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acute toxicity^[6]. The Pesticide Action Network (PAN) from India remain limited.

International designates paraquat as a highly hazardous

SHVWLFLGH DQG KDV OLVWHG LW DSS. Presentation ± EDG DFWRU · DQG ± GLUW GRJHQ · FK7H Toxicology

Toxicological Data Network (ToxNet) and the Integrated Risk Information System (IRIS) of the United States Environmental Protection Agency (US EPA), classify paraquat as a probable human carcinogen (Group⁹C)

Paraquat exposure has been associated with reproductive distress was the most common complaint reported by all WR[LFLW\ DQG D SRVVLEOH HWL RORSLAF DQG DSS. Paraquat is a highly toxic herbicide used for paraquat poisoning

disease^[10, 11]. Farmers, agricultural laborers and residents living near treated areas are particularly vulnerable to occupational, environmental and accidental exposure. The compound can be absorbed through intact skin, with significantly increased absorption when the skin is abraded or damaged^[12]. Acute poisoning may occur via dermal, ocular or inhalational routes and clinical signs may be delayed with fatal outcomes frequently result from progressive respiratory failure^[3]. Localized dermatitis, ocular injury and epistaxis are commonly reported among exposed individuals, while chronic low level exposure has been associated with structural lung changes, chronic bronchitis and exertional dyspnoea^[14, 15].

Although paraquat is registered for use in more than 130 countries, at least 32 countries including all member states of the European Union have banned or severely restricted its use because of serious human health concerns.

In Switzerland, the home country of Syngenta, the principal manufacturer of paraquat, its use has been prohibited since 1989 owing to its high acute toxicity^[17]. Several international, the Forest Stewardship Council and Rainforest Alliance, as well as multinational food corporations such as Dole and Chiquita and major retailers including Marks & Spencer, have voluntarily eliminated paraquat from their supply chains^[18].

In India, paraquat dichloride 24% SL is registered with the Central Insecticides Board and Registration Committee (CIBRC) as the sole approved formulation and is categorized as highly toxic despite the absence of explicit CIBRC recommendations, its use has been approved for nine crops^[19]. In contrast, paraquat dimethyl sulfate was banned in India in 1993. Paraquat dichloride continues to be widely used herbicide^[20].

Paraquat is frequently implicated in cases of deliberate self-poisoning, particularly in agricultural regions. No specific antidote exists and the reported case fatality rate for paraquat poisoning ranges from approximately 42% to 80%^[21, 22]. Global estimates suggest that tens of thousands of deaths annually may be attributable to paraquat ingestion often associated with a poor prognosis, whereas a negative presentation does not exclude paraquat toxicity in delayed northern, southern and northeastern states. In Kerala, distribution, sale and use of paraquat were halted in 2011 and re-distributed into tissues such as the lungs and skeletal muscle. Although the sodium dithionite test lacks the sensitivity and specificity of advanced analytical techniques such as high performance liquid chromatography that state^[24]. Despite its well recognized role in causing serious morbidity and mortality among agricultural workers and surrounding communities worldwide, systematic diagnosis of paraquat poisoning in dogs when interpreted in conjunction with clinical findings and exposure history.

including Marks & Spencer, have voluntarily eliminated paraquat from their supply chains^[18].

Diagnostic Techniques

The sodium dithionite test (fig.1) is a rapid, simple, qualitative screening method used for the detection of paraquat in biological samples and is widely employed in both human and veterinary toxicology. The test is based on the reduction of paraquat, a bipyridylium compound, by sodium dithionite under alkaline conditions to form a stable blue coloured paraquat radical ion. Urine is the preferred sample due to higher sensitivity, although plasma, serum or gastric contents may also be used. Following alkalisation of the sample, the addition of freshly prepared sodium dithionite results in the immediate development of a blue or blue purple colour in positive cases. The intensity of the colour roughly correlates with paraquat concentration, but the test remains qualitative in nature. A strongly positive reaction shortly after exposure supports the diagnosis and is often associated with a poor prognosis, whereas a negative reaction does not exclude paraquat toxicity in delayed presentations because paraquat is rapidly cleared from circulation and redistributed into tissues such as the lungs and skeletal muscle. Although the sodium dithionite test lacks the sensitivity and specificity of advanced analytical techniques such as high performance liquid chromatography or gas chromatography mass spectrometry, it remains a valuable bedside screening tool for early presumptive diagnosis of paraquat poisoning in dogs when interpreted in conjunction with clinical findings and exposure history.



