene. Leaves also contain eugenol, chavicol, hydroxychavicol and caryophyllene.Stems contain phytosterols , alkaloids , lignin, dehydropipernonaline, piperolein-B, Bornyl cis-4-Hydroxycinnamate and Bornyl p-Coumarate.Roots contain aristololactam, 4-allyl resorcinol and a diketosteroid stigmast-4-n-3,6-dione[3].Essential oil consisted of 50 different compounds, of which major components are eugenol, caryophyllene, terpinoleneterpinene, cadinene and 3 carene.

Materials and methods

Materials: The leaves of *Piper betle* was collected from the nearby areas from Chalakudy which was identified and authenticated from, Department of Botany and Research, St. Thomas college, Thrissur. Leaves were washed with water and dried under sunshade, made into powdered, stored into container for further studies. Betel leaves were extracted using water as solvent which was carried out using heating mantle and beaker of 1000ml then washed with water, dried and powdered. About 30g leaves were washed with water and dried under sunshade, made into powdered, stored into container for further studies[4].Betel leaves were extracted using water as solvent which was carried out using heating mantle and beaker of 1000ml then washed with water, dried and powdered. About 30 g of powdered leaves were boiled with 900 ml of water for 7 hours. The extract was filtered through a muslin cloth and dried. Measured quantity of Methyl paraben, extract, rosé oil, PVP and Polyethylene glycol were dissolved in water. Glycerin was added under continuous stirring and Triethanolamine was added drop wise while stirring till uniform gel was formed. Six different herbal hair gel formulations were prepared with carbopol gel base. Homogeneity factor was identified by the presence of any flocculates or aggregates. Transparency was checked visually. Ph meter was used to determine viscosity For pH, 1g of gel was dissolved in 100 ml of dissolved water, stored for 2 hours and pH was noted then average values were calculated[5],[6]. Brookfield viscometer was used to determine viscosity. Gel was filled and the RPM of spindle was adjusted to 2.5 then the viscosities of the formulations were recorded[7]. 0.5g of gel was placed between two glass side for 10 minutes and calculated by the formula S = (M X L)/T.

Result & Discussions

Physical Appearance

The Physical appearance was visually checked for colour, odour and Smoothness[8] and the results are given in Table 1 below.

Formulations F1 to F3 showed yellow colour, whereas F4 to F6 showed light brown to brown.Formulations F1 to F6 were pleasant in odour. Regarding smoothness, the formulation F1 was smooth , whereas F6 was not smooth.

Table 1

Parameters	F1	F2	F3	F4	F5	F6
Colour	Light Yellow	Yellow	Yellow	Light Brown	Brown	Brown
Odour	Pleasant	Pleasant	Pleasant	Pleasant	Pleasant	Pleasant
Smoothness	Very Smooth	Smooth	Smooth	Slightly Smooth	Slightly Smooth	Not Smooth

Homogeneity

Homogeneity factor was identified by the presence of any flocculates or aggregates and the results are given in the following table 2.

Table 2

Formulation	Results
F1	Excellent
F2	Excellent
F3	Good
F4	Average
F5	Poor
F6	Very Poor

Transparency

The Transparency was visually checked and the results are given in table 3.

Table 3

Formulation	Results
F1	Translucent
F2	Translucent
F3	Translucent
F4	Translucent
F5	Translucent
F6	Translucent

Measurement of pH

Table 4

Formulation	pH
F1	5.71±0.12
F2	4.52±0.21
F3	4.03±0.08
F4	3.75±0.17
F5	3.36±0.10
F6	3.12±0.05

Viscosity determination

Table 5

Formulation	Viscosity(cps)
F1	3220
F2	3661
F3	4121
F4	4725
F5	5278
F6	5821

Spredability

Table 5

Formulation	Spredability(gcm/sec)
F1	1.02
F2	0.77
F3	0.55
F4	0.46
F5	0.39
F6	0.28

Results and Discussion

The physical appearance was visually checked for colour, odour and smoothness. The colour of formulations F1 to F3 showed yellow colour whereas F4 to F6 showed light brown to brown. About odour, the formulations F1 to F6 were pleasant in odour. Regarding smoothness,the formuln. F1 was smooth whereas F6 was not smooth. By visual inspection appearance of flocculates in F1 and F2. All the hair gel formulations were Translucent. The PH of the herbal gel formulations F2, F3, F4 ranged between 3.75 to 4.52 that suited the hair, indicating the compatibility of the herbal gel formulations with the hair. Formulated gels showed increased viscosity as the concentration of the gelling agent was increased. Spredability of the formulated gel was decreased as the concentration of gelling agent increased.

Conclusion

In our study herbal hair gel was prepared from Piper beetle leaves extract using 6 formulations F1, F2, F3, F4, F5, and F6; then various parameters were evaluated. The Formulation F2 has shown good spreadability, homogeneity, pH and viscosity. Piper betle leaves extract hair gel improved the hair growth by maintaining the collagen in the body or the connective tissues of hair follicles[9,10]. It also protects the scalp cells in the blood vessels to promote the healthy hair growth and also prevents the premature graying of hair.

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Preparation and evaluation of hair gel from Piper betel Leaves extract

PHYTOCHEMICAL SCREENING AND ANTIOXIDANT PROPERTY OF PIPER BETLE LEAVES EXTRACT

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ABSTRACT

Aim of our present study was to formulate the Hair gel from Piper betle leaves Extract. We extract Piper betle leaves and study the formulation parameters. Piper betle leaves was extracted by aqueous Extraction method and study of antioxidant studies using DPPH assay method.

Key words: Piper betle, DPPH assay, spectrophotometer.

