PHARMACY BULLETIN BIL. 3/2022
(SEPTEMBER - DECEMBER)

SPECIAL TOPIC

Camphor

Staff Updates

Medication Safety:
Camphor Safety Issue

Disease Management:
Gout

Drug Updates:
Oxycodone HCl 1mg/ml Oral Solution
Vedolizumab 300mg Powder for Injection

Pharmacy R&D

Pharmacy Activities

PUBLISHED BY JABATAN FARMASI HOSPITAL TENGKU AMPUAN AFZAN, KUANTAN, PAHANG

HTAA STAFF UPDATES 2022

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Date Reported Duty: 17/10/2022

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To: Farmasi Logistik
Date Reported Duty: 3/10/2022

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To: Farmasi Bekalan Wad
Date Reported Duty: 1/11/2022

HTAA STAFF UPDATES 2022

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Date Resigned: 15/9/2022

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Position: Pegawai Farmasi UF41 (K)
From: Farmasi Bekalan Wad
Date Resigned: 19/9/2022

RETIRED

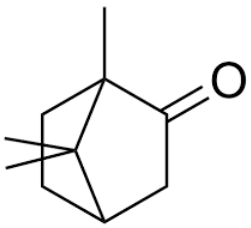
Name: Ahmad Suhaimi bin Abdul Rahman
Position: Penolong Pegawai Farmasi UF38
From: Farmasi Klinik Pakar
Date Retirement: 20/10/2022

CAMPHOR SAFETY ISSUES

By: Amira Nabila Binti Mat Roof

INTRODUCTION

Camphor, or also known in Bahasa Melayu as *"kapur barus"* from a class of naturally occurring organic chemicals known *as* terpenoids. It was originally extracted from the camphor tree, *Cinnamomum Camphora* and obtained through a distillation process. These days, camphor is chemically synthesised using turpentine oil.[1] It is commonly used as a fragrance in cosmetics, food flavouring, household cleaners as well as a rubefacient analgesic for muscle aches.[1] Additionally, it also has other beneficial properties such as insecticidal, antimicrobial and anti-nociceptive to name a few.[1] However, camphor toxic properties and camphor poisoning have been documented in human beings to date.



Camphor:
A bicyclic monoterpene known to potentiate both heat and cold sensations

CAMPHOR

Camphor is a bicyclic monoterpene known to potentiate both heat and cold sensations.[2] Camphor repeatedly sensitizes Transient Receptor Potential (TRP) Vanilloid Subtype 1 (TRPV1) and TRP V Vanilloid Subtype 3 (TRPV3) channels, that leads to the analgesic effects of camphor. It also activates cold-sensitive transient receptor potential melastatin 8 (TRPM8) and sensitizes cold-induced calcium transients, which explains the cooling effect of camphor following dermal application.[3]

INDICATION[4]

Camphor oil, when used topically works as a decongestant and cough suppressant. Camphor may also be used as a muscle rub to relieve muscle cramps, spasms, and stiffness. It may also be effective in relieving pain, inflammation, and swelling due to arthritis where the hot or cold sensations helps distract from the pain. Camphor has a wide variety of topical uses due to its antibacterial, antifungal, and anti-inflammatory properties. It can be used to treat skin irritation and itchiness, to relieve pain and inflammation, improve respiratory function. The antifungal properties of camphor make it beneficial in treating toenail fungus.

COMMON DOSAGES: ADULT

Topical applications:

Pruritus & Pain: 3-11% ointment applied 3 times – 4 times daily. [5,6]

Osteoarthritis: Topical product containing combination of camphor (32mg/gram), glucosamine sulfate (30mg/gram) and chondroitin sulfate (50mg/gram) applied when needed to sore joints for up to 8 weeks.[6]

Inhalation: [5,6]

Cough: 1 tablespoon in a quart of cold water in a steam vaporiser 3 times daily

COMMON SIDE EFFECTS

High doses of camphor can cause skin irritation and redness if used topically. Some who have taken camphor orally might experience burning of the mouth and throat as well as nausea, vomiting and troubled breathing. Camphor is easily absorbed through broken skin and can rapidly reach toxic levels in the body.[6]

Warning!

For paediatrics, camphor containing products are NOT for oral ingestion as it may cause severe, potentially fatal adverse reactions.

TOXICITIES

Camphor toxicity commonly occurs in children and has been extensively documented. A review of literature reveals persistent reports of toxicity resulting from exposure to even relatively small amounts. Around 3-5 mL of 20% camphor oil or > 30 mg/kg is a potentially lethal dose.[7] In the pediatric population, exposure to as little as 500 mg is cited as a cause of mortality. More commonly, 750mg to 1000 mg are associated with the development of seizures and death. [8,9] Camphor is rapidly absorbed after ingestion, thus onset of symptoms can be early, within 5-15 mins of ingestion.[8] Initially, patients may suffer from nausea, vomiting, headache, dizziness, and subsequently muscular excitability causing tremor and twitching.[1] Camphor concentration peaks in 1-3 hours post ingestion, leading to seizures or status epilepticus. Prolonged seizure may lead to exhaustion or asphyxia which causes coma and death. Inhalation of high doses of camphor may irritate the mucosal membrane, causing respiratory depression and apnoea.[1,8] Camphor is oxidized and conjugated by the liver and renally excreted.[8] Any poisoning and toxicity cases shall be referred to the Emergency Department for observation and treatment of the symptoms. To date, no available antidotes are approved for camphor poisoning. However, patient education is crucial to prevent camphor poisoning in both adults and children.

CAMPHOR POISONING: WHAT TO DO?[10]

- For asymptomatic patients with topical exposure to camphor products, the skin should be thoroughly washed with soap and water.
- For patients with topical splash exposures of camphor to the eye(s), the eye(s) should be irrigated with water.
- Patients with camphor inhalation exposures should be moved to an environment with fresh air.
- In cases of ingestion and presence of toxicity symptoms, immediately go to the emergency department.

Did you know? [5]

Camphor is also a fire hazard. It should be kept away from heat, including hot water and microwaves. Do not add the product in any container to be heated except when adding to cold water only in hot steam vaporizer. Heating camphor can cause splattering and result in burns.

Pregnancy Consideration

Camphor crosses the placenta and is toxic to the fetus following to exposure to large amounts, e.g., oral ingestion with poisoning symptoms. [5]

PRODUCT EXAMPLES

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Gout

By Safwah Suad binti Mohamed Yusof

BACKGROUND [1,2,3]

Gout is a disease that occurs in response to the deposition of monosodium urate (MSU) crystal in joints, bones, and soft tissues with one or a combination of gout flare, chronic gouty arthritis, or subcutaneous tophus. Gout can entail extra-articular conditions such as chronic nephropathy and urolithiasis while being primarily a musculoskeletal disorder involving acute and chronic arthritis, and bursitis. Gout is a consequence of persistent hyperuricaemia. Worldwide, the prevalence of gout ranges from 0.1% to 6.8% and 0.58 to 2.89 Incidence per 1,000 person-years. 7.44 million cases of gout were estimated in 2017 (incidence, 0.097%), with 41.22 million cases (0.54%) being the prevalence.