Fig 1: Sodium dithionite test for detection of paraquat in biological samples showing progressive colour intensity corresponding to paraquat concentration. The increasing intensity of blue coloration provides a semi-quantitative indication of paraquat toxicity and is generally associated with worsening prognosis.

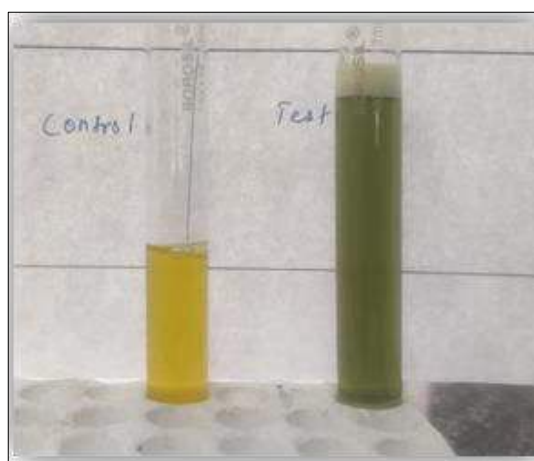


Fig 2: Sodium dithionite test for paraquat poisoning showing comparison between control and test samples. The control sample remains yellow without colour change, indicating absence of paraquat, whereas the test sample develops a greenish blue colour following alkalisation and addition of sodium dithionite, confirming the presence of paraquat in the biological sample.

3HULSKHUDO %ORRG 2[\JHQ 6DWXU D\WWSR95-100% 2w represents normal oxygen saturation; 90-95% indicates partial haemoglobin desaturation; 85-90% markedly reduced in dogs affected with paraquat reflects hypoxia; and values below 85% are associated with intoxication due to severe pulmonary dysfunction and cyanosis and severe, life-threatening oxygen deprivation. In impaired alveolar gas exchange. Progressive alveolar paraquat-PLVRQHG GRJV 6S2w OHYHOV damage, interstitial oedema, haemorrhage and fibrosis result progressively despite supportive therapy, correlating with in inadequate oxygen diffusion, leading to systemic worsening pulmonary injury and poor prognosis.

Table 1: 6HTXHQWLDO 3HULSKHUDO 2I\JHQ EDWXLUDWLRQ 6S2w LQ 'RJV

Case No.	Presentation (Day 1)	24-72 h	Advanced stage	Terminal stage
12165	94	88	56	42
12191	93	86	52	39
15746	95	89	58	45
8579	92	85	54	40
16771	94	87	50	38
14912	91	86	55	43
17711	93	88	57	44
12428	92	85	53	41
9453	95	89	59	46
7687	94	87	51	37

Echo cardiography

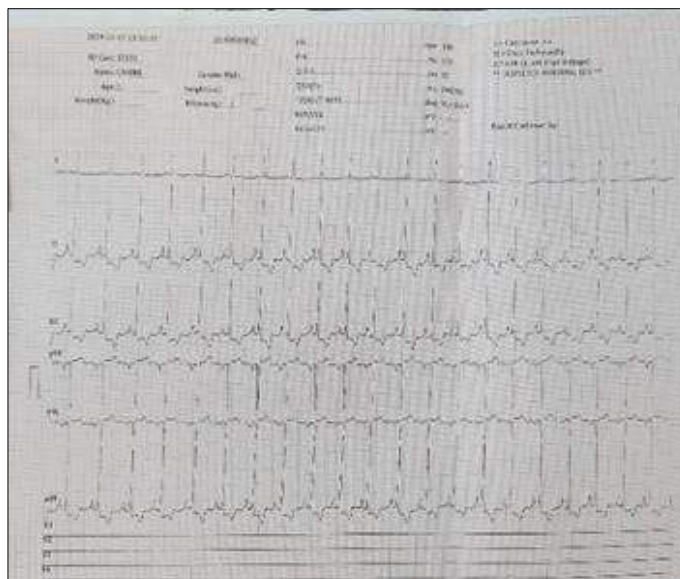


Fig 3: Electrocardiogram of a dog with paraquat poisoning showing sinus tachycardia and mild nonspecific ST-T changes, indicating secondary myocardial hypoxia due to pulmonary injury



