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Review

- **"Paraquat Brain": Have we Researched Enough?**
  Ananth Rupesh KATTAMREDDY, Mohit Kumar Moses THATHAPUDİ, Victor GHOSH, Jacinth Karunya MİDDE, Pravin KALYANKAR

Case Report

- **Peripheral Facial Paralysis After COVID-19 BioNTech ® vaccine**
  Ali KARAKUŞ, Alper TAŞKIN, Anıl YOLDAŞ
- **Intracranial Bleeding In Methanol Intoxication Case**
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Editorial

Dear Readers,

We present to you the last issue of our journal for 2023. In this issue, we have published 1 review and 3 case reports that we think you will read with pleasure and interest. We hope that your scientific support will continue to increase in 2024. We would like to thank everyone who contributed to our journal for their support and contributions.

We would like to inform you that we will accept the articles on environmental emergencies starting from the first issue of 2024.

Best Regards.

Eurasian Journal of Toxicology Editorial Board

Contents

Review

1. Paraquat Brain": Have we Researched Enough? 25
Ananth Rupesh KATTAMREDDY, Mohit Kumar Moses THATHAPUDİ, Victor GHOSH, Jacinth Karunya MİDDE, Pravin KALYANKAR

Case Report

2. Peripheral Facial Paralysis After COVID-19 BioNTech ® vaccine 29
Ali KARAKUŞ, Alper TAŞKIN, Anıl YOLDAŞ
3. Intracranial Bleeding In Methanol Intoxication Case 31
Yeşim İŞLER, İrem FİDAN YOLAY, Halil KAYA, Melih YÜKSEL, Mehmet Oğuzhan AY, Umut OCAK

Review

Eurasian Journal of Toxicology

"Paraquat Brain": Have We Researched Enough?

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Abstract

Paraquat (PQ), a widely used herbicide, has gained ill repute due to its highly toxic nature and involvement in suicidal poisoning incidents, particularly in Asian countries. PQ-induced cellular toxicity is characterized by the generation of reactive oxygen species (ROS) and oxidative stress, leading to death. While research has primarily focused on the mortality associated with PQ poisoning, its morbidity implications, notably a potential link with Parkinson's disease (PD), remain underexplored by the scientific community.

Parkinson's disease, a neurodegenerative disorder, has been associated with the exposure to neurotoxicants resembling PQ, such as MPTP (1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine). Animal studies suggest that PQ may serve as a valuable tool for modelling PD. However, limited human autopsy data on PQ-related brain effects raise critical questions.

This paper discusses the need for comprehensive toxicopathological studies on PQ-exposed brains, focusing on the potential correlation between PQ exposure and PD. The results of such studies could offer valuable insights into this intricate relationship, impacting both public health policy and potential legal implications. Despite the absence of conclusive evidence linking PQ and PD, the scientific community must remain open to the possibility and commit to further dedicated research in this area.

Keywords: Parkinson's disease, neurotoxicants

Introduction

Paraquat (PQ, 1,1′-dimethyl-4-4′-bipyridinium dichloride) is a highly toxic quaternary ammonium nonselective herbicide commonly used in agricultural practices the world over[1]. The escalating incidences of poisoning related to paraquat (PQ) are a primary global concern, notably in India, where cases of suicidal ingestion are prevalent in rural areas owing to lax regulation by the state and the inexpensive nature of PQ[2]. Looking at it from a clinician's standpoint, two pivotal factors compound this issue. Primarily, there is an absence of a viable antidote, coupled with the potential for fatal consequences even with the ingestion of small quantities of PQ.

PQ induces cellular toxicity by generating reactive oxygen species (ROS) and impairing NADPH-linked cellular defense mechanisms against oxidative stress[3]. PQ has the propensity to accumulate more in the lung even as the concentration in body fluids tend to decrease. The polyamine transporters on the type I, type II, and Clara cells are responsible for this phenomenon and further toxicity stems from redox cycling and intracellular oxidative stress due to the generation of ROS[4]. The common causes of death associated with PQ poisoning as reported in the literature are oesophageal perforation or corrosive effects of the gastrointestinal tract (often associated with high concentration PQ 20%), multiorgan failure (hepatorenal failure), progressive proliferative lung damage, or paraquat lung[5].