RISK FACTORS [1,4,5,6]

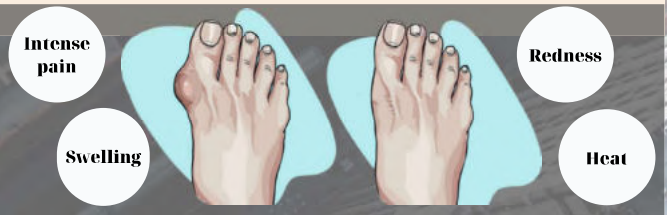
Non-modifiable risk factors such as race, older people and male gender increase the chance of developing gout. 85.2% of 54 cases in Perak was predominantly Malay, followed by other ethnics [4,5]. Mean age of gout patients were 60 years in one study [6], and 53 years in another study [4,5]. As a matter of fact, genetic variation in the ABCG2 gene and hypoxanthine-guanine phosphoribosyl transferase deficiencies are all contributing factors to gout.

Modifiable risk factors leading to gout are obesity with 68% of Malaysian being overweight in one study [6], high alcohol consumption, sugar-sweetened beverages, all red meat seafood, except n-3 PUFA-rich fish such as salmon, anchovies, mackerel, and sardine, certain drugs like diuretics, non-losartan angiotensin-receptor blockers, angiotensin-converting enzyme inhibitors, β-blockers, and cyclosporine as well as comorbidities like renal disease, hypertension, psoriasis and hematological neoplasm. Chances are low in patient receiving losartan or calcium channel blockers as well as diet rich with dairy products.

PATHOPHYSIOLOGY [7]

Overproduction or under-excretion of uric acid leads to the formation of monosodium urate crystals. These crystals can be phagocytosed by monocytes. Additional signaling by TLR2 and TLR4 is required to complete NLRP3 inflammasome activation and pro-inflammatory IL-1β production, leading to acute exacerbation of gouty arthritis. Flare resolution involves extracellular neutrophils trap that binds to sodium urate crystals. Aggregated extracellular neutrophil traps may also contribute to tophi formation.

SYMPTOMS [8]



MANAGEMENT AND TREATMENT [1]

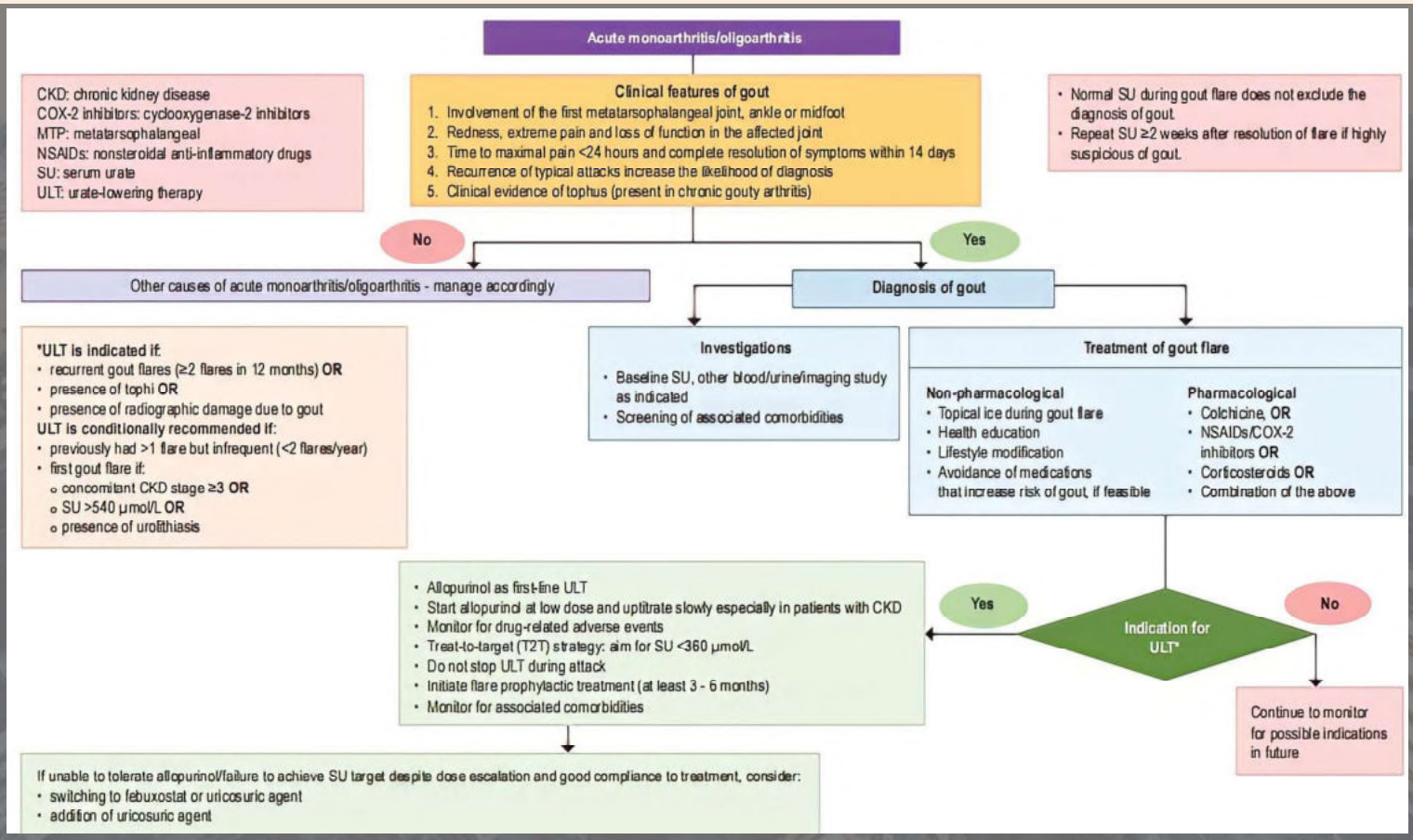
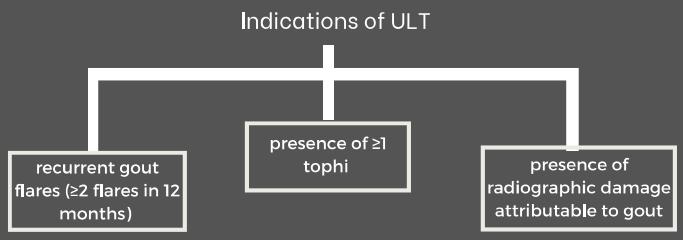


Figure 1 illustrates the algorithm on management of gout

INDICATIONS OF URATE LOWERING THERAPY (ULT) [1]



The American College of Rheumatology (ACR) tentatively recommends initiation of ULT in gout patients with a first gout attack based on the following indications:

- Moderate to severe chronic kidney disease (stage ≥ 3) or
- Serum urate level >9 mg/dL (540 µmol/L) or
- Urolithiasis

ACR also conditionally recommends initiating ULT in gout patients who have had at least one relapse in the past but infrequently (less than 2 times/year).