Fig 4: Correlation E H W Z H H Q (& * F K D Q J H V D Q G S H U L S K H U D O R [\ J H Q V D W X U D W L R Q 6 S 2 w W D F K \ F D U G L D Z L W K G H F O L Q L Q J 6 S 2 w U H I O H F W L Q J F D U G L R Y D V F X O

Electrocardiographic (ECG) evaluation in dogs (fig. 3, 4) fibrosis resulted in impaired gas exchange and declining with paraquat intoxication consistently revealed sinus tachycardia as the predominant abnormality. This finding partial haemoglobin desaturation occurring at 90-95%, was primarily attributed to severe hypoxaemia, respiratory hypoxia evident at 85-90% and values below 85% distress and heightened sympathetic stimulation secondary associated with cyanosis and severe oxygen deprivation. to progressive paraquat induced pulmonary injury. In some cases, mild nonspecific ST-segment and T-wave changes were observed, likely reflecting secondary myocardial cardiovascular response to pulmonary failure in paraquat poisoning and together these parameters provide valuable indicators of disease severity and poor prognosis in affected dogs. These ECG alterations closely paralleled changes in dogs.

SHULSKHUDO EORRG R [\ J H Q V D W X U D W L R Q 6 S 2 w 3 U R J U H V V L Y H
alveolar damage, pulmonary oedema, haemorrhage and radiographic examination

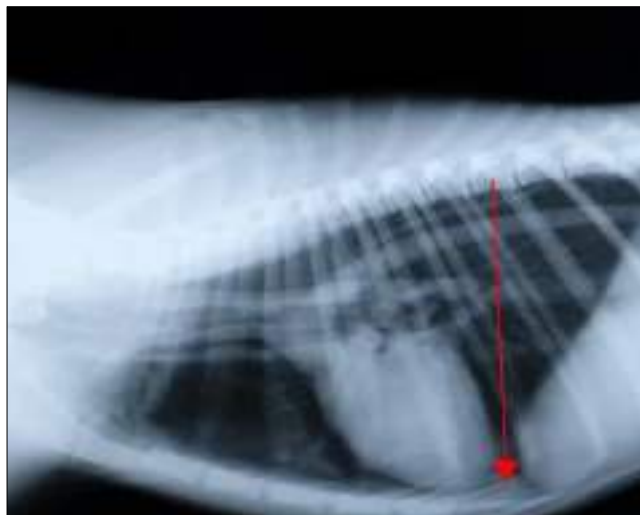


Fig 5: Pneumo- mediastinum



Fig 6: Pneumo abdomen

Thoracic and abdominal radiographic examination (Fig.5, 6) the abdominal cavity, resulting in pneumoperitoneum in dogs with paraquat intoxication revealed characteristic (pneumo abdomen). The presence of free intraperitoneal gas changes consistent with severe pulmonary and soft tissue injury. Pneumomediastinum was identified by the presence of radiolucent streaks outlining the cardiac silhouette, findings reflect the severity of paraquat-induced pulmonary injury. These radiographic findings of radiolucent streaks outlining the cardiac silhouette, trachea and major mediastinal structures, indicating the injury and are associated with a poor prognosis, as they presence of free air within the mediastinum. This finding is indicative of extensive alveolar destruction and air leakage along suggestive of alveolar rupture secondary to severe fascial planes. pulmonary damage and increased intrathoracic pressure associated with dyspnoea and coughing. In some cases, a haematology was observed tracking caudally from the mediastinum into