Introduction

The betel (*Piper betle*) is a vine of the family Piperaceae, which encloses Pepper and Kava[1]. The betel plant is native to Southeast Asia. It is an evergreen, dioecious, perennial, with glossy heart-shaped leaves and white catkins. Betel plants are sophisticated for their leaves which are most frequently used as flavouring in chewing[2]. In India and Sri Lanka, a sheaf of betel leaves is conventionally obtainable as a smudge of admiration and auspicious beginnings in traditional Indian culture. It may likewise be reused in cooking;Chemistry of betel leaf varies geographically and is mostly chavibetol, Eugenol, Isoeugenol, and Germacene. Leaves also contain eugenol, chavicol, hydroxychavicol and caryophyllene. Stems contain phytosterols , alkaloids , lignin, dehydropipernonaline, piperolein-B, Bornyl cis-4-Hydroxycinnamate and Bornyl p-Coumarate.Roots contain aristololactam, 4-allyl resorcinol and a diketosteroid stigmast-4-en-3,6-dione.Essential oil consisted of 50 different compounds, of which major components are eugenol, caryophyllene, terpinoleneterpinene, cadinene and 3 carene[3].

Materials And Methods

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The Percentage radical scavenging activity can be calculated using the formula % RSA=Abs of control-Abs of sample/ Abs of control [6,7]

Phytoconstituents	Tests	Observation
Alkaloids	Hagers test: 2ml Extract+ few drops of Hagers Reagent	Yellow Precipitate
Flavonoids	Ammonia test: Filter paper dipped in alcoholic solution of drug was exposed to ammonia vapour.	Formation of yellow spots on filter paper.
Carbohydrates	Molisch test: 2ml extract+ 10 ml of water +2 drops of ethanoic alpha napthol+ 2ml of conc sulphuric acid.	Reddish violet ring at the junction
Glycosides	Libbermans test: 2ml extract +2ml chloroform acetic acid	Violet to blue to green colour
Tannins	Braymer test: 2ml extract 2ml water 2-3 drops ferric chloride	Green precipitate
Steroids	Salkowski test: 2ml extract +2ml chloroform+ 2ml of conc sulphuric acid.	Reddish brown ring at the junction
Proteins	Ninhydrin test: 1ml extract+ Ninhydrin reagent	Violet precipitate
Saponins	Foam test: 5ml extract +5ml water heat	Froth appears
Phenols	Ferric chloride test: extracts+3-4 drops of ferric chloride.	Formation of bluish black colour.

Result & discussions: Antioxidant study

Sample	Percentage (% RSA)
F1	43.56%
F2	41.23%
F3	45.52%
F4	40.19%
F5	41.58%
F6	48.21%

In DPPH assay Piper betle leaf extract and formulations showed percentage RSA ranging from 41.23% to 48.21% indicating good radical scavenging activity.

Conclusion

Free radicals damage contributes to the etiology of many chronic health problems such as cardiovascular and inflammatory disease, cataract, and cancer[8]. Antioxidants prevent free radical induced tissue damage by preventing the formation of radicals, scavenging them, or by promoting their decomposition[9]. Synthetic antioxidants are recently reported to be dangerous to human health. Thus, the search for effective, nontoxic natural compounds with antioxidative activity has been intensified in recent years. In addition to endogenous antioxidant defense systems, consumption of dietary and plant-derived antioxidants appears to be a suitable alternative[10]. Dietary and other components of plants form a major source of antioxidants. The traditional Indian diet, spices, and medicinal plants are rich sources of natural antioxidants; higher intake of foods with functional attributes including high level of antioxidants in antioxidants in functional foods is one strategy that is gaining importance[11]. From the above study it was confirmed that leaves of Piper betel has very good antioxidant activity because of eugenol present in it.

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Phytochemical screening and anti oxidant property of Piper betle leaves extract

TRIAL OF PIMAVANSERIN IN DEMENTIA RELATED PSYCHOSIS

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ABSTRACT

Patients with dementia due to neurodegenerative disease can have dementia-related psychosis. The effects of the oral 5-HT2A inverse agonist and antagonist pimavanserin on psychosis related to various causes of dementia are not clear. Pimavanserin is a serotonin-receptor modulator that acts primarily as a selective 5-hydroxytryptamine receptor subtype 2A (5-HT2A) inverse agonist and antagonist. Its major active N-demethylated metabolite AC-279 have demonstrated a time to maximum plasma concentration of six hours. The recommended dose of pimavanserin is 34 mg taken orally as two 17-mg tablets. Longer and larger trials are required to determine the effects of pimavanserin in dementia-related psychosis

Key Words: Dementia, psychosis, pimavanserin, Parkinsons' disease

Introduction

The most common causes of dementia -Alzheimer's disease, dementia with Lewy bodies, Parkinson's disease dementia, vascular dementia, and frontotemporal dementia may be associated with hallucinations and delusions. This syndrome of dementia-related psychosis causes behavioural disturbances, increased caregiver burden, decreased quality of life, and more rapid cognitive decline. Typical and atypical antipsychotic agents are used off-label to manage psychotic symptoms, regardless of underlying dementia subtype, and are used cautiously in patients with dementia with Lewy bodies owing to the risk of worsening parkinsonism and other side effects. Antipsychotics may have modest short-term efficacy for dementia-related psychosis but may be associated with worsening cognition, extrapyramidal effects, sedation, falls, and metabolic abnormalities. Labels for these drugs include a warning for increased risk of death among elderly patients. This situation has contributed to clinical guidelines that focus on minimizing the use of antipsychotics, discontinuing treatment when improvement is not observed, and reassessing continued use after initial improvement[1].

Pimavanserin is a serotonin-receptor modulator that acts primarily as a selective 5-hydroxytryptamine receptor subtype 2A (5-HT2A) inverse agonist and antagonist, with lesser activity at 5-HT2C and no appreciable activity at other receptors in vitro. This profile is different from those of conventional antipsychotics, which bind to D2 dopamine receptors and have varying activity at other receptors, including at histaminergic and muscarinic receptors[2].

Description

Pimavanserin, an atypical anti psychotic, is present in Nuplazid as pimavanserin tartrate salt.Pimavanserin tartrate is freely soluble in water. Its molecular formula is (C25H34FN3O2)2 • C4H6O6, and its molecular weight is 1005.20 (tartrate salt). The molecular formula of pimavanserin free base is C25H34FN3O2, and its molecular weight is 427.55. Pimavanserin is a round, white to off-white, immediate-release, film-coated, once daily oral tablet containing 20 mg of pimavanserin tartrate, which is equivalent to 17 mg of pimavanserin free base, and inactive ingredients[3].