PHARMACOLOGICAL TREATMENT

URATE LOWERING THERAPY[1,9,10,11,12,13,14]

Medication	Mechanism of action	Dose	Possible side effects
Allopurinol	A purine analogue of xanthine oxidase that metabolises in the liver to transform into pharmacologically active metabolite, oxypurinol. It inhibit xanthine oxidase, an enzyme in the purine catabolism pathway that converts hypoxanthine to xanthine to uric acid. It is subjected to being metabolized by many enzymes involved in purine and pyrimidine synthesis metabolism.	**Initial**: 100 mg/day, 100 mg of dosage increments every 2-4 weeks based on SU concentration until the target is achieved. **Maintenance**: ≥300 mg/day, up to 900 mg/day. **Frequency**: Once daily in a single dose or in 2 or 3 divided doses if >300 mg/day	**Common**: Maculopapular rash, pruritus, nausea, vomiting. **Serious**: Hypersensitivity reactions ranging from mild maculopapular rash to severe cutaneous adverse reaction [Stevens-Johnson syndrome (SJS), Toxic Epidermal Necrolysis (TEN), drug reaction with eosinophilia and systemic symptoms (DRESS)]
Probenecid	It promotes disease-causing uric acid clearance by inhibiting renal tubular transporter and blocking reuptake.	**Initial**: 250 mg BD for 1 week; may titrated to 500 mg BD; if needed, may increase to a max of 1000 mg BD (500 mg increment of dosage in every 4 weeks)	**Common**: Rash, nausea, vomiting. **Serious**: SJS, aplastic anaemia, leukopenia, thrombocytopenia, neutropenia, hepatic necrosis, anaphylaxis, hypersensitivity reaction
Febuxostat	A potent, non-purine selective inhibitor of XO, forming a stable complex with both the reduced and oxidized form of the enzyme, thereby halting its function. It does not interfere with the activity of other enzymes involved in purine or pyrimidine synthesis or metabolism.	**Initial**: 40 mg OD; if SU level is >360 µmol/L after 2-4 weeks, consider titrating to 80 mg OD. **Maintenance**: 40 mg or 80 mg OD, dose may be increased to 120 mg OD if clinically indicated	**Common**: Rash, diarrhea, nausea, liver function abnormalities **Serious**: DRESS, SJS, TEN
Benzbromarone	An uricosuric drug that increases urate excretion in the renal proximal tubule by inhibiting the predominant apical urate exchanger in the human proximal tubule URAT1. This blockade reduces the reabsorption of uric acid and increases its excretion in the urine.	**Usual dose:** 50 - 100 mg/day. Doses of 50 - 200 mg daily may be used	**Common**: nausea, vomiting, diarrhea, liver damage
Pegloticase	A pegylated recombinant uricase (urate oxidase) produced by genetically engineered mutants of Escherichia coli that catalyzes uric acid to the water-soluble purine metabolite allantoin to be renally excreted, thus helping treat chronic gout. It does not block the formation of uric acid.	Infused 8 mg every 2 weeks	**Common**: Urticaria, constipation, nausea, vomiting **Serious**: Infusion-related reaction

GOUT FLARES TREATMENT AND PROPHYLAXIS [1]

Gout attacks should be treated promptly and appropriately. For gout attacks, the following monotherapy can be used:

- Colchicine
- Nonsteroidal anti-inflammatory drugs/cyclooxygenase-2 inhibitors
- Corticosteroids

Gout attack prophylaxis should be given for at least 3 to 6 months when uric acid-lowering therapy is initiated. A preferred option is dose escalation of urate-lowering therapy and/or co-administration of colchicine.

GOUT FLARES TREATMENT AND PROPHYLAXIS: COLCHICINE [1,7]

Medication	Mechanism of action	Dose	Possible side effects
Colchicine	Colchicine modulates multiple pro- and anti-inflammatory signaling pathways associated with gouty arthritis. It interferes with microtubule formation, thereby interfering with inflammasome activation, microtubule-based chemotaxis of inflammatory cells, formation of leukotrienes and cytokines, and phagocytosis.	**Gout flares** **Initial dose:** 1 mg, then 0.5 mg after 1 hour. Do not take more tablets for 12 hours. After 12 hours, treatment can be resumed as needed with a max dose of 0.5 mg every 8 hours until symptoms are resolved. Discontinue when symptoms subside or when a total of 6 mg has been taken. After completing a course, do not start any further courses for at least 72 hours. **Flare prophylaxis;** 0.5 mg OD or BD. Prophylactic therapy might be beneficial for at least the first 3-6 months of ULT therapy. **Treatment of gout flare during prophylaxis with colchicine** Do not exceed 1 mg at the first sign of flare, proceed to take 0.5 mg 1 hour later, and wait for 12 hours before resuming the prophylactic dose. Prophylaxis should begin at least 12 hours after treatment. Continue taking this medicine until the gout attack subsides.	**Common**: Nausea, vomiting, diarrhea **Serious**: Myelosuppression, neuromuscular disease, neuromyotoxicity

GOUT FLARES TREATMENT AND PROPHYLAXIS: NSAIDs [1, 15, 16]

Medication	Mechanism of action	Dose	Possible side effects
Ibuprofen Diclofenac Naproxen	Nonselective cyclooxygenase (COX) inhibitors inhibit indifferently all the COXs isoforms. It inhibits COX or prostaglandin synthase and affects the final conversion of arachidonic acid to prostaglandins, prostacyclins, and thromboxanes. The inhibition of COX-1 is linked with gastric irritation, as the prostaglandins produced by COX-1 in the gastric epithelium act as cytoprotective agents	**Ibuprofen**: 400-800 mg TDS (maximum: 3200 mg/day) **Diclofenac**: 50 mg BD/TDS **Naproxen**: 550-1100 mg in 2 divided doses (275 mg tablet); 750 mg initially, then 250 mg TDS (250 mg tablet)	**Common**: indigestion including stomach aches, feeling sick and diarrhea, stomach ulcers that can cause internal bleeding and anemia, headaches, drowsiness. dizziness, allergic reactions.
Meloxicam Etoricoxib Celecoxib	Selectively inhibits COX-2 that is responsible for inflammation and exert anti-inflammatory effects without affecting the gastric mucosa. Its expression increases during inflammatory conditions in response to mitotic stimuli.	**Meloxicam**: Maximum 15 mg/day **Celecoxib**: 400 mg stat followed by 200 mg BD subsequently **Etoricoxib**: 120 mg/day	**Common**: Insomnia, abdominal pain, fatulence (gas), headache, nausea, diarrhea

GOUT FLARES TREATMENT AND PROPHYLAXIS: CORTICOSTEROIDS [1, 17, 18]

Medication	Mechanism of action	Dose	Possible side effects
Prednisolone	Glucocorticoids exert their anti-inflammatory effects through various mechanisms. • stimulate the production of lipocortin to inhibit phospholipase A2 thereby reducing the production of arachidonic acid, and reducing the synthesis of inflammatory mediators such as prostaglandins, leukotrienes and platelet-activating factor; • inhibit the synthesis and release of cytokines (IL-1, IL-4, IL-6, and TNF-α), suppressing T cell activation and fibroblast proliferation, thereby reducing chemotaxis process. • inhibit pro-inflammatory transcription factors such as nuclear factor-κB and activating proteins and reduces transcriptional enhancement of genes encoding COX-2, cytokines, and nitric oxide synthase (iNOS)	**Gout flares** 30 to 40 mg/day once or twice daily for 5 days. A gradual taper over 7-10 days is an option if longer periods are required for greater flares. A slower taper (eg, 14–21 days or longer) may be required, especially in patients with multiple recent attacks.	**Common**: Body fluid retention, hypertension, acne, gastrointestinal bleeding, decreased body growth, hyperglycemia, osteoporosis, headache
Triamcinolone		**Intra-articular** **Large joint:** 40 mg as a single dose, **Medium joint:** 30 mg as a single dose, **Small joint:** 10 mg as a single dose **Intramuscular**: 40-80 mg as a single dose; Can be repeated at intervals of 48 hours or longer if flare resolution does not occur	**Common**: Cough, sinusitis, bruise, joint swelling

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DRUG UPDATES

Oxycodone HCL 1mg/ml Oral Solution

A. DESCRIPTION

Oxycodone is an opioid used in the management of moderate to severe pain. It binds to opiate receptor in the central nervous system (CNS), causing inhibition of ascending pain pathways.