Table 2: Haematological parameters of paraquat affected dogs

Case No.	12165	12191	15746	8579	16771	14912	17711	12428	9453	7687
Haematology										
Hb (g/dL)	15.6	16.1	14.3	17.6	18.9	22.8	20.8	15.2	18.2	16.2
PCV (%)	50.8	49.4	41.1	58.6	49.4	71.9	57.1	30.5	41.1	49.1
RBC (10^6 / O)	7.4	7.28	6.86	8.51	7.61	10.52	8.95	3.06	7.71	6.23
WBC (10^3 / O)	31.64	21	8.12	22.7	12.97	14.31	45.79	11.66	10.65	11.21
Platelet count (10^3 / O)	353	110	301	110	325	167	89	210	220	210
Neutrophils (%)	92.5	81.9	84.4	77.8	68.8	83.5	86.2	82.4	80.5	82
Lymphocytes (%)	2.7	7.5	7.2	5.6	21.9	8.5	4.8	8.5	7.3	7.0
Monocytes (%)	4.7	10.2	8.1	16.5	9.2	7.8	8.9	8.8	8.2	9.5
Eosinophils (%)	0.1	0.3	0.2	0	0	0.1	0	0.2	0.1	0.2
Basophils (%)	0	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1

Table 3: Serum biochemistry parameters of paraquat affected dogs

Case No.	12165	12191	15746	8579	16771	14912	17711	12428	9453	7687
Serum biochemistry										
Total protein(g/dl)	8.53	8.19	8.09	9.1	7.57	7.3	7.22	7.88	6.9	7.5
Albumin(g/dL)	3	3.86	3.99	4.11	4.31	4.12	4.15	3.76	3.9	3.2
Globulin(g/dL)	5.53	4.33	4.1	4.99	3.26	3.18	3.07	4.12	3	4.6
ALT(U/L)	64.6	25.1	30.7	29	32.7	74.5	65.1	29.6	26.2	30.2
ALP(U/L)	39	149	105	152	40	115	151	53	58	152
LDH(U/L)	218	149	280	186	352	368	1525	872	565	780
Direct bilirubin (mg/dL)	0.2	0.3	0.5	0.5	0.4	0.5	0.6	0.04	0.5	0.6
Total bilirubin (mg/dL)	0.03	0.02	0.06	0.04	0.05	0.37	0.06	0.05	0.04	0.06
BUN(mg/dL)	28.95	96.9	12.35	107.5	37.2	27.72	47.7	2.81	25.06	29.1
Creatinine(mg/dL)	1	2.02	0.83	2.5	0.98	1.01	0.94	1.1	0.86	1.9
Calcium(mg/dL)	9.7	10.1	9.4	10.8	11.1	10.8	9.8	7.42	9.5	8.6
Phosphorous(mg/dL)	5.9	5.45	4.9	5.24	4.87	5.78	5.1	4.83	4.9	4.2
Magnesium(mg/dL)	2.1	2.3	2.1	3	2.5	1.9	2.1	2.5	2.4	1.8

Haematological evaluation (table 2) of dogs with paraquat intoxication revealed variable but consistent abnormalities, leading to the development of secondary pulmonary response. Haemoglobin concentration, packed cell volume and red blood cell counts were increased in several dogs suggesting haemoconcentration likely due to dehydration and fluid losses secondary to vomiting, diarrhoea and reduced intake. Marked leukocytosis was observed in most cases, predominantly characterised by neutrophilia with response, stress leukogram and severe tissue injury. The main pulmonary artery is often dilated, with an Thrombocytopenia was evident in some dogs, which may be attributed to systemic inflammation, consumption or toxic effects on bone marrow or platelets.

On echocardiographic evaluation, affected dogs typically show enlargement of the right atrium and right ventricle due to sustained pressure overload and in some cases mild thickening of the right ventricular free wall. Flattening or leftward displacement of the interventricular septum produces a characteristic D-shaped left ventricle on short-axis views, indicating elevated right ventricular pressure. The main pulmonary artery is often dilated, with an increased pulmonary artery-to-aorta ratio, reflecting increased pulmonary arterial pressure. Doppler examination commonly reveals tricuspid regurgitation caused by right ventricular dilation and measurement of the regurgitant jet velocity allows estimation of increased systolic pulmonary arterial pressure. Pulsed-wave Doppler assessment of pulmonary artery flow may demonstrate shortened acceleration time and a mid systolic notch, findings consistent with increased pulmonary vascular resistance. In advanced cases, secondary signs of right-sided cardiac congestion, such as dilation of the caudal vena cava and reduced respiratory variation, may also be observed.