Paraquat is rapidly absorbed through the gastrointestinal system, and it gets eliminated unchanged within 24 hours via urine. However, the observed clinical features are due to formation of intracellular Reactive Oxygen Species (ROS) which damages tissues by cellular lipid peroxidation, mitochondrial damage and kickstarting apoptotic changes leading to multi organ failure.

The clinical features of Paraquat poisoning are nausea, vomiting, epigastric pain, lesions over the mucosal lining of oral cavity, lethargy, and loss of consciousness. Upon endoscopy, lesions over the mucosal linings of pharynx and

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oesophagus, ulcerations, and inflammatory changes over the gastro intestinal tracts are observed. The simple diagnostic test at the bed side is dithionate test. Clinical chemistry shows leucocytosis, anaemia thrombocytopenia, elevated liver enzymes, raised serum creatinine and metabolic acidosis. Serum paraquat nomograms and Severeity Index of Paraquat Poisoning (SIPP) score are of practical interest for clinical toxicologists.

Due to the unavailability of specific antidote only supportive treatment can be done which includes gastric lavage, activated charcoal or sorbitol as adsorbent, and drugs such as metoclopramide, ranitidine, pantoprazole, soda bicarbonate and glucocorticoids (hydrocortisone, dexamethasone, cyclophosphamide, methyl prednisolone) for immune suppression. Haemodialysis is used as a last resort with less success[6].

While researchers are actively investigating PQ poisoning mortality and searching for anitdotes[7], there has been insufficient attention to its morbidity implications, notably its potential connection with Parkinson's disease (PD), which remains a matter of contention[8]. There are some systematic reviews indicating an association between PQ and PD, yet warranting further studies in this area[9]. Paraquat's toxicity closely resembles that of a neurotoxin MPTP (1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine), making it a valuable tool for inducing Parkinson's disease models in animal studies (safety evaluation studies in drug development)[10,11]. With the growing recognition of environmental pesticide exposure as a contributing factor to PD, researchers began to rigorously explore the potential connection between PQ and PD. The research in this domain encompasses exploring the structural, ultrastructural, and functional basis of PD upon exposure to PQ in animals[12].

Parkinson's disease is characterized by reduced dopaminergic neuron levels in the striatum, resulting in immobility and rigidity. It involves the degeneration of dopaminergic neurons in the substantia nigra and a decline in striatal dopamine content. The neurotoxicant MPTP (1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine) and PQ induce acute Parkinsonian syndrome by damaging neurons in the substantia nigra of mammals. Acting as a substrate for MAO-B(Monoamine oxidase B), MPTP crosses the Blood Brain Barrier (BBB) and transforms into the damaging MPP+(1-Methyl-4-phenylpyridinium). The acute and chronic toxicity exerted by MPP+ in causing acute Parkinson-like syndrome and a slow neurodegenerative disease is well documented in the literature. Notably, the latent manifestation of early PD symptoms following low-concentration MPTP exposure over time underscores the complexity of the disease progression, marked by the gradual loss of dopaminergic neurons in the substantia nigra. The striking parallelism between the neurotoxic effects of MPTP and certain pesticides, particularly paraquat, has raised a compelling flag of concern regarding the potential link between pesticide exposure and the development of Parkinson's disease (PD).

On gross examination of the brain in PD induced by MPTP and other toxicants, there was depigmentation in the substantia nigra and locus ceruleus, while the cortex, hippocampus, and amygdala showed relative preservation. On histopathology, alpha-synuclein-containing aggregates were observed in the medulla, contiguous brain regions, brainstem, limbic structures, and neocortex. Additionally, there's pallor in the substantia nigra and locus ceruleus, with Lewy bodies present in the locus ceruleus, dorsal motor nuclei of the vagus, cortex, and brainstem. Neuronal loss areas are accompanied by gliosis, and cortical Lewy bodies contain alpha-synuclein[13].

Human autopsy data of the PQ brain is extremely limited except for a few case reports published by toxicopathologists across the globe. In one study it was shown that the brain was edematous with or without focal minimal hemorrhages[14]. In another remarkable study, hemorrhagic leukoencephalopathy/ 'purpura cerebri' was noticed throughout the white matter of the brain. Further, it was associated with focal hemorrhages of various stages accompanied by demyelinating processes of different extents. Globular and/or amorphous proteinaceous materials were also seen in the vascular lumina throughout the brain[15].