Mechanism of Action

Although the exact mechanism of action of pimavanserin is unknown, a combination of inverse agonist and antagonist activity at the serotonin 2A receptors (5-HT2A) and, to a lesser extent, at the 5-HT2C receptors, has been theorized. During clinical trials, pimavanserin showed no appreciable binding affinity for dopamine (including D2), histamine, muscarinic, or adrenergic receptors[4].

Pharmacokinetics

Pimavanserin and its major active N-desmethylated metabolite AC- 279 have demonstrated a time to

maximum plasma concentration of six hours (range,four to 24 hours) with mean plasma half lives of approximately 57 hours for pimavanserin and 200 hours for AC-279. In the presence of high-fat meals, its maximum plasma concentration and area under the curve were decreased by 9% and increased by 8%, respectively. Pimavanserin has dose-proportional pharmaco-kinetics after single oral doses from 17 to 255 mg and yields similar results in both patients with PD and healthy individuals. The bioavailability of pimavanserin oral tablets and oral solution is identical[5]

Safety Profile

Warnings and Precautions

The prescribing information for pimavanserin contains a boxed warning for increased mortality in elderly patients (65 years of age or older) with dementia-related psychosis. Elderly patients with dementia-related psychosis treated with antipsychotic drugs are at an increased risk of death. Pimavanserin is not indicated for the treatment of patients with dementia-related psychosis unrelated to PD-associated hallucinations and delusions[6].

Pimavanserin prolongs the QT interval, and its use should be avoided in patients with known QT prolongation or in combination with other drugs known to prolong the QT interval. This includes some antiarrhythmics quinidine, procainamide, amiodarone certain anti-psychotic medications like ziprasidone, chlorpromazine, thioridazine and certain antibiotics such asgatifloxacin, moxifloxacin[5]

Drug-Drug Interactions

Coadministration of pimavanserin and strong CYP3A4 inhibitors (e.g., itraconazole, ketoconazole, clarithromycin, indinavir) increases the plasma concentration of pimavanserin; therefore, a dose reduction of pimavanserin is recommended. Patients should be monitored for efficacy, and a dose increase may be needed if pimavanserin is administered with strong CYP3A4 inducers like, rifampin, phenytoin, carbamazepine.

No dosage adjustment is required when carbidopa/levodopa is administered concomitantly with pimavanserin based on pharmacokinetic studies[6].

Dosage and administration

The recommended dose of pimavanserin is 34 mg taken orally as two 17-mg tablets once daily with or without food and without titration. With strong CYP3A4 inhibitors (e.g., ketoconazole), the recommended dose of pimavanserin is 17 mg once daily[7]. Monitor patients for reduced efficacy and potentially increase the dose when pimavanserin is administered with strong CYP3A4 inducers.

Discussion

This randomized discontinuation trial examined sustained response to pimavanserin treatment followed by the effect of treatment discontinuation on recurrence of psychosis in patients with several types of neurodegenerative disease[8]. After randomization, the percentage of patients who had a relapse of psychosis was 13% among those who continued to receive pimavanserin and 28% among those who were switched to placebo, with an estimated difference of 16 percentage points. The risk of trial discontinuation for any reason was lower with pimavanserin than with placebo[9].

This trial has limitations. Requiring sustained response in the open-label phase limited the ability to assess future treatment response in patients who did not meet the full response criteria at week 8. Pimavanserin has shown efficacy in patients with hallucinations and delusions associated with Parkinson's disease psychosis and is approved for that indication, and approximately 15% of the patients in the trial had Parkinson's disease, which may have skewed the results in favor of pimavanserin. Previous trials that involved patients with Parkinson's disease–related psychosis included only those with normal cognition or with MMSE scores of 21 or more[10]

Conclusion

Psychotic symptoms are common in Parkinson's disease as a result of patho-physiological changes involving many factors, including the motor symptoms of the disease; the drug-related adverse effects of treatment; multiple neuro-chemicals (i.e., dopamine, serotonin, acetylcholine); changes in sleep and perception; and the patient's genetics. PD-associated psychosis, which is associated with significantly increased morbidity and mortality, had no FDA approved treatment until the agency OK'd pimavanserin in 2016. Although this new treatment has demonstrated its efficacy and relative safety, additional studies—including those of longer duration, larger sample size, and more specific patient populations—are needed to enhance pimavanserin's safety and efficacy profile. a trial that was stopped early for efficacy, patients with dementia related psychosis who had a response to pimavanserin had a lower risk of relapse with continuation of the drug than with discontinuation. Longer and larger trials are

required to determine the effects of pimavanserin in dementia -related psychosis.

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WILSON'S DISEASE-A RVIEW

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ABSTRACT

Wilson's disease (WD), also known as hepatolenticular degeneration, is an autosomal recessive inherited disorder resulting from abnormal copper metabolism. Reduced copper excretion causes an excessive deposition of the copper in many organs such as the liver, central nervous system (CNS), cornea, kidney, joints, and cardiac muscle where the physiological functions of the affected organs are impaired. It is now believed that a defect in P-type adenosine triphosphatase (ATP7B), the gene encoding the copper transporting P-type ATPase, is responsible for hepatic copper accumulation. Deposited copper in the liver produces toxic effects via modulating several molecular pathways. WD can be a lethal disease if left untreated.