B. REGISTRATION NUMBER

MAL13025039AZ

C. PRICE

RM 42.40/bottle of 250ml

D. DEPARTMENT

Palliative Unit

E. PRESCRIBER CATEGORY

A* (Consultant/Specialist for specific indications only)

F. INDICATION IN FUKKM

As a second-line drug in the management of responsive, moderate to severe pain in patients who:

i. have difficulty swallowing or
ii. require a low dose oxycodone (<5mg).

G. DOSE AND ADMINISTRATION

Initial dose for opioid naïve patients or patients presenting with severe pain uncontrolled by weaker opioids is 5 mg, 4-6 hourly.

The dose should then be carefully titrated, as frequently as once a day if necessary, to achieve pain relief. Maximum daily dose is 400mg daily.

H. MECHANISM OF ACTION

Oxycodone and its active metabolites can selectively bind to the mu opioid receptor, but also the kappa and delta opioid receptors in the central nervous system and periphery and induce a G protein-coupled receptors (GPCRs) signaling pathway. GPCRs are important mediators of pain or analgesia. Activation of mu opioid receptors inhibit N-type voltage operated calcium channels, inhibiting responses to pain.

I. ADVERSE REACTIONS

- **Metabolism and nutritional disorders:** Anorexia
- **Psychiatric disorders:** Anxiety, depression, insomnia, nervousness
- **Nervous system disorders:** Headache, dizziness, somnolence, tremor, lethargy, sedation
- **Respiratory, thoracic and mediastinal disorders**: Dyspnea, cough, respiratory depression
- **Gastrointestinal disorders**: Constipation, nausea, vomiting, dyspepsia, abdominal pain, diarrhea
- **Skin and subcutaneous tissue disorders:** Pruritus, hyperhidrosis, rash

DRUG UPDATES

J. CONTRAINDICATION

- Respiratory depression
- Known or suspected paralytic ileus
- Acute abdomen
- Delayed gastric emptying
- Chronic obstructive pulmonary disease
- Cor pulmonale
- Acute or chronic bronchial asthma
- Hypercarbia
- Chronic constipation
- Concurrent admin of MAOIs or within 2 weeks of discontinuation of use
- Moderate to severe hepatic and severe renal impairment
- Lactation

K. USE IN SPECIFIC POPULATION

- **Adults with mild to moderate renal impairment and mild hepatic impairment**: the plasma concentration in this patient population may be increased. Therefore, the starting dose for opioid naïve patients is 2.5 mg, 6-hourly.

- **Children under 18 years:** should not be used in patients under 18 years.

- **Pregnancy:** not recommended for use in pregnancy nor during labor. There are limited data from the use of oxycodone in pregnant women. Regular use in pregnancy may cause drug dependence in the fetus, leading to withdrawal symptoms in the neonate.

- **Lactation:** may be secreted in breast milk and may cause respiratory depression in the newborn. Therefore, it should not be used in breast-feeding mothers.

L. PRECAUTION

- Patient with raised intracranial pressure, hypotension, hypovolemia, toxic psychosis, biliary tract diseases, pancreatitis, inflammatory bowel disorders, prostatic hypertrophy, adrenocortical insufficiency, history of drug abuse or acute alcoholism, delirium tremens, thyroid dysfunction.

- Mild to moderate renal impairment.

- Mild hepatic impairment.

- Pregnancy.

M. STORAGE

Store between 15-30 C. Protect from light

N. PHARMACIST ROLE

- Counsel patient/caregiver to report breathing difficulties or symptoms of respiratory depression such as shortness of breath, slow or shallow breathing and tiredness.

- Advise patient to inform the doctor if planning to get pregnant before taking the medication.

- Advise patient to avoid drive or operate machinery as this medication can cause drowsiness.

- Advice patient do not stop taking the medication, exceed the dose recommended or change the dosage without checking with doctor or pharmacist.

O. REFERENCES

Product information leaflet, FUKKM, QUEST3+, MIMS Gateway, Medscape, Drugbank.com

By: Nurul Hazirah Aminudin

DRUG UPDATES

VEDOLIZUMAB 300MG, POWDER FOR CONCENTRATE FOR SOLUTION FOR INFUSION

DESCRIPTION

Vedolizumab is an integrin receptor blocker and anti-inflammatory agent used to manage ulcerative colitis and Crohn's disease in adults with inadequate clinical response to immunomodulators.

REGISTRATION NUMBER

MAL17025031ACZ

PRICE

RM 11,870.00/vial

DEPARTMENT

Gastroenterology

PRESCRIBER CATEGORY

A* (Consultant/Specialist for specific indications only)

MECHANISM OF ACTION

Vedolizumab is a gut-selective immunosuppressive biologic. It is a humanized monoclonal antibody that specifically binds to the α4β7 integrin and blocks the interaction of α4β7 integrin with mucosal address in cell adhesion molecule-1 (MAdCAM-1) and inhibits the migration of memory T-lymphocytes across the endothelium into inflamed gastrointestinal parenchymal tissue, thereby preventing lymphocytic cells from entering the gut lamina propria and gut-associated lymphoid tissue (GALT). Specifically inhibiting this pathway alleviates GI inflammation without impairing systemic immune responses.

INDICATION IN FUKKM

i) Indicated for the treatment of adult patients with moderately to severely active ulcerative colitis who have had an inadequate response with, lost response to, or were intolerant to tumour necrosis factor-alpha (TNF-α) antagonist.

ii) Indicated for the treatment of adult patients with moderately to severely active Crohn's disease who have had an inadequate response with, lost response to, or were intolerant to tumour necrosis factor-alpha (TNFα) antagonist.

DOSE AND ADMINISTRATION

300 mg administered by intravenous infusion at zero, two and six weeks and then every eight weeks thereafter.

USE IN SPECIFIC POPULATIONS

Paediatric: The safety and efficacy of Vedolizumab in children aged 0 to 17 years old have not been established.

Elderly: No dose adjustment is required in elderly patients. Population pharmacokinetic analyses showed no effect on age.

Impaired renal or hepatic function: Vedolizumab has not been studied in patients with renal or hepatic impairment.

Pregnancy: Vedolizumab should be used during pregnancy only if the benefits to the mother are considered to outweigh the risk to the unborn child.

Lactation: Vedolizumab has been detected in human milk. The effect of Vedolizumab on infants is unknown. The use of Vedolizumab in lactating women should take into account the benefit of therapy to the mother and potential risks to the infant.

DRUG UPDATES

ADVERSE REACTIONS

Common

- **Gastrointestinal**: Nausea (9%)
- **Musculoskeletal**: Arthralgia (12%)
- **Neurologic**: Headache (12%)
- **Respiratory**: Nasopharyngitis (13%), upper respiratory infection (7%)
- **Other**: Fatigue (6%), fever (9%)

Serious

- **Hepatic**: Aminotransferase abnormal increased (<2%), hepatitis, serum bilirubin raised
- **Immunologic**: Anaphylaxis (0.07%), hypersensitivity reaction, infusion reaction (4%)
- **Neurologic**: Progressive multifocal leukoencephalopathy
- **Respiratory**: Tuberculosis
- **Other**: Cancer (0.4%), infectious disease, sepsis (0.03%)

PRECAUTIONS

Infusion-Related Reactions (IRR) and Hypersensitivity Reactions: In clinical studies, IRR and hypersensitivity reaction have been reported with majority being mild to moderate in severity.

Infection: Physician should be aware of the potential increased risk of opportunistic infections or infections for which the gut is a defensive barrier. Patient should be monitored closely for infections before, during and after treatment.

Malignancies: The risk of malignancy is increased in patients with ulcerative colitis and Crohn's disease.

Vaccination: Prior to initiating treatment with Vedolizumab all patients should be brought up to date with all recommended immunizations. Patients receiving Vedolizumab receive non-live vaccines (e.g., subunit or inactivated vaccines) and may receive live vaccines only if the benefits outweigh the risks.