During an autopsy in a case of paraquat poisoning, the brain revealed anoxic neuronal depletion and damage to the central white matter around the lateral and third ventricles. Electron microscopy showed edema and destruction of myelin with abundant myelin breakdown products, and astrocytic fibrous gliosis[16] .

In a fatal case of diquat poisoning (which shares a structural similarity with PQ), brain stem infarction, generalized hyperemia of the brain, and purpuric brain findings were reported histologically[17]. In another autopsy study following exposure to PQ, the brain showed intense congestion, and no changes attributable to anoxia were found[18].

According to a paper that published early histopathological changes in paraquat which compared human findings with animal studies, there was a proportionate increase in Virchow Robin spaces, indicating edema of the brain. Further, it was stated that the brain also showed substantial edema and hypoxic purpuric staining/mottling of the basal ganglia[19].

The plethora of brain findings presented herein offers a compelling impetus to earnestly pursue further research within this domain, with the prospect of yielding significant insights to turn some grey areas into black and white. Curiously, recent times have witnessed a dearth of autopsy studies on paraquat, raising questions about the motivations of the research community. Could it be that corporate interests are influencing researchers, or has the decline in

reported poisonings, potentially due to bans in the Western world, played a role?

In the context of India, a unique opportunity emerges to continue "*paraquat brain*" autopsy studies, providing a vantage point to explore and comprehend the intricate intersections of science, policy, and public health. The outcome of this research might have a lot of legal implications in the form of revision of PQ regulation and compensation lawsuits as well.

While autopsy results from fatal poisonings may not accurately reflect situations of prolonged unintentional exposure to neurotoxic substances in work or environmental settings, they still provide valuable insights for modeling. This assumes that data from animal experiments can effectively guide risk assessment in such a world. In a scenario where animal experimental data is considered dependable for human risk assessment, it is logical to also model data from human autopsies for similar purposes.

The field of toxicological research often fails to recognize the untapped arena of toxicopathological data garnered from human autopsy studies, despite its potential to serve as a pivotal link connecting the outcomes of animal studies to their applicability in human contexts. An illustrative example lies in a thorough toxicopathological examination of the brain in fatal human poisonings involving paraquat, which can offer invaluable insights for investigating the intriguing correlation between paraquat exposure and Parkinson's disease. This avenue offers a unique opportunity to transcend the confines of animal study data and delve into a deeper understanding that is both scientifically sound and ethically aligned, sidestepping the infeasibility of creating randomized controlled trial evidence in this particular domain. Similarly, neuro morbidity assessment in paraquat poisoning survivors is also a useful tool to know the link between PQ and PD by carefully studying the clinical phenotype (if any) that exists.

While current laboratory investigations employing the C57BL/6J mouse model[20] and epidemiological studies have not definitively established a direct connection between PQ exposure and PD, it is vital to emphasize that the absence of conclusive evidence should not discourage the continuation of research efforts. It is essential to acknowledge that the present lack of correlation does not necessarily preclude the potential association between PQ exposure and PD. Despite not fully meeting Hill's criteria for causation in accordance with existing literature, it is essential to remain open to the possibility that a causal link might exist, justifying further dedicated exploration. In epidemiology, the criteria are carefully designed for attribution rather than exclusion, and the pursuit of understanding demands a balanced recognition of these nuances before ruling out things. *Simply said, "Paraquat Brain" is yet another unexplored black box at autopsy.*

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Case Report

Eurasian Journal of Toxicology

Peripheral Facial Paralysis After COVID-19 BioNTech ® Vaccine

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Abstract

Bell's palsy, also known as acute peripheral facial nerve palsy of unknown cause, commonly manifests with sudden onset of unilateral facial paralysis. This case report describes two patients who has Bell's palsy after their first dose of the Pfizer-BioNTech vaccine.

Keywords: Bell's palsy, Pfizer-BioNTech vaccine, side effect.