Key Words: Wilson's disease, osteomalacia, trientine

Introduction

Wilson's disease (WD), also known as hepatolenticular degeneration, is an autosomal recessive disorder resulting from abnormal copper metabolism, subsequently leading to the accumulative deposition of copper in the target organs and impairing the normal functions of the affected organs. WD is not a new disease, first described by Wilson in 1912, but the exact molecular mechanisms leading to the abnormal copper metabolism are a myth. Liver is the major organ for copper deposition in patients with WD. Excessive copper deposition in the liver causes hepatic dysfunction, resulting in a large spectrum of manifestations ranging from mild abnormalities in liver function tests, to acute or chronic hepatitis, cirrhosis, or even fulminant hepatitis. Excessive deposition of the copper in brain may cause neurological disorders such as Parkinson-like symptoms, including bradykinesia, tremor and dystonia, or neuropsychiatric symptoms, such as hypomnesia, dysgnosia, and personality abnormalities. The Kayser-Fleischer (KF) ring, a rusty brown ring around the cornea of the eye, is the result of copper deposition in the cornea. Copper deposition in other organs may cause corresponding clinical disorders such as osteoarthritis, abnormal kidney function tests, and cardiomyopathy.

ATP7B is currently believed to be the key culprit gene for WD. Expression of ATP7B is found in most organs with a particularly high level in liver, kidney and placenta. ATP7B encodes the copper transporting P-type ATPase, a very important enzyme for copper transport in the body. Mutation of the ATP7B gene is closely linked to the impairment of copper excretion, leading to abnormal deposition of copper in the target organs. Increased tissue copper level may induce a series of harmful biochemical reactions, particularly oxidative stress, which can damage the structure and integrity of mitochondria, leading to cell injury[1]

Pathogenesis

Copper is needed by the body for a number of functions, predominantly as a cofactor for a number of enzymes such as ceruloplasmin, cytochrome c oxidase, dopamine β-hydroxylase, superoxide dismutase and tyrosinase. Copper enters the body through the digestive tract. A transporter protein on the cells of the small bowel, copper membrane transporter 1 (Ctr1; SLC31A1), carries copper inside the cells, where some is bound to metallothionein and part is carried by ATOX1 to an organelle known as the trans-Golgi network. Here, in response to rising concentrations of copper, an enzyme called ATP7A (Menkes' protein) releases copper into the portal vein to the liver. Liver cells also carry the CMT1 protein, and metallothionein and ATOX1 bind it inside the cell, but here it is ATP7B that links copper to ceruloplasmin and releases it into the bloodstream, as well as removing excess copper by secreting it into bile. Both functions of ATP7B are impaired in Wilson's disease. Copper accumulates in the liver tissue; ceruloplasmin is still secreted, but in a form that lacks copper and is rapidly degraded in the bloodstream. When the amount of copper in the liver overwhelms the proteins that normally bind it, it causes oxidative damage through a process known as Fenton chemistry ; this damage

eventually leads to chronic active hepatitis, fibrosis and cirrhosis. The liver also releases copper into the bloodstream that is not bound to ceruloplasmin. This free copper precipitates throughout the body but particularly in the kidneys, eyes and brain. In the brain, most copper is deposited in the basal ganglia, particularly in the putamen and globus pallidus; these areas normally participate in the coordination of movement as well as playing a significant role in neurocognitive processes such as the processing of stimuli and mood regulation. Damage to these areas, again by Fenton chemistry, produces the neuropsychiatric symptoms seen in Wilson's disease[2].

Clinical Manifestation

Clinical Manifestations of Wilson's Disease

Hepatic asymptomatic hepatomegaly, persistently elevated transaminases, acute hepatitis, chronic hepatitis, cirrhosis (compensated and decompensated), acute liver failure, acute chronic liver failure, fatty liver, isolated splenomegaly, cholelithiasis, neuropsychiatric tremors, dystonia, Parkinsonism, choreoathetosis, seizures, dysarthria, drooling, clumsiness, incoordination, gait disturbance, behavioural changes, deteriorating school performance, depression, anxiety, psychosis, Osseo muscular arthralgia, arthritis, fractures, osteoporosis, osteomalacia, chondromalacia, hemolytic anemia, thrombocytopenia, pancytopenia, coagulopathy, ocular Kayser-Fleischer rings, sunflower cataracts, renal stones, renal tubular acidosis, Fanconi syndrome

Other organ systems

Eyes: Kayser–Fleischer rings (KF rings) may be visible in the cornea of the eyes. Wilson's disease is also associated with sunflower cataracts exhibited by brown or green pigmentation of the anterior and posterior lens capsule.

Kidneys: renal tubular acidosis a disorder of bicarbonate handling by the proximal tubules leads to nephrocalcinosis and occasionally aminoaciduria

Heart: cardiomyopathy is a rare but recognized problem in Wilson's disease; it may lead to heart failure and cardiac arrhythmias

Hormones: hypoparathyroidism, infertility, and recurrent miscarriage.

Diagnosis

Ceruloplasmin: Levels of ceruloplasmin are abnormally low (<0.2 g/L) in 80–95% of cases. It can, however, be present at normal levels in people with ongoing inflammation as it is an acute phase protein. Low ceruloplasmin is also found in Menkes disease and aceruloplasminemia, which are related to, but much rarer than Wilson's disease.

Serum and urine copper: Serum copper is low, which may seem paradoxical given that Wilson's disease is a disease of copper excess. However, 95% of plasma copper is carried by ceruloplasmin which is often low in Wilson's disease. Urine copper is elevated in Wilson's disease and is collected for 24 hours in a bottle with a copper-free liner. Levels above 100 µg/24h (1.6 µmol/24h) confirm Wilson's disease, and levels above 40 µg/24h (0.6 µmol/24h) are strongly indicative.

Slit-lamp examination: The eyes of the patient are examined using a slit-lamp to look for Kayser–Fleischer rings, which are strongly associated with Wilson's Disease and are caused by copper deposition on the inner cornea in Descemet's membrane.

Liver biopsy: This is assessed microscopically for the degree of steatosis and cirrhosis, and histochemistry and quantification of copper are used to measure the severity of the copper accumulation. A level of 250 µg of copper per gram of dried liver tissue confirms Wilson's disease.

Genetic testing Mutation analysis of the ATP7B gene, as well as other genes linked to copper accumulation in the liver, may be performed. Once a mutation is confirmed, it is possible to screen family members for the disease as part of clinical genetics family counseling[3].