CONTRAINDICATIONS

Hypersensitivity to the active substance or to any of its excipients.

STORAGE

Store in a refrigerator (2°C-8°C). Keep the vial in the outer carton in order to protect from light.

PHARMACIST ROLES

- Advice patient to not drive or operate machinery as this drug may cause mild dizziness.
- Counsel patient to immediately report new or worsening neurological symptoms indicative of progressive multifocal leukoencephalopathy such as unilateral weakness, visual disturbances, change in cognition and memory associated with confusion and personality changes.
- Advice patient to report immediately if experiencing any of the following symptoms:
  - rashes, breathlessness, swelling of the face, eyes or mouth
  - blurred, loss of or double vision, difficulty speaking, persistent numbness, memory loss or confusion, facial drooping
  - difficulty breathing, wheezing, flushing, rash, hives, fast heartbeat

REFERENCES

Product leaflet, MIMS, FUKKM, rxlist.com, Medscape, Micromedex.

BY: NUR AMEERA BT MUSLIM

PHARMACY R&D

Deferoxamine Injection Practice Among Thalassemia Patients

AUTHOR: Muhammad Nasri bin Yusoff, Toh Kit Yeng, Chua Peck Wei, Nurul 'Izzati Liyana binti Azlan, Atikah binti Ali @ Jaafar

INTRODUCTION

Thalassaemia is a hereditary blood disorder characterized by defective synthesis of globin chains, which results in haemolysis and impaired erythropoiesis. Routine blood transfusion is required, which may lead to iron overload in a long run. Deferoxamine (DFO) provides effective iron chelation, however, there were challenges faced by the patients to administer it.

OBJECTIVES

- Identify the current DFO administration practices among thalassaemia patients
- Common administration errors
- Patient compliance towards iron chelation therapy
- Factors associated with ferritin levels

METHODOLOGY

A cross-sectional study was done in Hospital Tengku Ampuan Afzan, included all patients using DFO from January 2019 until November 2020. Patients were assessed on DFO injection practices, dilution steps, and compliance. DFO injection practices were evaluated by using DFO score, and compliance was assessed by using compliance score.

RESULTS

A total of 40 patients were obtained from the registry, however only 24 patients were able to complete the study. Median age 24.5 years old, 58.3% not working, 80% were Hb E β-Thalassaemia, 91.7% were transfusion-dependent, 16% had severe cardiac iron overload, and 25% had severe liver iron overload. Median serum ferritin was 3361.4 µg/L. For DFO errors, 91.7% occurred during administration. Overall, 75% of the patients had good DFO score, however, compliance rate was 29.2%. Working status (-5557.06, 95% CI -9549.12 to -1655.01) and frequency of missed dose (1152.39, 95% CI 382.18 to 1922.6) were identified as factors that associated with ferritin level.

CONCLUSIONS

As a conclusion, a few errors were identified and highlighted in DFO administration. Dilution error was the most common error among thalassemia patients. Although most of the patients had good DFO administration practice, overall compliance rate was low. Working status and patient compliance were identified as factors that may affect serum ferritin level. The findings of this study may provide information for further interventions to educate patients on DFO administration and patient compliance.

PHARMACY R&D

Analgesic Drug Utilization in Orthopaedic Ward

AUTHOR: Chan Mei Fong @ Sohbana, Nur Izzati Dhamirah bt Mohd Yusof, Logene A/P Somasundram, Najah Nadhiera Syazwanie bt Syahrul Anwar Young

INTRODUCTION

Management of pain in the field of orthopaedic surgery incorporating analgesia and opioid has become increasingly relevant as a result of efforts to improve postoperative recovery, minimize hospital stay and reduce morbidity and mortality.

OBJECTIVES

This study aimed to determine the analgesic drug utilization after surgery among orthopaedic in-patients. We also explored the effects of various analgesic drugs on pain control at various post-operative time intervals.

METHODOLOGY

Participants were recruited from orthopedic ward in Hospital Tengku Ampuan Afzan from January until December 2020. Drug utilization was quantified by using the World Health Organisation (WHO) Anatomical Therapeutic Chemical classification (ATC)/Defined Daily Dose (DDD) methodology and DDD per 100 bed days were applied to determine drugs that were utilized by in-patients. Repeated measure of analysis of variances (ANOVA) was used to compare pain scores and analgesia in different post-operative time intervals.

RESULTS

A total of 123 participants were recruited in the study from orthopedic ward. Tablet paracetamol was the most commonly prescribed analgesia where the DDD/100 beds days was 79.04 followed by intravenous tramadol and capsule celecoxib where DDD/100 beds day were 28.48 and 25.34 respectively. The least prescribed drug was intravenous paracetamol which DDD/100 beds days was 0.14. In this study, between 6 and 12 hours post-operative period showed significant increase of pain score by an average of 0.390 units which is approximately 21% increase ($p=0.007$) from mean pain score 1.818 among patient that received combination of paracetamol and opioid group. Besides that, it was noted that mean pain score post-operative among treatment group at different time intervals showed were less than 4.

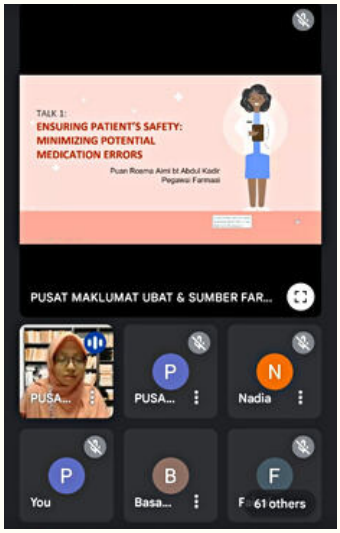
CONCLUSIONS

Paracetamol, tramadol and celecoxib were the common drug used in orthopaedic ward which identify the prescribing pattern of surgeon in this setting. Hence, improve estimation of drug supply in the future and cost estimation of analgesia. Besides that, the overall study conclude that most patient received adequate pain control post-operative (pain score <4) This result is crucial for surgeon to anticipate there will be increase of pain in between 6 and 12 hours post-surgery among patients. The limitation of this study did not take into account the complexity of surgery and mode of anaesthesia.