Serum biochemical analysis (table.3) demonstrated evidence of multi-organ involvement. Total protein and globulin concentrations were mildly to moderately elevated in several cases, reflecting dehydration and inflammatory response. Mild to moderate elevations in alanine aminotransferase (ALT) and alkaline phosphatase (ALP) were noted, suggesting hepatocellular injury and resistance. In advanced cases, secondary signs of right-sided cardiac congestion, such as dilation of the caudal vena cava and reduced respiratory variation, may also be observed. These ultrasonographic findings (fig.7) correlate with the damage and tissue hypoxia, particularly involving severity of paraquat induced pulmonary injury and hypoxic pulmonary and muscular tissues. Serum bilirubin values were generally within normal limits or only mildly elevated, indicating the absence of significant haemolysis or severe hepatic dysfunction.

Renal involvement was evident in several dogs, as indicated by elevated blood urea nitrogen (BUN) and creatinine concentrations, consistent with acute kidney injury due to direct paraquat nephrotoxicity and reduced renal perfusion. Electrolyte analysis revealed mild variations in calcium, phosphorus and magnesium concentrations, likely reflecting renal dysfunction, anorexia and altered metabolic status. Overall, the haematological and biochemical abnormalities observed in these cases reflect the systemic nature of paraquat toxicity, with prominent inflammatory response, cellular hypoxia, hepatic stress and variable degrees of renal impairment, correlating with the severe clinical course and poor prognosis observed in affected dogs.

Ultrasound study

Paraquat intoxication in dogs causes marked oxidative damage to the lungs, particularly affecting the alveolar lining cells and pulmonary capillary endothelium. This results in acute lung inflammation, pulmonary oedema, haemorrhage and progressive scarring of the lung tissue.



Fig 7: Thoracic ultrasonography of a dog with paraquat intoxication showing reduced respiratory variation, consistent with severe pulmonary injury. These findings reflect hypoxic vasoconstriction and the development of secondary pulmonary hypertension

Quantitative Confirmation

Paraquat can be quantitatively detected and confirmed in clinical and forensic investigations.

biological samples using several analytical techniques

available in specialised laboratories. Spectrophotometric methods are commonly employed for rapid screening and estimation based on the characteristic absorption of species (ROS), resulting in widespread oxidative stress and paraquat, although their sensitivity and specificity are multi organ injury. Following ingestion, paraquat is rapidly limited. High performance liquid chromatography (HPLC) offers improved accuracy and sensitivity and is widely used for quantitative determination of paraquat in blood, urine and tissue samples. Gas chromatography coupled with mass spectrometry (GC-MS) provides highly sensitive and specific identification and quantification, allowing definitive confirmation even at low concentrations. Radioimmunoassay (RIA) is another sensitive technique that enables detection of paraquat in biological matrices through antigen antibody reactions, though its availability is restricted to specialised facilities. These analytical methods

play a crucial role in confirming exposure, assessing severity and supporting the diagnosis of paraquat