Management

Diet : In general, a diet low in copper-containing foods is recommended with the avoidance of mushrooms, nuts, chocolate, dried fruit, liver, sesame seeds and sesame oil, and shellfish.

Physical and occupational therapies: Physiotherapy and occupational therapy are beneficial for patients with the neurologic form of the disease. The copper chelating treatment may take up to six months to start working, and these therapies can assist in coping with ataxia, dystonia, and tremors, as well as preventing the development of contractures that can result from dystonia.

Transplantation: Liver transplantation is an effective cure for Wilson's disease but is used only in particular scenarios because of the risks and complications associated with the procedure. It is used mainly in people with fulminant liver failure who fail to respond to medical treatment or in those with advanced chronic

liver disease[4].

Pharmacological Treatment

D-Penicillamine : DP is the preferred standard therapy for WD. It is rapidly absorbed from the intestine, bound to plasma proteins, and more than 80% is excreted in the urine. DP binds to copper through disulfide bonds, and every gram promotes urinary excretion of 200 mg of copper. It also induces hepatic metallothionein, a cytosolic metal-binding protein that sequesters copper, and renders it nontoxic. DP chelates several heavy metals, not just copper, and has many adverse effects necessitating discontinuation in up to 30% of patients with WD . Despite the serious adverse effects, DP is still the primary drug for management of hepatic WD in view of its time-tested efficacy, easy availability, and reasonable cost[5].

Trientine : Trientine (triethylenetetramine-2-hydrochloride) is a chelator with a mechanism of action similar to DP, with fewer adverse reactions. The dosage is 750–1500 mg/day in 3 divided doses on empty stomach for adults (20 mg/kg/day for children). Although trientine has traditionally been used for patient's intolerant to DP, recent studies suggest that it can be used as a first-line drug. It chelates iron and other heavy metals as well; hence, treated subjects should be monitored for iron deficiency. Trientine can also cause paradoxical worsening in neurological WD, Hence, it should be started in low doses and increased slowly similar to DP[6].

Zinc : Zn acts by inducing metallothionein in enterocytes which preferentially binds absorbed Cu, sequesters it in the enterocytes, and prevents its entry into the portal circulation. As the enterocytes are naturally sloughed into the lumen, copper is excreted in the feces. Zinc also induces metallothionein in hepatocytes and protects against Cu toxicity. Unlike DP and trientine, Zn acts by increasing the fecal excretion of Cu. Although all 3 salts—acetate, sulfate and gluconate are effective, acetate salts are preferred because of lesser incidence of gastric side effects. Adults require 150 mg/day of elemental zinc in 3 divided doses, whereas children and those under 50 kg are given only 75 mg/day. Zinc should be taken on empty stomach to ensure better absorption[7].

Ammonium tetra thiomolybdate: Ammonium TTM, originally used to treat copper poisoning in veterinary angiogenic properties. If ammonium TTM is taken after meals, it binds to the copper in the food, thus preventing its absorption. If taken on empty stomach, it is absorbed into the blood and forms a complex with circulating copper preventing cellular uptake, leading to its excretion in urine. The dosage used is 20 mg 3 times a day with meals and 20 mg 3 times a day in between the meals[8].

Conclusion

Wilson's disease is a disorder of hepatobiliary copper excretion manifested predominantly by hepatic and neurologic copper toxicosis and inherited in an autosomal recessive pattern. Although the specific underlying biochemical defect remains to be defined, specific therapy is available and usually successful. Maintaining a high index of suspicion is critical in diagnosing this readily treatable inherited disease.

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GUIDELINES TO ANTIBIOTIC THERAPY-A REVIEW

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ABSTRACT

Antibiotics are chemotherapeutic substances that inhibit or kill the growth and replication of microorganisms. The primary aim of the hospital antimicrobial policy is to minimize the morbidity and mortality due to antimicrobial-resistant infection; and to preserve the effectiveness of antimicrobial agents in the treatment and prevention of communicable diseases. An antibiogram is an overall profile of antimicrobial susceptibility testing results of a specific microorganism to a battery of antimicrobial drugs. Judicious use of antibiotics is necessary to control antibiotic resistance and ADRs associated with antibiotics but also improves patient outcomes.

Key Words: Antibiotics, resistance, antibiogram, stewardship

Introduction

Antibiotics are chemotherapeutic substances that inhibit or kill the growth and replication of microorganisms. They are derived from microorganisms or derived synthetically. The term antibiotics-literally means "opposing life".

Waksman and woodruff in 1942 formally defined an antibiotic as a chemical substance produced by a microorganism, which at a high dilution can inhibit the growth and / or multiplication, or kill another microorganism. [1]

Antibiotics have been used since ancient times. Many civilizations used topical application of moldy bread, with many references to its beneficial effects arising from ancient Egypt, Nubia, China, Serbia, Greece, and Rome[2]. The first person to directly document the use of molds to treat infections was John Parkinson (1567–1650). Antibiotics revolutionized medicine in the 20th century. Alexander Fleming (1881–1955) discovered modern day penicillin in 1928.

WHO- Hospital Antibiotic Policy

The primary aim of the hospital antimicrobial policy is to minimize the morbidity and mortality due to antimicrobial-resistant infection; and to preserve the effectiveness of antimicrobial agents in the treatment and prevention of communicable diseases.

Scope of hospital antibiotic policy: The antibiotic policy is essentially for prophylaxis, empirical and definitive therapy. The hospital antibiotic policy shall be based upon:

spectrum of antibiotic activity, pharmacokinetics or pharmacodynamics of these medicines, Adverse effects, potential to select resistance, cost and Special needs of individual patient groups.

Establish a multidisciplinary antibiotic management team to draft policy:

An efficient antibiotic policy in a health-care setting shall demand from the top management their full commitment as well as their total support to the development and implementation of this policy.

To develop an antibiotic policy each hospital shall establish a multidisciplinary antibiotic management team (AMT). The team's functions should include developing a hospital antimicrobial policy, monitoring the implementation of the antibiotic policy, receiving feedback, assessing outcome and discussing with clinicians, conducting a revision of the policy every year.

