# Paraquat Poisoning Cases Treated at Shohada Ashayer Hospital of Khorramabad in 2001-2006

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Abstract: The purpose of this research is to study the socio demographic pattern and mode of presentation of Paraquat intoxication in Shohada Ashayer Hospital, Khorramabad, Iran. In this descriptive study, 52 cases of Paraquat toxin poisoning, admitted through the emergency department of internal medicine ward, were evaluated in the study. In all the cases a previously established clinical and therapeutic protocol was applied and the outcome was recorded. Twenty nine (55.76%) patients were male, 23 (44.24) were female with the mean age of 25.04 years. Male to female ratio was 1.26. 40 (76.9%) cases were attempted suicides and 12 cases (23.1%) were due to accidental events. Mean age was 22.1 years in the 40 suicidal cases and 34 of them were less than 24 years. Among the suicide attempts, 31(77.5%) were female patients. The patients' mean arrival time to the hospital after poisoning was 2.1±6.01 h. Exposure routes were gastrointestinal in 52 patients (100%) of patients. Death of 37(71.1%) patients was occurred in the patients of this study. In 40 suicidal cases, 34 patients were died (85%). In cases that using more than 20cc of liquid, the mortality was 94.4 % (27 case). Causes of poisoning (suicide vs. non-suicide), the time interval between poisoning to arrival at hospital and the amount of Paraquat toxin were major determinants of lethality.

Key words: Paraquat, poisoning, suicide, intoxication

#### INTRODUCTION

Paraquat is an Active Ingredient (AI) used in Crop Protection Products (CPPs, sometimes called pesticides). Being an herbicide, Paraquat protects crops by controlling a wide range of annual and certain perennial weeds (unwanted plants) that reduce both crop yield and quality by competing with the crop for water, nutrients and light (Ramdial and Poon-King, 1984; Fock, 1987).

Paraquat is one of the most widely used herbicides in the world and is used to control annual and perennial weeds in a wide variety of crops. The key characteristics that distinguish the non-selective contact herbicide Paraquat from other active ingredients used in plant protection products are (Chandrasiri, 1999; Hampson and Pond, 1988): Paraquat is non-selective, which means it kills a wide range of annual grass and broad-leaved weeds and the tops of established perennial weeds, Paraquat is very fast-acting and Paraquat is rain-fast within minutes of application. Paraquat becomes biologically inactive upon contact with soil (Hampson and Pond, 1988).

Chemical properties of Paraquat are: Extremely soluble in water, insoluble in most organic solvent, Not volatile, sprayed droplets too large to enter lung, Potentially lethal if swallowed (Hettiarachchi and Kodithuwakku, 1989), Directly caustic (Casey and Vale, 1994) and Suppress reduction of NADP¬NADPH during photosynthesis in plant and production of super oxide, singlet oxygen, peroxide radicals. Destroy cell membrane (Fock, 1987; Hampson and Pond, 1988).

Clinical manifestations are Depends on route of absorption. For example in: GI tract: Ulceration of oral cavity, pharynx and esophagus (Esophageal perforation is possible) (Ramdial and Poon-King, 1984; Fock, 1987; Bismuth et al., 1986), Skin: Skin rash, Respiratory epistaxis, haemoptysis, pneumothorax, pneumomediastinum and subcutaneous emphysema (Fock, 1987), Systemic toxicity: Acute renal failure (Chandrasiri, 1999), Acute heart failure (Ozyyurt et al., 1997), Acute pulmonary edema (in 24-48 h), Metabolic acidosis (cardiac failure, adrenal insufficiency, hypotension, hypoxemia, renal failure (Jayaratnam, 1990).

Mild poisoning (<20 mg Paraquat /kg body weight) presentation is: Asymptomatic or vomiting and diarrhoea, Minimal renal and hepatic dysfunction, Decrease in pulmonary diffusion capacity and Complete recovery expected (Bismuth *et al.*, 1986; Fock, 1987; Hampson and Pond, 1988).