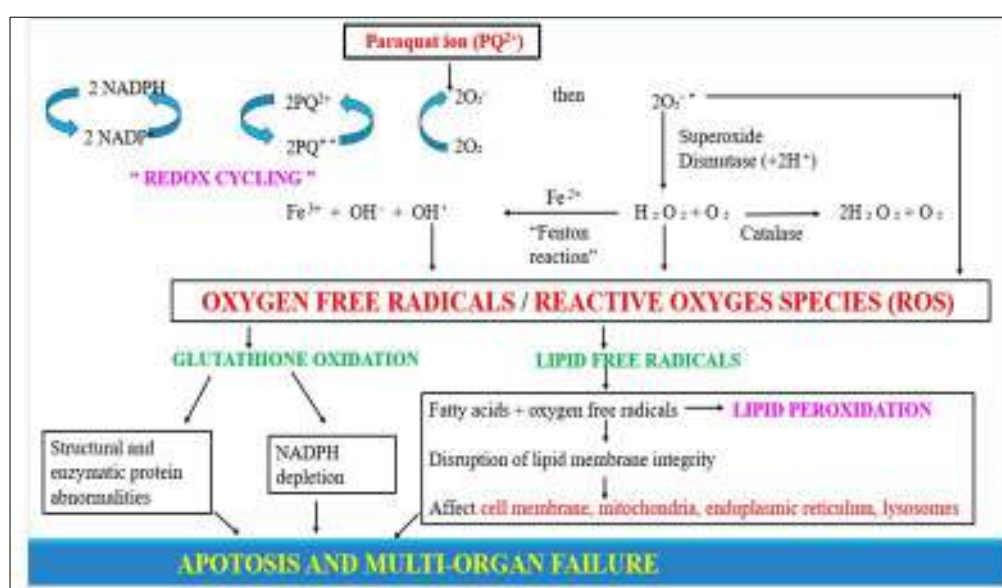


Fig 8: Schematic representation of paraquat-induced toxicity in dogs. Paraquat generates reactive oxygen species (ROS) leading to lipid peroxidation, protein oxidation, DNA damage, and mitochondrial dysfunction, resulting in cell death by necrosis and apoptosis. Pulmonary injury manifests as alveolitis, oedema, haemorrhage, and progressive fibrosis, while extra-pulmonary effects include acute kidney injury, gastrointestinal ulceration, hepatic damage, and secondary CNS involvement, collectively causing multi-organ failure.

The generated ROS cause lipid peroxidation of cellular and nervous system involvement may occur secondary to organelle membranes, oxidation of proteins and DNA hypoxia and metabolic derangements. Collectively, the damage, ultimately resulting in mitochondrial dysfunction, mechanism of paraquat toxicity in dogs is characterized by depletion of intracellular antioxidants such as glutathione persistent oxidative stress organ specific accumulation and cell death by necrosis and apoptosis. In the lungs particularly in the lungs and progressive, often fatal, multi oxidative injury to alveolar epithelium and pulmonary organ failure in the absence of a specific antidote.

capillary endothelium leads to acute alveolitis, pulmonary

oedema, haemorrhage and severe impairment of gas exchange.

Surviving dogs often develop progressive interstitial fibrosis due to fibroblast activation and excessive collagen deposition, culminating in irreversible respiratory failure. Following ingestion, paraquat is rapidly absorbed and selectively taken up by the lungs (fig.9) within the first few hours, achieving pulmonary concentrations several-fold higher than plasma levels. Within approximately 6 hours,

Paraquat also induces significant extra pulmonary toxicity. paraquat induces acute alveolitis through intense oxidative injury to alveolar epithelial and capillary endothelial cells.

and ischemia, resulting in acute kidney injury that further impairs paraquat excretion and exacerbates systemic toxicity. By the third day post-exposure, profibroblasts migrate into the alveolar spaces, initiating fibroproliferative responses.

By around day seven, progressive pulmonary fibrosis becomes evident, resulting in marked impairment of alveolar gas exchange. This culminates in severe hypoxaemia, tissue anoxia and death due to asphyxia in

most affected dogs. In parallel, paraquat is also taken up by skeletal muscle, which acts as a secondary reservoir associated with extremely poor survival. Survival Although plasma paraquat levels may decline and probability decreases sharply with increasing plasma levels qualitative tests such as the sodium dithionite test may become negative, paraquat continues to be released slowly from muscle tissue (fig.10) back into circulation. This guarded outcomes. However, declining plasma sustained redistribution perpetuates pulmonary exposure and concentrations do not necessarily indicate recovery, as contributes to delayed worsening of lung injury and fatal continued release from tissue stores, particularly muscle, outcomes. The prognostic relationship between plasma can maintain toxic pulmonary concentrations and drive paraquat concentration and survival demonstrates a strong progressive respiratory failure.

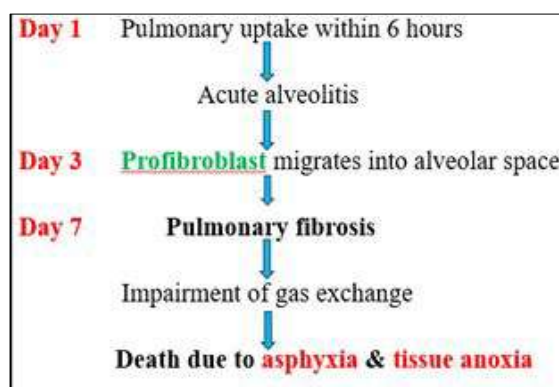


Fig 9: Pulmonary accumulation of paraquat following ingestion in dogs. Paraquat is rapidly absorbed and selectively taken up by the lungs within the first few hours, causing acute alveolitis through oxidative injury, followed by fibroproliferation and progressive pulmonary fibrosis, leading to severe hypoxaemia and respiratory failure