PHARMACY ACTIVITY

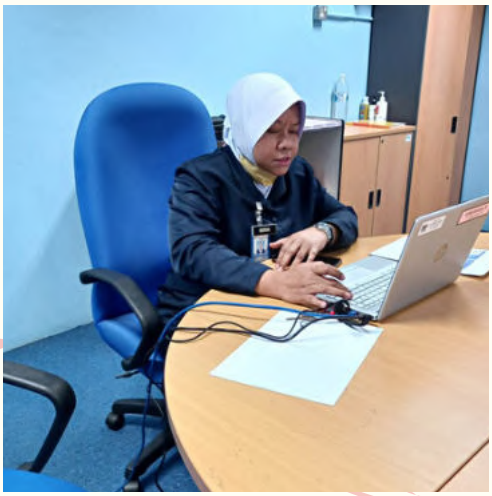
Kursus Medication Safety 2022

Google Meet | 28th September 2022

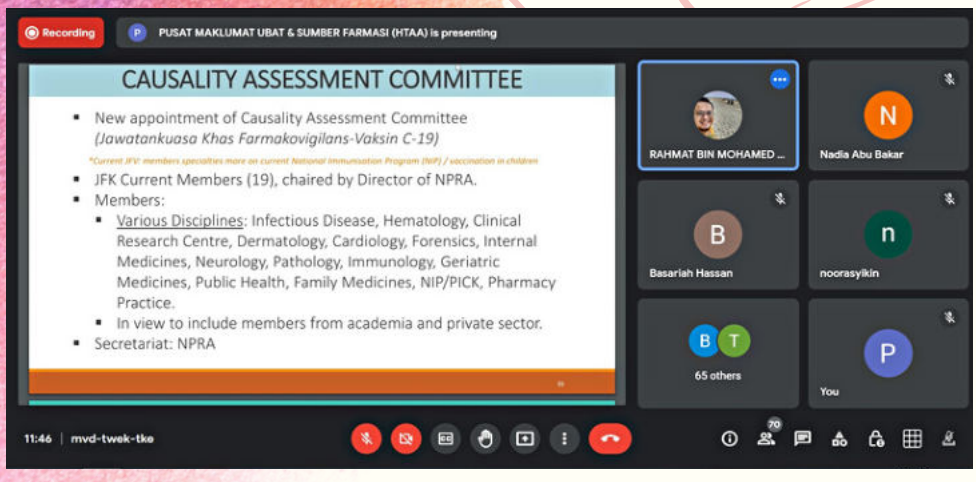


Session 1 -
Ensuring Patient's Safety: Minimizing Potential Medication Errors
by
Pn. Rosma Aimi Abdul Kadir

Session 2 -
Pengurusan Pengubatan dan Penyimpanan Ubat
by
Pn. Zawiah Ahmad

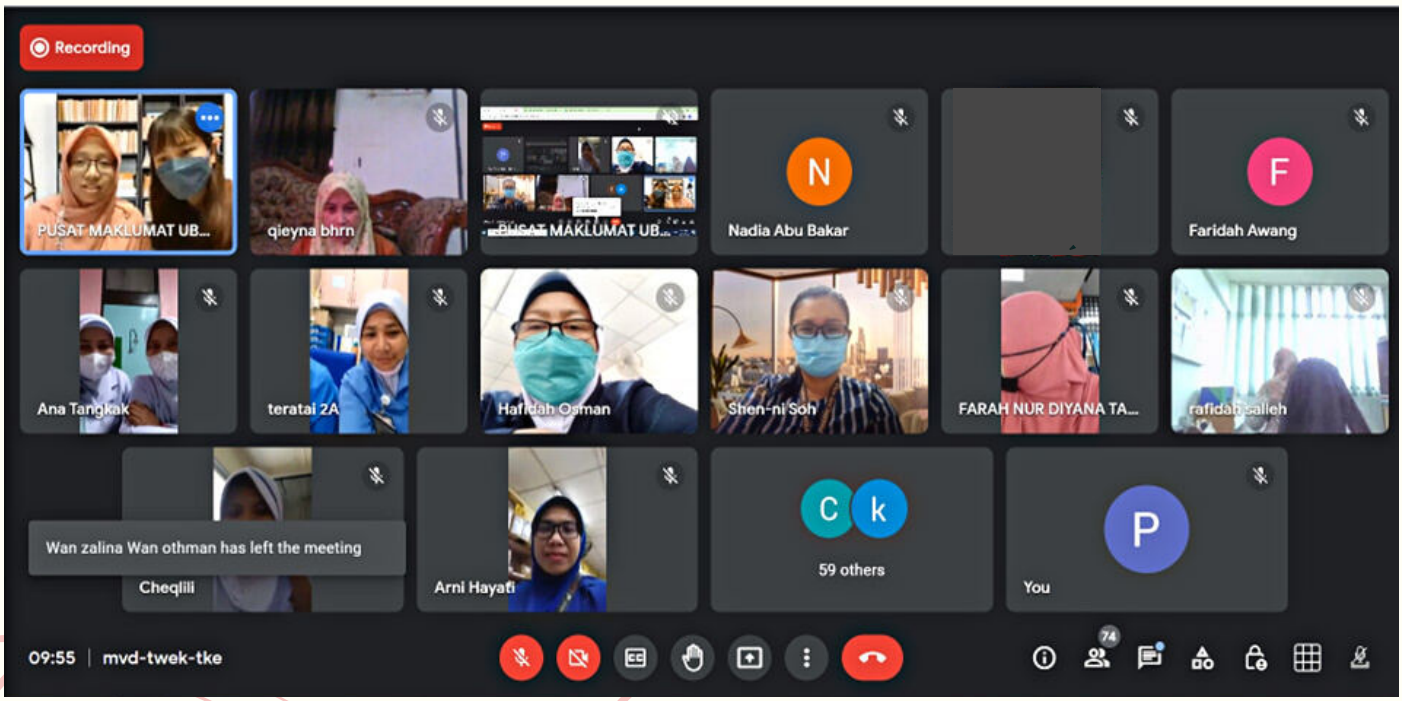


Session 3 -
Safe Practice in Medication Administrtaion
by
Matron Nurha Wahab@Salleh



Session 4 -
Adverse Events Following Immunization (AEFI); Discover the Unknown
by
En Rahmat bin Mohamed Tahir
(JKN Pahang)

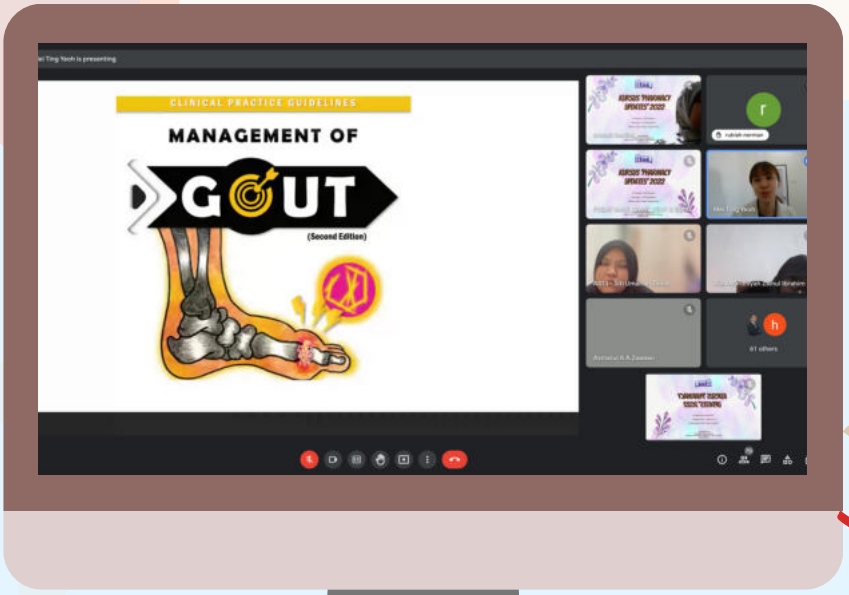
Session 5 -
Ubat dan Kosmetik: Racun dalam Penawar
by
Pn. Wong Li Yee
(JKN Pahang)