Fig-1

Antibiotic Resistance

Antibiotic resistance is one of the biggest threats to global health, food security, and development today. Antibiotic resistance occurs when bacteria change in respose to use of antibiotics[3]. Antibiotic resistance is rising to dangerously high levels in all parts of the world. New resistance mechanisms are emerging and spreading globally, threatening our ability to treat common infectious diseases[4].

Antibiotic Stewardship

Antimicrobial Stewardship: Principles and Practice include Colonization vs. Infection,Narrow vs. Broad Spectrum Therapy,Antibiotic Resistance,Monotherapy vs. Combination TherapyPO and IV-to-PO Switch Antibiotic Therapy,Antibiotic De-escalation, Empiric Antibiotics for Fever and Leukocytosis,Pharmacoeconomic Considerations[5].

Factors In Antibiotic Selection

Spectrum, Tissue Penetration, Antibiotic Resistance[5]

Antibiogram: An antibiogram is an overall profile of antimicrobial susceptibility testing results of a specific microorganism to a battery of antimicrobial drugs[6]. This profile is generated by the laboratory. Only results for antimicrobial drugs that are routinely tested and clinically useful should be presented to clinicians.[6]

Uses Of Antibiogram

Antibiograms help guide the clinician and pharmacist in selecting the best empiric antimicrobial treatment in the event of pending microbiology culture and susceptibility results. They are also useful tools for detecting and monitoring trends in antimicrobial resistance. When antimicrobial susceptibility testing data are summarized cumulatively for a hospital, healthcare system, or other healthcare facility periodically (such as yearly or quarterly), trends in resistance can be identified and investigated[7].

Conclusion

Antibiotics are among the safest of drugs which are used to treat community infections. The discovery of antibiotics have had a major impact on the life-threatening infections and in reducing the morbidity associated with surgery and many common infectious diseases. Judicious use of antibiotics is necessary to control antibiotic resistance and ADRs associated with antibiotics but also improves patient outcomes.

Antibiotic stewardship is important for preserving existing antibiotics and improving patient outcomes. Overuse of antibiotics and negligence to complete full course of antibiotics once prescribed is the leading cause for resistance. Therefore, antibiotic prescribing should be prudent, thoughtful and rational.

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FOOD POISONING- MENACE OF THE DAY

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Introduction

Food poisoning is caused by eating or drinking contaminated food or liquid that contains bacteria or other harmful elements. It normally results in nausea, vomiting, and diarrhoea. Most minor ailments on food poising heal without treatment. It can sometimes cause significant illness. Symptoms might linger from 24 hours to one week after eating infected food. Food poisoning is often misdiagnosed due to its varied onset and length.

Rotavirus, Salmonella poisoning, E. coli, Listeriosis, and Norovirus are the most common source of food borne illnesses.

Common causes for food poisoning are many. The following are some of the major areas of foodborne illnesses, the time from exposure to the beginning of symptoms and common sources of contamination.

Food-poisoning symptoms

Food poisoning symptoms vary by cause. They may start hours or weeks after the cause. Upset stomach, vomiting, diarrhoea with or without blood, stomach discomfort and cramps, fever, headache, etc. Food poisoning seldom affects the neurological system, but it can produce impaired or double vision, limb paralysis, swallowing issues, voice changes, and more.

Food additive impact the health of human being in various degrees. It may enhance taste, appearance, and shelf life. Approved additives are safe to consume to an extent only. So, it is advisable to avoid the non-essential additives from your kitchen.

Savoury foods are often flavoured with MSG (mono sodium glutamate). Frozen dinners, salty snacks, and canned soups contain it. Restaurant and fast-food recipes often include it in greater proportions for the yummy class. Artificial food colouring brightens candy and sauces. Hypersensitive youngsters and allergic responses might result from artificial food colouring. Red 3 increased thyroid cancer risk in animals. Processed meats include sodium nitrite, which can form lethal nitrosamine. Nitrites and processed meats may increase cancer risk. Long chain carbohydrate guar gum thickens and binds food. It improves digestion, lowers blood sugar and cholesterol, and increases satiety. High-fructose corn syrup causes obesity, diabetes, and inflammation. It adds empty calories to your diet.

Artificial sweeteners may help regulate glucose and weight. Some types may cause headaches, but when used moderately, they are safe. Carrageenan may raise blood sugar and produce gut ulcers and growths in test tubes and animals. Carrageenan also caused early ulcerative colitis relapses. Sodium benzoate may cause hyperactivity. It may produce cancer-causing benzene when combined with vitamin C. Trans fats cause inflammation, heart disease, and diabetes. Animal studies suggest artificial flavouring may harm bone marrow cells. Human effects need further study. Yeast extract is high in salt and glutamate, which may cause symptoms. Adding little amounts of yeast extract to food is unlikely to cause harmful responses in most people.

Preservatives and organs

Food preservation prevents microbial growth. Lids keep flies and other insects out of rice and curries so as to prevent infection. Preservatives can trigger transient illness, though food preservation extends shelf life.

For ages, salt and edible oils have been utilised to suppress microbial growth. Sugar, salt, nitrites, BHA, BHT, TBHQ, vinegar, citric acid, and calcium propionate are preservatives. Salt, nitrite, spices, vinegar, and alcohol have preserved food for ages. Preservatives include benzoic acid, calcium sorbate, erythorbic acid, potassium nitrate, sodium benzoate, etc. Jams and jellies are preserved with sugar. Smoking, canning, sterilising, dehydration, heat and cold procedures, and lyophilization.

FDA has approved over 10,000 food additives to preserve, package, or change flavour, appearance, texture, or nutritional value. However, rising research supports avoiding some chemicals, especially by

children. Fresh, unprocessed foods are best. The American Academy of Paediatrics guideline on food additives and child health states that a handful may affect hormones, growth, and development. Some may also cause childhood obesity. These substances may affect children more due to their size and diet. Thus, they may have additional adverse effects.