Introduction

Bell's palsy, also known as acute peripheral facial nerve palsy of unknown cause, commonly manifests with sudden onset of unilateral facial paralysis. Bell's palsy is usually transient, with 70% of patients recovering within 6 months without treatment. The facial nerve, supplies motor innervation to the muscles of expression of the face and scalpand the satapedius muscle and taste to anterior two thirds of the tongue. Onset of facial paralsis is acute, with maximal semptoms in 2 to 3 days. The symptoms of Bell's palsy include sudden weakness in your facial muscles. The weakness makes half of your face appear to drop. Your smile is one-sided, and your eye on that side resists closing. Facial numbness or hyperesthesia can accompany paralysis. On exam patient will have facial drop effacement of wrinkles and forehead burrows and inability to completely close the eye.

Diagnosis of the Belly's palys is based on history and physical examination. Bell's palsy remains idiopathic, but a proportion may be caused by reactivation of herpes viruses from cranial nerve ganglia.Bell's palsy is a rare adverse event reported in clinical trials of COVID-19 vaccines.

Case Report

This case report describes two patients who has Bell's palsy after their first dose of the Pfizer-BioNTech vaccine. Two female patients, 42 years old and 19 years old, with no previous history of facial nerve palsy, suffered a Bell's palsy after 3 days BioNTech vaccination, one on the left side of the face and the other 19 years old on the right side of the face. The 42 years old woman noticed muscle weakness on the left side of the face and visited the emergency department. She presented facial droop, effacement of the nasolabial fold, and flaccidity on the left side of the face. Physical examination revealed complete paralysis of the left side of the face (the patient was unable to raise his eyebrow, close his right eye, or lift the labial commissure. The other has the same complaints on the right side of her face. Routine blood tests and a head Computerized Tomography (CT) scan are normal. These patients has no history of trauma or systemic infection. After prednisone treatment both patients showed improvement at follow-up examinations.

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Discussion

Coronaviruses (CoV) are large family of viruses. Symptoms of COVID-19 are often variable, but common symptoms include fever, cough, fatigue, difficulty breathing, anosmia and loss of sense of taste. About one in five people who are infected do not have any symptoms.Although most people have mild symptoms, it can cause acute respiratory distress syndrome (ARDS) in some people. ARDS can cause cytokine storms, multi-organ failure, sepsis and thrombosis. Long-term damage to organs (especially lungs and heart) has been observed. COVID 19 is most known for affecting the upper respiratory tract and the lower respiratory tract. A COVID 19 vaccine is a vaccine intended to provide acquired immunity against severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Vaccines prevent the spread of viral infection and reduce its mortality. Although in many cases, mild side effects such as injection site pain, mild fever, fatigue, headache, arthralgia, and myalgia are reported, several cases with neurological manifestations after COVID-19 vaccination have been observed. One of the neurological manifestations of COVID-19 vaccines is Bell's palsy. Two clinical trials reported seven cases of Bell's palsy. The FDA did not consider a clear relationship between the vaccine and Bell's palsy[1-4].

Conclusion

Although still unproven, growing evidence suggests a relationship between the covid vaccine and bell's palsy. Therefore, it would be helpful to follow patients for bell palsy after administration of mRNA vaccines.

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Case Report

Eurasian Journal of Toxicology

Intracranial Bleeding in Methanol Intoxication Case

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Abstract

Methyl alcohol, which is mostly used in the industrial sector, is also widely used in our country to obtain cheap drinks. It is a colorless, volatile liquid with a distinctive odor. Even 8-10 ml of methanol taken into the body from outside is toxic. Approximately 25-30 ml of methanol can cause poisoning that can lead to permanent blindness. The best method for the definitive diagnosis of methyl alcohol poisoning is to measure the methanol level in the blood. Patients may develop headache, central nervous system depression, coma and seizure. Imaging is recommended in patients with altered consciousness. Putaminal necrosis and intracranial hemorrhage (SAH, putaminal intraparenchymal hemorrhage) may develop in these patients. Our case is a 50-year-old male patient with a history of constant homemade alcohol consumption. The patient was brought with complaints of visual impairment and confusion after drinking alcohol. Basal ganglia hemorrhage was detected on the patient's cranial tomography. The patient, who was admitted to intensive care, deteriorated and died on the 4th day of his treatment. Morbidity and mortality rates can be very high in patients presenting with suspected methanol intoxication. Therefore, we wanted to emphasize the importance of early diagnosis and treatment.