Moderate poisoning (>20 mg but <40 mg Paraquat/kg body weight) presentation is: Immediate: Vomiting (Gnyp and Lewandowska, 1997), H: Diarrhea, abdominal pain, oropharyngeal ulceration, 1-4 days: Acute renal failure, hepatic derangement, hypotension and tachycardia (Fock, 1987; Hampson and Pond, 1988), 1-2 weeks: Cough, haemoptysis, pleural effusion, pulmonary fibrosis and Majority of cases die within 2-3 weeks from respiratory failure (Fock, 1987).

Acute fulminant poisoning (> 40 mg Paraquat per kg) presentation is: Immediate: vomiting, 2-H to days: Diarrhea, abdominal pain, renal and liver failure, GI ulceration, pancreatitis, myocarditis, hypotension, coma and convulsion (Fock, 1987) and Death from multi-organ failure in 4 days (Malik *et al.*, 1998).

Toxicity has occurred after topical application, oral ingestion or inhalation of Paraquat (Fock, 1987). Systemic toxicity has not been reported from smoking of Paraquatmarijuana but heavy abusers of contaminated contaminated marijuana may experience coughing, hemoptysis and mouth irritation (Guloglu and Kara, 2005). Following ingestion of 30 mg kg<sup>-1</sup> or 50 mL of a 21% (w/w) solution of Paraquat (as the base), hepatic, cardiac or renal failure or death may occur. Smaller doses (greater than or equal to 4 mg kg<sup>-1</sup> of Paraquat base) may cause respiratory distress, renal dysfunction or, occasionally, jaundice or adrenal cortical necrosis. When Paraquat ingestion is suspected, the drug should be removed immediately from the gastrointestinal tract by gastric lavage or by whole-gut irrigation (Hattiarachchi and Kodithuwakku, 1989). Adsorbents such as Fuller's earth, bentonite or activated charcoal may be used during gastric lavage (Sheu et al., 1998). Combined use of forced diuresis (with Furosemide, Mannitol and I.V. Dextrose in water or Normal Saline), hemodialysis or hemoperfusion is recommended until the compound cannot be detected in body fluids or the dialysate. Immediate and effective treatment is necessary to prevent systemic toxicity or death from Paraquat intoxication (Ramdial and Poon-King, 1984; Fock, 1987; Bismuth et al., 1986).

## MATERIALS AND METHODS

This is a descriptive study of 52 patients of Paraquat poisoning presenting at Shohada Ashayer hospital of Khorramabad, Lorestan, Iran. Cases were admitted through emergency department of internal medicine ward and diagnosed on the basis of history of ingestion/exposure of the compound (Eddleston *et al.*, 1998). Attendants of the patients were asked to bring the bottle through which the patient took the poison. Some of them were already carrying the container with them. Typical odor of the compound in breath and clothes were also taken into account (Gnyp and Lewandowska, 1997). Other agrochemicals, organophosphous toxins or drugs poisonings were not entertained in the study.

Data collected includes age, gender, and marital status, address, socioeconomic levels, time, dose and route of exposure of the toxic agents, frequency of different clinical features, treatment before admission, duration of hospitalization and complications. Awareness of patients about the poisonous substance was recorded and previous attempt of suicide with history of psychiatric consultation and management was also inquired (Kara *et al.*, 2002; Agarwal, 1993).

Gastric lavage and activated charcoal was done in all patients and administered according to the patient's clinical situation. In some patients dialysis started at proper time and in non of them oxygen prescribes. A large of the patients had to be put on circulatory and respiratory support. Patients admitted with intentional intake and suicide attempt were referred for psychiatric assessment. All patients and their attendants were given telephone number to obtain follow-up interview.

## RESULTS

The major characteristics of 52 patients along with mode and means of exposure of poisoning are given in Table 1. Mode of poisoning is Suicidal attempt 40 (76.9%) and accidental 2 (23.1%). Routes of exposure are ingestion 52(100%). The youngest patient was 12 years of age and the oldest was 51. The male to female ratio is 1.26/1.

Forty of cases had suicidal at temped (76.9%) and 12 of cases used this poison accidentally (23.1%).

Of 40 suicidal cases, 12(30%) used <20 mL of the poison and 7 patients (58.3 %) died, while 28 patients (70%) consumed >20 mL and 27 patients (96.4%) died.