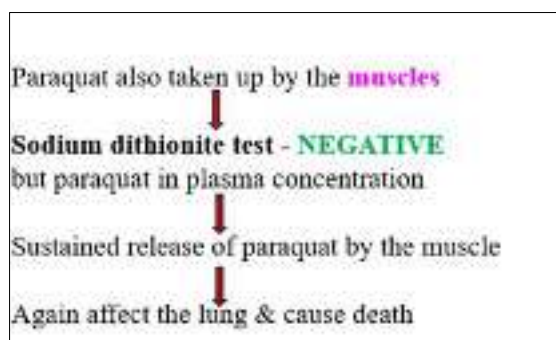


Fig 10: Skeletal muscle as a secondary reservoir of paraquat. Paraquat released slowly from muscle back into circulation sustains pulmonary exposure, contributing to delayed worsening of lung injury and poor prognosis, even when plasma levels initially decline

Necropsy findings



Fig 11: Congested lung with edema

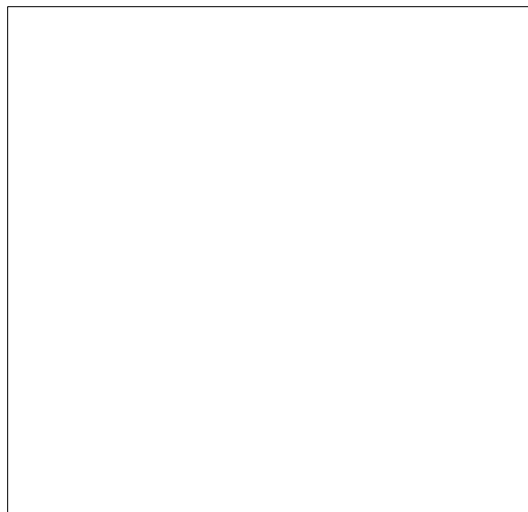


Fig: 12 Hepatomegaly



Fig 13: Splenomegaly

Detailed post-mortem examination revealed severe hepatocellular injury compounded by hypoxia and multisystem lesions, with the lungs (fig.11) showing the circulatory compromise. Mild bile staining was occasionally most prominent and diagnostically significant changes. The observed, although no focal necrosis was evident grossly. lungs were markedly congested, enlarged and heavy, failing. The spleen (fig.13) was moderately to markedly enlarged to collapse upon opening of the thoracic cavity. The pleural and congested, with a dark red to purplish coloration. On cut surfaces were dark red to bluish-red and glistening, with section, the splenic pulp was soft and oozed blood, multifocal petechial to ecchymotic haemorrhages. On indicating vascular engorgement and systemic inflammatory sectioning, copious amounts of serosanguinous, frothy fluid response. Splenic enlargement was interpreted as secondary oedema. The pulmonary parenchyma was firm and rubbery. Overall, the necropsy findings were dominated by severe in consistency in several areas, indicating consolidation and pulmonary congestion and oedema, accompanied by early fibroproliferative changes consistent with paraquat-hepatomegaly and splenomegaly, reflecting the systemic induced alveolar injury. nature of paraquat toxicity. The severity of pulmonary lesions correlated with the rapid clinical deterioration and fatal respiratory failure observed in affected dogs. The liver (fig.12) was diffusely enlarged with rounded edges and a tense capsule. The hepatic parenchyma appeared dark, congested and friable, with accentuation of the lobular pattern on cut section. These changes are suggestive of toxic distopathology of Lung.