Keywords: Emergency medicine, methanol, intoxication

Introduction

Methyl alcohol, which is most frequently used in the industrial sector, is also widely used in our country to obtain cheap beverages. It is a colorless, volatile liquid with a distinctive odor. It is highly and rapidly absorbed from the gastrointestinal tract (within 60-90 minutes). After absorption, most of the methanol (90-95%) is eliminated by the liver, 2-5% by the kidneys, and a very small amount by the lungs. The cause of toxicity is the conversion of methanol into formaldehyde and formic acid by the alcohol dehydrogenase enzyme in the liver. Formic acid accumulation is associated with clinical symptoms and formic acid is responsible for toxicity and has fatal toxicity. It causes a very fatal toxicity due to metabolic acidosis with a high anion gap. Formaldehyde formation in the retina causes optic papillitis and retinal edema, which causes blindness, especially defined as blind drunkenness. End organ damage begins when methanol levels exceed 6 mmol/L[1-4].

Case Report

A 50-year-old male patient has a history of constantly drinking homemade alcohol. He doesn't have any illnesses in his history. There was a history of drinking homemade alcohol before presenting to the emergency department. He fell asleep after drinking alcohol and his vision deteriorated after waking up. The patient, who lost consciousness, was brought to the hospital by 112 Emergency Ambulance Service. The patient's arrival vital values; temperature: 36.5 C, blood pressure: 132/79 mmHg, heart rate: 92/min, SpO2: 96%, blood sugar: 217 mg/dl, Arterial blood gas values; pH: 6.94, pCO2: 27, pO2: 131, HCO3: 7.2, lactate: 8. Sinus rhythm was noticed in electrocardiogram. When the patient was taken to the emergency room, his GCS was 3, his breathing was shallow, and his pupils were mydriatic. The patient was electively intubated after the initial evaluation. The right femoral dialysis catheter was opened.

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The patient's vitals were taken every hour. After consulting the nephrologist, he was placed on hemodialysis for 4 hours. After 4 hours of hemodialysis in the emergency department, the pH in the blood gas was determined as 7.13. The patient, whose vitals were stable, was taken to the radiology unit for brain tomography. Brain tomography revealed bilateral hemorrhage in the basal ganglia (Figure 1). Surgery was not recommended by the neurosurgeon. The patient was given 10% Ethanol 800 mg iv with 30 minute infusion, Ca folinate 50 mg iv (4x1), 5% Dextrose 500 cc iv (250cc/hour) infusion, Mannitol 6x75 ml iv infusion, 10% Ethanol 500 cc iv infusion (80 cc/hour), 10% Ethanol 500 cc iv infusion (125cc/hour) (during hemodialysis), 10% Dextrose 500cc + 18 iu HR insulin iv (500cc/hour) Since the patient became hypotensive in the 3rd hour of hemodialysis, inotrope support was started. Steradin 2 amp + 0.9% SF 100cc (20cc/hour) iv, Dopamine 2 amp + 0.9% SF 100cc (10cc/hour) iv were administered. The patient was admitted to the general intensive care unit on the second day of emergency department follow-up. The methanol value measured upon admission to the intensive care unit was 194 mmol/L. It was learned that the patient died on the 4th day of methanol intoxication after being admitted to the intensive care unit.

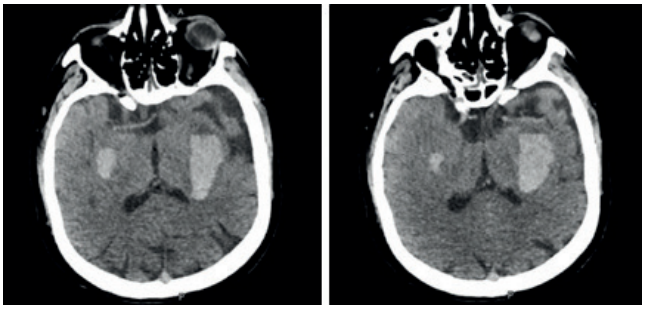

Figure 1: Brain tomography revealed bilateral hemorrhage in the basal ganglia.

Conclusion

When a case is encountered, rapid evaluation should be made, early treatment should be started and dialysis should be initiated if necessary. Despite treatment, in patients presenting with suspicion of methanol intoxication Unfortunately, morbidity and mortality rates can be quite high.

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