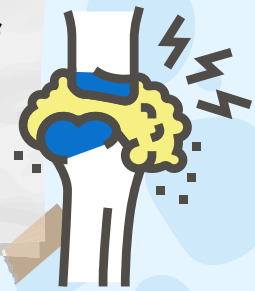
Photography Session

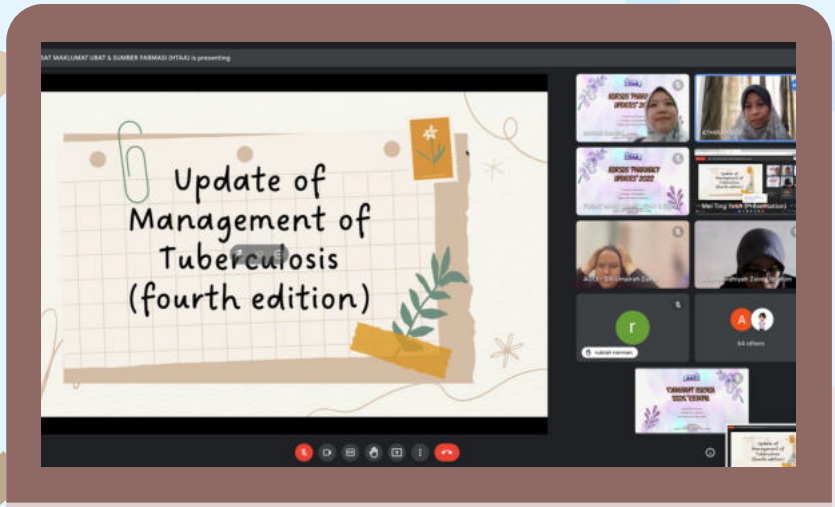
KURSUS PHARMACY UPDATES 2022

Google Meet | 15th October 2022

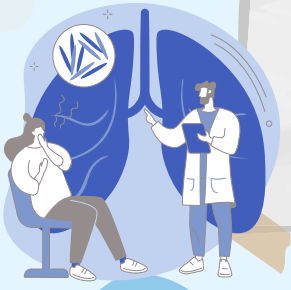


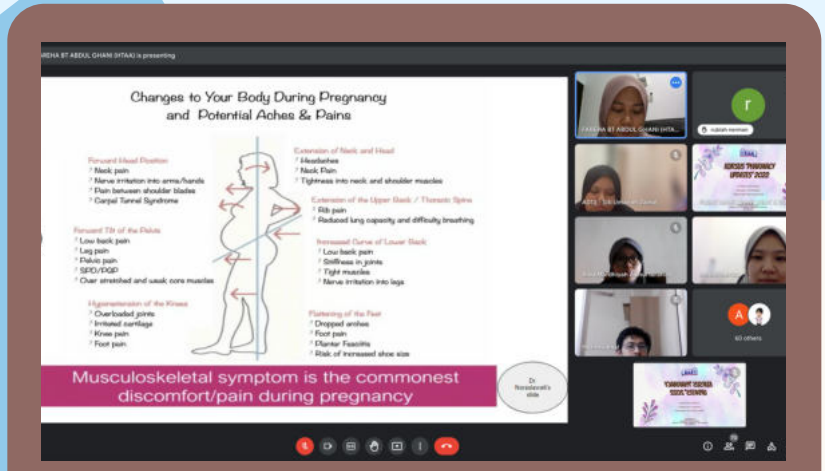
Session 1:
Updates on CPG Management of Gout by Pn. Yeoh Mei Ting





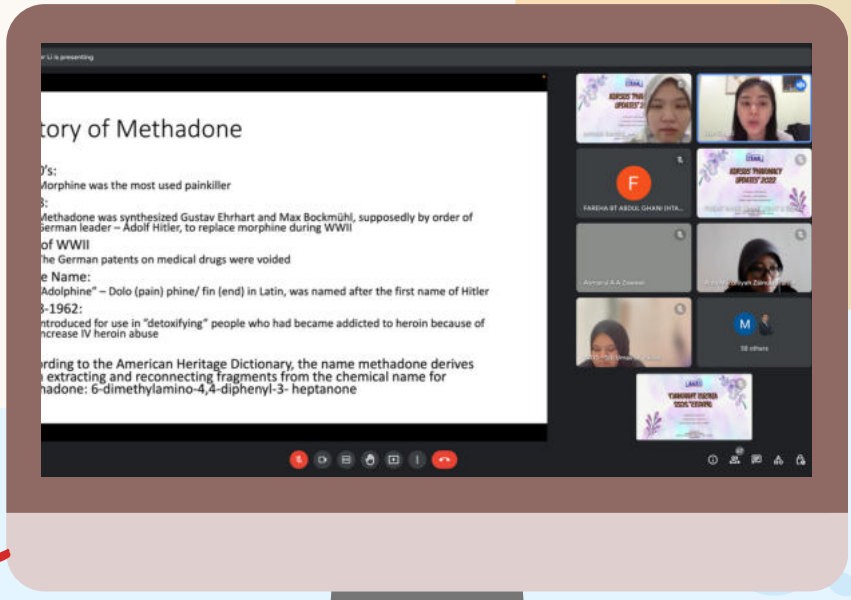
Session 2:
Updates on CPG Management of Tuberculosis by Pn. Nurul Athirah Abd Aziz





Session 3:
Analgesia in Pregnancy by Pn. Fareha Abdul Ghani

Session 4:
Updates on Methadone for Pain by Pn. Han Ser Li

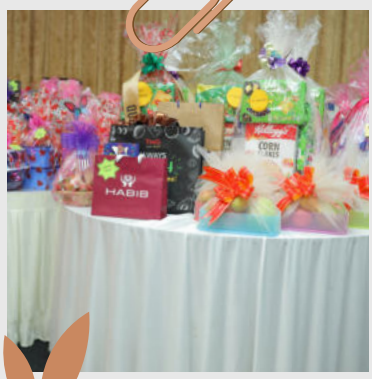


Session 5:
Updates on CPG Management of Chronic Heart Failure by Pn. Ainul Mardhiyyah Zameram

Photography Session!

PharmNight

15th December 2022
CMI Diamond Hall, Berjaya Megamall Kuantan



Registration

Welcoming Speech from
Pn. Hjh. Samehah Almuna

PharmCare
Hospital Tengku Ampuan Afzan

Opening Gimmick!

Committees

Welcoming Speech from Dr. Mastura

EKSA Prize Giving (2nd Place)

EKSA Prize Giving (1st Place)

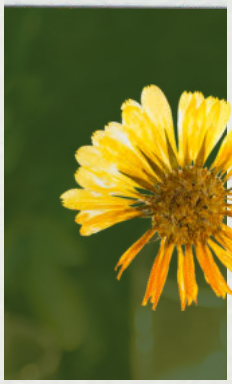
Prayer Recitation

Dance Performance

Lucky Draw Session

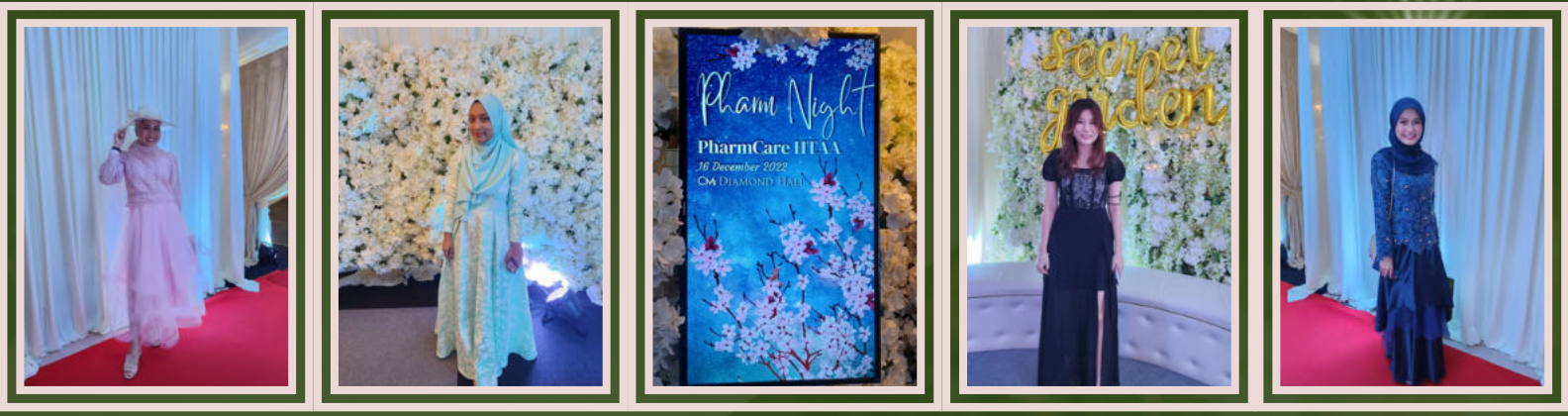
Lucky Draw Grand Prize Winner


garden




garden

Photography Session



PharmNight Best Dressed

(Winners & Nominees)