BHA (Butylated hydroxyanisole) is a potential human carcinogen, according to the IARC (International Agency for Research on Cancer). BHA is a Category 1 priority substance for the European Commission on Endocrine Disruption because it disrupts hormone function.

Avoid sodium benzoate during pregnancy due to its toxicity. It means the best way for healthy diet is keeping away from packed foods. Animal research showed that sodium benzoate/sodium phenylacetate prenatally damaged layer 5 cortical pyramidal cells. More research is needed to validate foetal effects. Preservative-laden processed foods should be avoided wherever possible.

You can resort to honey, salt, and some fruits prevent rotting. Natural preservatives stop harmful germs. Carrageenan, xanthan gum, guar gum, ascorbic acid, agar, gelatine, natural flavours, lecithin, etc. are natural preservatives.

Food poisoning prevention

Avoid food poisoning by frequently washing hands and dishes with hot, soapy water, washing uncooked vegetables and fruits, handling raw foods hygienically, especially meat, refrigerating or freezing perishables, and cooking meat to the right temperature. Developing countries struggle with cold chain food processing and transit, notably meat and fish preparations and extremely sensitive foods. As a rule, many families are keeping vegetables and fruits in a mixture of salt, turmeric powder & water to get off the poisonous coating they might have. salt infusion can be best utilized for disinfecting the vegetables and fruits, turmeric solution can eliminate harmful bacteria off your greens. Vinegar mixture can remove pesticides and germs. Mixture of 1-2 spoons of baking soda in lemon juice, diluted with water can be sprayed on fruits & vegetables for cleansing.

Kerala has witnessed many major and critical cases of food poisoning owing to contamination of nonvegetarian diets. At least 65 people, most of whom were college students, were taken to the hospital in Ernakulam district's Parur recently and food poisoning is thought to be the cause. Later, the town's authorities shut down the hotel where the food was ordered. The most recent problem happened at the same time that the government announced a set of steps, one of which was to ban egg-based mayonnaise in all food stores. They ate Al-faham and Shawai (popular Arabic dishes); the mayonnaise sauce served along with the dishes was suspected to be main villain, and most of them later got sick with diarrhoea, and feeling sick and few people were critical. Death also was reported from Kasaragod.

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Food Poisoning- Menace of the day

BRIEF REPORT ON KPGA ACTIVITIES

Abdul Nazeer PU
Gen.Secretary, KPGA

There were numerous activities conducted by KPGA from the month of September to till date, A few of them are listed below.

World Pharmacist Day Celebration

On September 25, 2022, the WPD celebration was held on a virtual platform jointly by KPGA and IPGA. The year's theme was "Pharmacy united in action for a healthier world".

Dr. Joseph Marthoma College of Pharmaceutical Science and Research, Kattanam

The World Pharmacist Day program and Women's Wing declaration were held at the Dr. Joseph Marthoma College of Pharmaceutical Science and Research in Kattanam on September 24th, 2022. The program was inaugurated by Adv. Seema S, Special Public Prosecutor and delivered an excellent speech. There were three scientific sessions.

Declaration of KPGA Women's Wing

Happy to inform that the KPGA Women's Wing was declared by Dr. P. K. Sreekumar at the Dr. Joseph Marthoma College of Pharmaceutical Science and Research, Kattanam, Kayamkulam. Principal Dr. Ansa Mathew and Vice Principal Dr. Subash Philip were present on the occasion.

Poster Competition

The students' wing of KPGA conducted an online poster competition in connection with World Pharmacists Day 2022. The topic of the competition was "Pharmacists: Help people to get the best from their medicines and stay healthy". Cash prizes were given to the winners. The first prize was bagged by Ms. Niranjana Raju (B. Pharm), Chemists College of Pharmaceutical Sciences and Research, Puthencruz, Ernakulam. The second and third prizes were won by Mr. Muhammed Danish Haneefa (B. Pharm), Moulana College of Pharmacy, Perinthalmanna and Ms. Sharon Emilia James (Pharm.D), KVM College of Pharmacy, Cherthala respectively.

Nirmala College of Health Science, Chalakudy

KPGA Student's Wing program was conducted at on 27th September 2022. Dr. Suthorsan, Principal- NCHS, Dr. P K Sreekumar, Dr. Boby Johns G, Dr. Jeny Samuel and Mr. P. P John addressed the audience.

Representation for the pharma city project

In a strategic move for our dream project ' The Pharma City ', KPGA office bearers had given a representation to the honorable minister for industries Mr. P. Rajeev. The delegates consisted of Dr. P. K Sreekumar, Mr. Mathew Kokad, Mr. Abdul Nazeer and Mr. Unnikrishna Paniker. KPGA made a representation to the Private Pharmacy College Owners Association too in this regard. A delegation handed over letters and brochures in connection with the Pharma City to the Vice Chancellor, Pro Vice Chancellor and Registrar of Kerala University of Health Sciences.

Webinar on Rabies

KPGA Women's Wing conducted a webinar on 'Rabies- One Health Zero Death'. Speaker was Dr. Karthika M (Assoc. Professor, Dept. of Community Medicine Govt. College, Konni, Kerala) on 8th October, 2022.

Chemist's College of Pharmaceutical Science & Research, Puthencruz, Ernakulam

KPGA jointly with Chemist's College of Pharmaceutical Science & Research, Puthencuruz, Ernakulam

conducted a first aid basics workshop 'Save a Life' on 4th November 2022, by the Kerala Pharmacy Graduate Association women's forum, Doctor of Pharmacy Association and Apollo Adlux hospital, Angamaly at the College Auditorium. Participants included B.Pharm, D.Pharm, Pharm.D students from different colleges and community Pharmacists across Kerala. Prof. Dr. C. Vijayaraghavan, principal CCPSR delivered presidential address. Dr. P.K Sreekumar, President KPGA inaugurated the workshop by lightning the lamp. Mr. Abdul Naseer, general secretary KPGA and Dr. Kala D, chairperson KPGA women's forum conveyed the message on importance of training on first aid skills Dr. Nobil Scaria, head of clinical pharmacy, Apollo Adlux hospital, Angamaly gave an overview of the workshop. Dr. Binoy Xavier, department of emergency medicine led the training session. Certificates were provided to the participants upon completion of the training session.