Table 2 indicates the patient arrival time to hospital after the poisoning. The time ranged from 45 min-5 h with a median of 1 h. The longer time gap between the use of poison and the arrival in hospital determined the increased deaths. Before arrival at hospital only 50 patients received some medical aid at the periphery which uncluded gastric lavage and intravenous line.

Table 1: Characteristics of 52 patients with Paraquat poisoning

Characteristic		Num of patients (%)
Gender	Male	29(55.76)
	Female	23(44.24)
Age (years)	14	2(3.84)
	15-24	26(50)
	25-34	13(2.47)
	35-44	8(14.68)
	45-51	3(5.76)

Table 2: Time interval between poisoning and arrival at hospital in all cases of Paraguat poisoning

tuses of furadate possessing		
Num of patients	Num of death	
12 (23%)	4 (33%)	
14 (27%)	8 (60%)	
18 (34.4		
7 (13.4%)	7 (100%)	
1 (1.9%)	1 (100%)	
52 (100%)	37( )	
	Num of patients 12 (23%) 14 (27%) 18 (34.4 7 (13.4%) 1 (1.9%)	

The deaths were related to larger amounts of the poison consumed and the delayed arrival in the hospital. The most presenting symptoms and complications in 52 patients with Paraquat poisoning is headache, nausea, oral pain and dyspnea in 52 patients (100%), then tachycardia with 50(96.1%), dysphagia and lung fibrosis with 48 (92.3%), Oral ulcer 43 (82.6%), soar throat 40 (76.9%), abdominal pain 28 (53.8%), diarrhea 31 (59.6%), epistaxis, ATN, bradycardia, shock and convulsion.

### DISCUSSION

Paraquat compounds account for many suicide attempts and also many accidental poisoning each year worldwide (Gunnel and Peters, 1995; Eddleston *et al.*, 1998; Sheu *et al.*, 1998). These are the most significant poisons in Asia, being both widespread and coupled with a towering mortality rate (Hettiarachchi and Kodithuwakku, 1989). In several areas, some pesticides have become the trendiest method of suicide, gaining unsavory reputation amongst both health-care personnel and community (Rivera and Rivera, 1990; Karalliedde and Senanayake, 1988). In agricultural areas of Srilanka the agent responsible for 77% of the deaths was pesticides Nalin, 1973; Yelmos *et al.*, 1992; Casey and Vale, 1994). Self-poisoning with pesticides is uncommon in urban areas (Guloglu and Kara, 2005; Kamenczak *et al.*, 1997).

In agricultural countries like Iran and especially in some provinces like Lorestan, toxicity of pesticides as well as lack of medical services is taking its toll in the form of high case fatality rates (Hettiarachchi and Kodithuwakku, 1989; Yelmos *et al.*, 1992). Few data are available on the Paraquat compound poisons most commonly used in Lorestan. So an attempt was made to carry out a study at Khorramabad in Lorestan of Iran to observe the sociodemographic and clinical features of Paraquat intoxication.

Table 3: Correlation between source of stresses and number of Paraquat intoxications

Kind of stress	Num of patients (%)
Failure in love	13 (32.55)
Failure in exam	2 (5%)
Chronic illness	1 (2.5%)
Married friction	3 (7.5%)
Strained social	9 (22.5%)
Finantional stresses	8 (20%)
Unemployment	4 (10%)

In this study, most of the victims of poisoning were in the age group of 15-24 years. In a study at Khorramabad, Iran, patients' age with Paraquat intoxication ranged from 11-60 years with maximum number of cases between the ages of 21-30 years (Table 3).

More than half of the patients were married (54.54%) but married females were found to be more prone to self-harm as compared to married males. In Southeast Anatolian region of Turkey, OP intoxication especially affected young unmarried females, and most of them resulted from a suicidal purpose (Yelmos *et al.*, 1992; Kara *et al.*, 2002; Ozyurt *et al.*, 1997).