BEST DRESSED (MALE)

EN. SHAFF KAMIL

BEST DRESSED (FEMALE)

PN. JURAINI

PHARMNIGHT KARAOKE BATTLE

En. Harizul Amri

Pn. Mimi Nurul Syafilla

En. Muhammad Muhaimin

JABATAN FARMASI HTAA ACHIEVEMENTS 2022

Pertandingan Quality Assurance (QA) Peringkat JKNP 2022

19 MAY 2022 | AUDITORIUM INSTITUT LATIHAN KEMENTERIAN KESIHATAN MALAYSIA, KAMPUS KUANTAN

Kategori Pembentangan Oral (Kumpulan *Iron Superhero*)

Konvensyen Inovasi 2022

27 - 28 JULY 2022 | JABATAN KESIHATAN NEGERI PAHANG

2nd Place - Kategori Proses / Perkhidmatan / Teknologi

Project Name:
ASSIST

Konvensyen Kumpulan Inovatif dan Kreatif (KIK) 2022

25 AUGUST 2022 | AUDITORIUM ARKIB NEGARA PAHANG

3rd Place

Project Name: Quick-M

Pertandingan Tayangan Video Peringkat Hospital Kluster - World Patient Safety Day 2022

15 SEPTEMBER 2022 | AUDITORIUM HTAA

2nd Place: Farmasi Satelit

Konvensyen Kualiti, Inovasi dan Research & Development Farmasi Negeri Pahang (CONQRD) 2022

27 AUGUST 2022 | HOTEL ANCASA ROYALE PEKAN

HTAA for Overall Winner!

Oral Presentation R&D Category

[1st Place]
Pn. Yasmin Ibrahim

Poster Presentation QA Category

[2nd Place]
En. Tan Yaw Shen
En. Raiz Rasyid Mohamed Iqubal

Innovation Category

[BEST EXHIBITOR]
Quick-M

[1st Place]
Quick-M

Pn. Noor Wahida • Pn. Aryani • Pn. Siti Husna Izzati • En. Ahmad Bistari • Pn. Nurul Athirah • Pn. Shahida • Pn. Sayidah Nafisah • Pn. Noor Fatin Hanani

[3rd Place]
ASSIST

Pn. Tou Pui Yee • Pn. Hawa • Pn. Fareha • Cik Terrina Lim Ju Ann • En. Muhammad Arif

Poster R&D Category

[BEST POSTER]

- **Pn. Immiratul Saadiah Mohd Saat**
- **Cik Fong Swiit Xin**

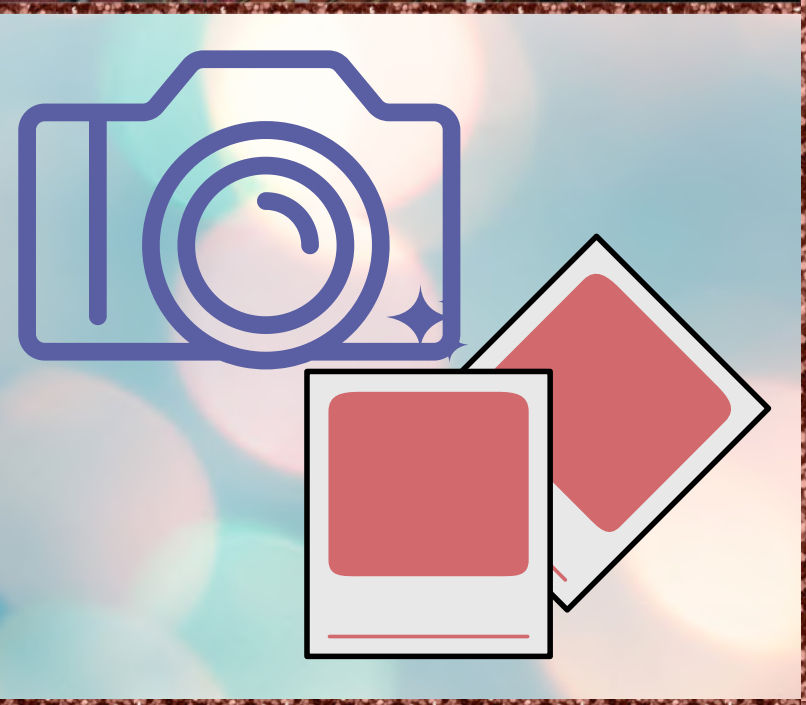
[1st Place]

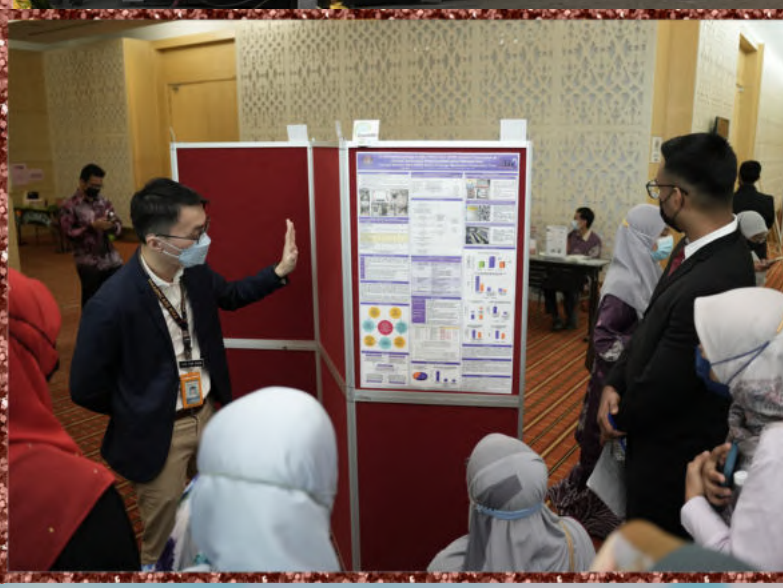
- **Pn. Immiratul Saadiah Mohd Saat**
- **Cik Fong Swiit Xin**

[2nd Place]

- **En. Muhammad Nasri Yusoff**

TEAM HTAA IN ACTION!
@ CONQRD '22





Pahang Research Day 2022

11 OCTOBER 2022 | AUDITORIUM HTAA

Oral Presentation Category

1st Place: En. Muhammad Nasri Yusoff

2nd Place: Pn. Yasmin Ibrahim

Consolation: En Muhammad Nasri Yusoff

Poster Presentation Category

Consolation: Pn. Amnah Berdal

Consolation: En. Khairul Naim Zainal Abidin

National Conference for Clinical Research 2022

18 - 20 OCTOBER 2022 | THE WEMBLEY A ST GILES, PENANG

Congratulations to Dr Mastura Ahmad 5th out of 78 in Poster Category!

Winner of Dr Wu Lien-Teh Research Award (Consolation)

Every moment is an opportunity for a fresh beginning

Welcome 2023!