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VISION

To emerge as one of the premier pharmacy colleges in the country and produce pharmacy professional of global Standards.

MISSION

- To deliver quality academic programs in Pharmacy and empower the students to meet industrial standards.
- To build student community with high ethical standards to undertake R&D in thrust areas of national and international standards.
- To extend viable outreach programs for the health care need of the society.
- To develop industry institute interaction and foster entrepreneurial spirit among the gradutes

NEEM OIL POISONING

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Pharm D II nd Year

Case report:

Abstract :

We report a case of neem oil poisoning in a previously normal 6year old child. The child presented with status vomiting after milk consumption which was mixed with neem powder.

KEYWORDS:Neem oil, Poisoning,Status vomiting.

INTRODUCTION :

Neem(Azardiracta indica) tree is traditionally known as medicinal tree whose every part is beneficial for treatment of various conditions.Neem tree also known as margosa. It mostly found in India , Pakistan, Bangladesh .Neem contain constituent like azardirachtin, nimbolinin , nimbin, salanin, gedunin ,quercetin[1]. Neem oil is extracted from neem seeds. Neem oil is used as pesticide and also in preparation of cosmetics such as soap, shampoos. Neem oil consumption is usually accidental, sometimes suicidal or may due to instillation for common cold in children as practiced in some parts of South India. Small doses of neem oil consumption are known to cause vomiting within minutes to hours, drowsiness, tachypnea .High doses cause metabolic acidosis along with seizures, toxic encephalopathy and also death common in childrens, rare in adults[2]. Case report: A 6 year old previously healthy boy was admitted in Adichunchangairi hospital and research centre,

- NagamangalaTaluk, Mandya district, Karnataka on 23/03/2023 at 7pm, later child was brought to Paediatric department following which baby developed fever .The patient was admitted with complaints of vomiting after milk consumption which is mixed with neem powder.
- On admission his glassgow coma scale was normal.The patient was conscious and experiencing tachypnea (>20cpm). Stomach wash had done.
- The management was started immediately, by giving Inj.Pantoprazole and Inj.Emeset,to control vomiting. On admission heamogram showed neutrophilic leucocytosis (WBC count- 18860cell/cum,Neutrophils -83%)and liver function test shows increased levels of SGOT (55U/L),Slight increase in ESR (21mm/hr and Serum electrolytes ,Creatinine, CRP are normal.
- The patient had no significant past medical history.on general physical examination it shows elevated levels of respiratory rate i.e 26cpm .remaining vitals are normal.Systemic examination shows umbilical appear central and everted. The toxicological report showed negative result on 24/03/2023. The patient was discharged on 24/03/2023 with discharge medication i.e lansoprazole and syrup
- ondansetron.Follow up after 4days showed improvement.

DISCUSSION :

Neem oil is extracted from the neem seed kernals. Neem oil is a mixture of steroids, triglycerides, palmitic acid, stearic acid and terpenoids along with small amount of aflatoxins[3]. The active ingredient in neem is azardiractin which shows pesticidal action, because it interferes with mitochondrial bioenergetics which results in inhibition of generation of electrochemical proton gradient. Acute poisoning with inhibitors of electron transporting complexes causes nausea, vomiting, muscle weakness, tachypnea.

Ingestion of high amount of neem oil causes toxic encephalopathy,Reye's syndrome, metabolic acidosis.Sinnaiah et al noted extensive mitochondrial damage in liver,proximal tubule of kidney and brain of mice that experimentally ingested neem oil[4].They later on reported many cases Reye's syndrome in children and infants with neem oil ingestion[5].Animal studies performed on mice shows symptoms such as salivation, diarrhoea,tremors, convulsions and even death to detect neem oil.

In our case, patient consumed small amount of neem powder with milk so it showed vomiting within 1hour. The severity of symptoms was dose dependent. Exact toxicity level doses for humans are not known. Laborotary reports shows mild hepatic toxicity with elevated levels of SGOT and leucocytosis this was probably due to viral respiratory tract infection. No specific antidote is available, but gastric lavage is recommended in our case with IV fluids. The treatment is primarily symptomatic. In our case mild vomiting is present which is far better than those with CNS symptoms. The outcomes are usually good.

CONCLUSION:

Neem oil poisoning is very rare. Accidental poisoning in children with powders and oils. Easy availability of neem oil and promotion of usage without proper warning of the life threatening side effects of neem oil.

about it will helps in early detection. Clinical features helps in diagnosis and increases survival rate of children.

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REVIEW ON NIPHA VIRUS INFECTION

M. Prathyusha, P.Himavarsha,

III Pharm D

INTRODUCTION:

Nipah virus (NiV) infection is a zoonotic ailment caused by a Henipavirus belonging to the Paramyxoviridae family. It is characterized by respiratory and neurological symptoms in humans, with bats of the Pteropus genus serving as the natural reservoir. The virus was first identified during an outbreak in Malaysia in 1988 and has since caused outbreaks primarily in Southeast Asia. The emergence of NiV is linked to factors such as habitat loss in bat populations.