Fig 14: Microscopic lung changes in a dog with paraquat poisoning showing alveolar oedema, congestion, inflammatory infiltrates, epithelial necrosis, and early fibroblast proliferation, indicative of acute alveolitis progressing to fibroproliferative injury

Microscopic examination (fig.14) of the lung revealed techniques such as haemoperfusion and haemodialysis have severe and diffuse pathological alterations consistent with limited or questionable benefit however, they may prolong acute toxic lung injury due to paraquat. The alveolar spaces were variably collapsed or distended and filled with fluid. Fluid therapy is generally contraindicated due to the eosinophilic proteinaceous oedema fluid, fibrin strands and presence of severe respiratory distress and the risk of inflammatory cell infiltrates. Marked congestion of alveolar capillaries and interalveolar septa was evident, along with gastric lavage are also contraindicated because of the multifocal haemorrhages. The alveolar septa were markedly thickened due to oedema, infiltration by neutrophils and mucosal injury to the gastrointestinal tract.

macrophages and early fibroblast proliferation.

Degeneration and necrosis of alveolar epithelial cells were observed, with desquamation of pneumocytes into the alveolar lumen. In some areas, bronchiolar epithelial toxicity, and inflammation and fibrosis.

Antioxidant Therapy Given the central role of oxidative stress in paraquat damage with luminal cellular debris and inflammatory exudate was noted. These histological features are indicative of acute alveolitis progressing towards fibroproliferative mediated cellular damage. N-acetyl cysteine is used to changes, which correlate with paraquat induced oxidative injury and explain the severe impairment of gas exchange and fatal respiratory failure observed clinically.

Antioxidant Therapy followed by 300 mg/kg over 24 hours for up to three weeks.

Desferrioxamine (100 mg/kg over 24 hours) acts as an iron chelator, thereby limiting iron-catalysed hydroxyl radical formation.

Other Clinical Signs

In addition to respiratory compromise, dogs with paraquat intoxication commonly exhibit systemic and gastrointestinal manifestations. Cardiovascular involvement is reflected by an elevated heart rate, often secondary to hypoxaemia and systemic stress. Progressive multi-organ failure may occur as a consequence of widespread oxidative injury.

Conclusion Gastrointestinal signs such as vomiting and diarrhoea are frequent and are attributed to the corrosive effects of paraquat on the gastrointestinal mucosa. Laryngo-respiratory distress, gastrointestinal signs, renal dysfunction pharyngitis, excessive salivation and oral discomfort are also commonly observed due to direct chemical irritation.

Paraquat toxicity should be considered an important differential diagnosis in dogs presenting with unexplained paraquat on the gastrointestinal mucosa. Laryngo-respiratory distress, gastrointestinal signs, renal dysfunction pharyngitis, excessive salivation and oral discomfort are also commonly observed due to direct chemical irritation. paraquat. Given the relative ease of access to paraquat-Respiratory distress remains the predominant clinical feature containing formulations such as Gramoxone 250 and progressively worsens despite therapeutic intervention. (Syngenta), the risk of intoxication in both humans and animals remains significant, whether accidental, intentional as in cases of self-harm or through malicious exposure. The

Treatment

Therapeutic management of paraquat poisoning in dogs is largely supportive and aimed at limiting pulmonary and inflammatory, oxidative injury and secondary progression of severe pulmonary injury and irreversible complications, as no specific antidote is available. Attempts may be made to reduce pulmonary inflammation and fibrosis through the administration of anti-inflammatory and immunosuppressive agents, as well as inhibitors of collagen synthesis. Corticosteroids such as methylprednisolone (0.5-1 mg/kg) or dexamethasone (0.1-0.2 mg/kg), alone or in combination with cyclophosphamide, have been used with the objective of attenuating fibroproliferative lung injury, although clinical efficacy remains inconsistent.

Broad-spectrum antibiotics, including amoxicillin (10-20 mg/kg), enrofloxacin (4-7.5 mg/kg) and ceftiofur sodium (1-2 mg/kg), may be administered to prevent or treat secondary bacterial infections, particularly those involving the respiratory tract.

Angiotensin-converting enzyme inhibitors such as enalapril and captopril have been reported to reduce paraquat-induced pulmonary fibrosis by modulating profibrotic pathways.

Oxygen supplementation is contraindicated in paraquat poisoning, as increased oxygen tension enhances free-radical generation and exacerbates oxidative lung injury. Diuresis using agents such as furosemide (1-2 mg/kg) is recommended to reduce paraquat concentration within renal tubules and mitigate nephrotoxicity. Saline cathartics may aid in the rapid elimination of paraquat from the gastrointestinal tract if administered early. Extracorporeal

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