Majority of the enrollments happened during the month of summer reflecting the easy availability of the Paraquat compound poisoning in the peak spray season on cotton plats (Jayaratnam, 1990; Eddleston *et al.*, 2000). In Diyarbakir, Turkey, most plants intoxication cases occurred during the summer season (93-170 patients) on a monthly basis, admissions during April, May and July were most common (Ozyurt *et al.*, 1997; Sahin *et al.*, 2003).

All of patients belonged to lower middle class and only 34 patients were having qualifications up to secondary school level or above. Five cases were having previous history of suicide in the past and 2 patients were having previous history of psychiatric illness. In cases of Kara for organophosphorous intoxication, most of them had a primary education level (66.7%) and a lower socioeconomic status (58.3%) (Guloglu and Kara, 2005; Abdollahi *et al.*, 1997). Hence illiteracy and poverty are also major factors to compel the people to commit suicide (Kara *et al.*, 2002; Kamenczak *et al.*, 1997; Ingianna *et al.*, 1983).

In this study, quantity of poison ingested and time interval between poisoning and arrival at hospital were directly linked to the death. A total of 28 cases use more than 20 mL of the liquid and 26 of them died. In the same manner eleven patients reached hospital with a delay of >1 h and 29 of them died.

In contrast to the claims of relative safety of some brands, no difference was observed in the frequency of symptoms and complications in accidental cases of poisoning. Among the symptomatology, nausea, vomiting, dyspnea, oral pain and headache were more

frequent with 100% frequency. A total of 43(82.6%) patients were having oral ulcer at presentation. The frequency of these symptoms is almost similar to what reported in previous studies (Ozyurt *et al.*, 1997; Sheu *et al.*, 1998; Sahin *et al.*, 2003).

Most of the patients were having tachycardia in casualty before admission to hospital and 31(59.6%) patients experienced diarrhea about 28 of patients had abdominal pain and 7 of them had ATN with rising of creatinine. Only 3 of cases showed convulsion in this study Shock occurred in 33 (63%) of cases and all of them died. Lung fibrosis occurred in 48 patients and dysphagia reported in 48 (92.3%) cases.

The time interval between poisoning and treatment is very crucial in the prognosis of such cases. Medical personnel involved with primary care should be accustomed to deal with such problems and provide their patients with the necessary management. Only two of the patients were given some sort of therapy before shifting to our center. These findings demand a swing in emphasis in community education towards first-aid management of poisoning cases. Medical authorities should be steered to supply proper resuscitative equipment and guarantee a regular delivery of drugs to all primary health care centers. Treatment of Paraguat compound poisoning should be stressed in the syllabus for health care workers. Firm rules and regulations concerning the trade, delivery and storage of such chemicals should be followed in order to reduce the incidence of poisoning and resulting mortality.

## CONCLUSION

The death in poisoning cases depended on a variety of factors like the quantity taken, the duration between poisoning and hospitalization. Illiteracy, poverty and female gender are among the major inciting agent for suicidal impulsive behavior. Probability of improvement was high when the patient was taken to hospital as soon as possible. Confinement of unsafe pesticides away from houses will reduce the accessibility for impulsive act.

## REFERENCES

- Abdollahi, M., N. Jalali, O. Sabzevari, R. Hoseini and T. Ghanea, 1997. A retrospective study of poisoning in Tehran. Clin. Toxicol., 35: 387-393.
- Agarwal, S.B., A clinical, biochemical, neuro-behavioural and sociopsychological study of 190 patients admitted to hospital as a result of acute organophosphate poisoning. Environ. Res., 62: 63-70.
- Bismuth, C., J.M. Scherrmann, R. Garnier, F.J. Baud and P.G. Pontal, 1986. Elimination of paraquat. Dev. Toxicol. Environ. Sci., 12: 147-356.