Mode of Transmission: Transmission occurs primarily through the Pteropus-swine-human interface, consumption of contaminated food, and possibly direct bat-to-human contact. During outbreaks in pigs, control measures include culling infected animals, movement restrictions, and vaccination efforts.

EPIDEMIOLOGY:

The epidemiology of NiV varies across regions. In Malaysia, transmission to humans primarily occurs through infected pigs, while in Bangladesh, Pteropus bats serve as the reservoir. Outbreaks in India have shown higher mortality rates compared to Malaysia, with respiratory symptoms being more prevalent. Human-to-human transmission, including potential sexual transmission, has also been reported.

Clinical Presentation: Symptoms of NiV infection include fever, headache, respiratory distress, cough, sore throat, gastrointestinal symptoms, muscle pain, and weakness. Severe cases can progress to encephalitis, which is often fatal. The incubation period ranges from 4 to 14 days after exposure.

Preventive Measures/Treatment: Currently, there is no specific treatment or vaccine for NiV infection. Preventive measures focus on reducing exposure to bats, pigs, and infected individuals, particularly in outbreak areas.

CONCLUSION:

NiV remains a significant public health concern due to its high mortality rate and potential for human-to-human transmission. The development of targeted treatments and vaccines is hindered by a lack of comprehensive understanding of the disease's pathogenesis and immunology. Effective surveillance, coordinated international efforts, and conservation measures to preserve bat habitats are crucial for mitigating the impact of NiV outbreaks on global health.

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OJJAARA (MOMELOTINIB) THE NEWLY APPROVED DRUG FOR MYELOFIBROSIS PATIENTS WITH ANAEMIA

H. Shilpa and G. Mounika Priya

III Year Pharm D

Drug	: Ojjaara
Pronunciation	: Oh-JAR-uh
Generic name	: Momelotinib
Drug class	: Multikinase inhibitors, Antineoplastic agent
Dosage form	: Oral tablet
Dose	: 100mg,150mg,200mg
Brand name	: Ojjaara
Molecular formula	: C22H23N6O2
Molecular weight	: 414
Manufacturing company	: GlaxoSmithKline
Date of approval	: September 15,2023

INDICATION:

Ojjaara is JAK1/JAK2 and activin A receptor type 1 inhibitor. It is indicated for the treatment of intermediate or high risk myelofibrosis, including primary myelofibrosis or secondary myelofibrosis and post-essential thrombocytopenia, in adults with anemia.

MECHANISM OF ACTION:

OJJAARA is an inhibitor of wild type Janus Kinase 1 and 2 (JAK1/JAK2) and mutant JAK2V617F ,which contribute to signalling of a number of cytokines and growth factors that are important for hematopoiesis and immune function.

Momelotinib and it's major human circulating metabolite, M21 have higher inhibitory activity for JAK2 compared to JAK3 and tyrosine kinase 2.Momelotinib and M21 additionally inhibit Activin receptor like kinase 2,which produces subsequent inhibition of liver hepcidin expression and increased iron availability resulting in increased red blood cell production.

PHARMACOKINETICS:

Absorption -Effects of food: Not clinically significant
Tmax, oral: 2 hours
Distribution -Protein binding, plasma proteins: Approximately 91%
Volume of distribution: 984 L
Metabolism –Hepatic via multiple CYP450 enzymes, including CYP3A4 (36%), CYP2C8 (19%), CYP2C9 (17%), CYP2C19 (19%), and CYP1A2 (9%)
Excretion -Renal excretion, oral: 28%, less than 1% unchanged
Fecal excretion, oral: 69%, 13% unchanged
Total body clearance: 103 L/hr.
Elimination half-life: 4 to 8 hour

ADVERSE DRUG REACTIONS:

Thrombocytopenia
Hemorrhage
Bacterial infection
Fatigue
Dizziness
Diarrhea
Nausea

DRUG INTERACTIONS:

Concurrent of Momelotinib with Rosuvastatin may result in increased Rosuvastatin exposure and an increased risk of Rosuvastatin related adverse drug reactions.
Concurrent use of Momelotinib and Cyclosporine may result in increased Momelotinib exposure and an increased risk of Momelotinib related adverse reactions.

CONTRAINDICATIONS:

Lactating mothers and pregnancy women

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Departmental Activities in October - 2023

PERFECT CLICK

INTERNATIONAL DAY OF OLDER PERSON'S

WORLD'S ANIMAL WELFARE DAY, AT ANIMAL CARE LAND TIRUPATHI

INTERNATIONAL CONFERENCE ORGANIZATION BY NEW DELHI

SHCP FACULTY ATTENDED THE RESEARCH INNOVATION &BUILDING SKILS FOR PHARMACY EDUCATORS