National Pharmacy week- Virtual program

KPGA jointly with IPGA conducted a webinar on 20th November 2022. P. K Sreekumar welcomed the delegates, Dr. Satheesh Kumar C. S delivered the presidential address and the session was was inaugurated by Mr. Ravi Uday Baskar, Director General of the Pharmaceutical Export Promotion Council of India. Dr. Atul Kumar Nasa (National President, IPGA), and Mr. Jayan P. M (Drugs Controller Kerala State) were the Guests of Honor. Mr. Ranjit Basrshikar delivered the Keynote Address. Dr. Jeny Samuel (Assoc. Prof. St. Joseph's College of Pharmacy, Cherthala) was the Moderator of the program. Mr. PU Abdul Naseer expressed the vote of Thanks.

Refresher's Programme

The KPGA women's forum organized a refresher's program on "The expanding role of Pharmacists: A Paradigm for Health Care" on February 11th, 2023, conducted at AM College of Pharmacy, Vavvakkavu, Karunagapally. More than 150 students participated in the program. The Principal Mr. Shafeeq S presided the program. The key speakers were Dr. PK Sreekumar, Dr. Kala D and Dr. Jeny Samuel.

Kerala Pharmaceutical Congress -2023

For a Self Reliant Pharma Industry for the future of Pharmacy in Kerala

At St. James' College of Pharmaceutical Sciences, Chalakudy,Kerala,India

||| Activities & News |||

Campus News

Nirmala College of Health Science, Chalakudy

On the eve of 61st National Pharmacy Week Celebration - Nirmala College of Health Science, Chalakudy has conducted a Free Medical camp for Meloor Grama Panchayat people in association with Apollo Adlux hospital on 24th Nov 2022. The camp was inaugurated by Sri. T. J. Saneesh Kumar Joseph, MLA, Chalakudy Constituency and it was presided by the Meloor Panchayat President Mrs. Sunitha M.S . The Specialist Doctors from General Medicine, Orthopedics, Ophthalmology and ENT of Apollo Adlux have rendered their service to around 150 people. Clinical Pharmacy service was provided by Dr Nobil Skaria, Pharm.D. The diagnostic procedures have been conducted with all modern equipment. Students of Nirmala College of Health Science have supported as volunteers for this camp. Indeed, it was very helpful to the local community of Meloor Panchayat.

Free Medical camp at Meloor Grama Panchayat organized by Nirmala College of Helath Science, Chalakudy

Gallery

KPGA ACTIVITIES


AM College of Pharmacy, Karunagappally


Ezhuthachan College of Pharmaceutical Sciences, Neyyattinkara


Nirmala College of Health Science, Chalakudy

|| Upcoming Mega event ||

First **Kerala Pharmaceutical Congress** on February 25 & 26 ,2023
At St. James' College of Pharmaceutical Sciences, Chalakudy, Kerala, India

25th -26th FEBRUARY 2023

1ST KERALA PHARMACEUTICAL CONGRESS (KPC 2023)

Theme : A self reliant pharma industry for the future of pharmacy in Kerala

Organized by

KERALA PHARMACY GRADUATES' ASSOCIATION (KPGA)

Venue: St. James College of Pharmaceutical Sciences, Chalakudy, Thrissur

KPC 2023

25-26 FEBRUARY 2023

REGISTRATION

(QR code for registration)

No refund or transfer of payment Please Contact : kpcregistation2023@gmail.com

Registration link: https://forms.gle/E2n58Umx6YhSvkXe9 CLICK HERE

Registration link for KPGA membership: https://kpga.in/downloads/KPGA-membership-form-new-pdf.pdf

			EARLY BIRD (31/12/2022 TO 31/01/2023	REGULAR (01/02/2023 TO 15/02/2023)	SPOT REGISTRATION
Registration fees	STUDENT DELEGATES	KPGA MEMBERS	₹ 600	₹ 700	₹ 800
		KPGA NON-MEMBERS	₹ 700	₹ 800	₹ 900
	RESEARCH SCHOLARS/FACULTY/ OTHER DELEGATES	KPGA MEMBERS	₹1000	₹ 1100	₹ 1200
		KPGA NON-MEMBERS	₹ 1200	₹ 1300	₹ 1400
	FOREIGN DELEGATES		$25	$30	$35

Accomodation details

	Tariff Details/ Day/ Person*
Single occupancy Hostel	₹ 750
Double occupancy Hostel	₹ 375
Triple occupancy Hostel	₹ 275
Hotel Type I (A/C)	₹ 2750
Hotel Type II (A/C)	₹ 2250

*Rate mentioned is inclusive of all taxes

For accomodation enquires e-mail:kpcaccomodation2023@gmail.com

FOR ACCOMODATION BOOKING

CLICK HERE

VENUE DETAILS

St James College of Pharmaceutical Sciences
St James Medical Academy
River Bank
Chalakudy-680307
Phone: 04802-710936
04802-710981

FOR ENQUIRIES, PLEASE CONTACT

e-mail:keralapharmcongress@gmail.com

Kerala Pharmacy Graduates' Association

PHARMLINE-The Official Publication Of Kerala Pharmacy Graduates' Association

PHARMLINE is the official publication of KPGA and is published since 1981. Pharmline is a tri annual publication. The main aim of the publication is to keep Pharmacists informed on current research and best practices, as well as serving as a platform for the exchange of ideas, knowledge and opinion among pharmacists and related disciplines. The publishers' welcome contributions of Pharmaceutical relevance. Original articles are considered for publication on the condition that they have not been published, accepted or submitted for publication elsewhere. The editor reserves the right to edit manuscripts to fit articles with in space available and to ensure conciseness, clarity and stylistic consistency. All scientific articles submitted for publication are subject to a double blind review procedure.

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