- Casey, P. and J.A. Vale, 1944. Deaths from pesticide poisoning in England and Wales: 1945-1989. Hum Exp. Toxicol., 13: 95-101.
- Chandrasiri, N., 1999. The first ever report of homicidal poisoning by intramuscular injection of Ggramoxone (paraquat). Ceylon Med. J., 44: 36-39.
- Eddleston, M., 2000. Patterns and problems of deliberate self-poisoning in the developing world. Q. J. Med., 93: 715-731.
- Eddleston, M., M.H.R. Sheriff and K. Hawton, 1998. Deliberate self-harm in Sri Lanka: An overlooked tragedy in the developing world. BM J., 317: 133-135.
- Fock, K.M., 1987. Clinical features and prognosis of paraquat poisoning: A review of 27 cases. Singapore Med. J., 28: 53-56.
- Gnyp, L. and S.H. Lewandowska, 1997. The analysis of organophosphates poisoning cases treated at the Centre for Acute Poisonings in Lublin Provincial Hospital in 1994-1996. Przegl Lek, 54: 734-736.
- Guloglu, C. and I.H. Kara, 2005. Acute poisoning cases admitted to a university hospital emergency department in Diyarbakir, Turkey. Hum. Exp. Toxicol., 24: 49-54.
- Gunnel, D.J. and T.J. Peters, 1995. Relations between para-suicide, suicide, psychiatric admissions and socioeconomic deprivation. BMJ., 311: 266.
- Hampson, E.C. and S.M. Pond, 1988. Failure of haemoperfusion and haemodialysis to prevent death in paraquat poisoning. A retrospective review of 42 patients. Med. Toxicol. Adverse Drug Exp., 3: 64-71.
- Hettiarachchi, J. and G.C.S. Kodithuwakku, 1989. Pattern of poisoning in rural Sri Lanka. Int. J. Epidemiol., 18: 418-22.
- Ingianna, J., R. Herrero and C. Albertazzi, 1983. Estudio comparativo de casos de intoxicaciones por insecticidas organofosforados en diferentes zonas de Costa Rica. Revista. Biol. Trop., 31: 139-144.
- Jayaratnam, J., 1990. Acute pesticide poisoning: A major global health problem. World Healthstate, 43: 139-144.
- Kamenczak, A., K. Jasinska-Kolawa, D. Targosz, B. Szkolnicka and K. Sancewicz-Pach, 1997. Acute pesticides poisoning in the Krakow department of clinical toxicology in 1986-1995. Przegl Lek, 54: 671-676.
- Kara, I.H., C. Guloglu, A. Karabulut and M. Orak, 2002. Sociodemographic, clinical, and laboratory features of cases of organic phosphorus intoxication who attended the Emergency Department in the Southeast Anatolian Region of Turkey. Environ. Res., 88: 82-88.
- Karalliedde, L. and N. Senanayake, 1988. Acute organophosphorus insecticide poisoning in Sri Lanka. Forensic Sci. Int., 36: 97-100.

- Malik, G.M., M. Mubarak and D.J. Romshoo, 1998. Organophosphorous poisoning in the Kashmir valley, 1994-1997. New Engl. J. Med., 338: 1078-1079.
- Nalin, D.R., 1973. Epidemic of suicide by malathion poisoning in Guyana. Trop. Geogr. Med., 25: 8-14.
- Ozyurt, G., A. Yilmazlar and F. Tamagac, 1997. The myocardium and brain SPECT findings in organophosphorous poisoning. Eur. J. Emerg. Med., 4: 29-31.
- Ramdial, S. and T. Poon-King, 1984. High dosage cyclophosphamide and dexamethasone treatment of paraquat poisoning with 75% survival. West Indian Med. J., 33: 220-226.

- Rivera, J.A. and M. Rivera, 1990. Organophosphate poisoning. Bol. Asoc. Med. P.R., 82: 419-422.
- Sahin, H.A., I. Sahin and F. Arabaci, 2003. Sociodemographic factors in organophosphate poisonings: A prospective study. Hum. Exp. Toxicol., 22: 349-353.
- Sheu, J.J., J.D. Wang and Y.K. Wu, 1998. Determinants of lethality from suicidal pesticide poisoning in metropolitan. HsinChu Vet. Hum Toxicol., 40: 332-336.
- Yelmos, F., F. Diez, C. Martin, J.L. Blanco, M.J. Garcia, A. Lardelli and J.F. Pena, 1992. Acute organophosphate insecticide poisonings in the province of Almeria: A study of 187 cases. Med. Clin. (Barc), 98: 681-4.