HAEMATOLOGICAL PROFILE, SERUM ELECTROLYTES AND IRON LEVEL INVESTIGATIONS IN HEROIN ADDICTS

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Heroin is the most dangerous drug of abuse. The rapid increase of its abuse in Pakistan has created an alarming situation because it has destructive impact on physical and mental health of an individual. The present study showed a statistically significant decrease in haemoglabin, haematocrit, RBC count, platelets count, serum potassium, iron and total iron binding capacity in heroin addicts as compared to control subjects. There was a significant increase in the total leucocyte count and erythrocyte sedimentation rate in heroin addicts as compared to control subjects.

Keywords: Heroin; Haematological profile; Serum electrolytes; Serum iron

Introduction

Drug addiction is a worldwide problem of universal nature. Heroin is the most common drug of abuse among youth and is a widely used narcotic in Pakistan. Dependence on narcotics is increasing rapidly due to its easy availability. The use of psycho-active drugs can be traced far back through the history of man, who always seems to have used them not only to enhance pleasure and relieve pain, discomfort, frustrations on guiltiness but also to achieve social, religious and ritualistic goals and mask realities of life. The upward trend in drug dependence has been reported in recent years from most of the countries around the world as some people may take drugs more frequently, become drug dependent and thus abuse drugs. Adams and coworkers (1978) observed low platelet count, presence of a typical lymphocytes and toxic granulation of neutrophils in peripheral blood. Sapira (1968) reported a decrease in haemoglobin concentration, leucocytosis, erythrocytosis and reticulocytosis with a syndrome resembling acute immune thrombocytopenic purpura.

Sodium and potassium are concerned in several fundamental physiological processes like maintenance of normal water and acid base balance, osmotic equilibrium and neuromuscular function. The maintenance of hydration and osmotic pressure depends primarily upon total cation concentration of the body fluid. Sodium ion constitutes the largest fraction of the total cations of the extracellular fluid whereas potassium is the chief cation of the muscles and intracellular fluid (Gowenlock et al., 1990).

The circulating blood volume may increase or decrease independent of changes in the volume of other body fluid compartments as a result of abnormalities of haemodynamics, haematopoiesis and plasma protein concentration. Pathological variations in total blood and plasma volumes may be dependent upon primary changes in number and volume of the corpuscles. The marked decrease in circulating blood volume is a prominent feature of the shock syndrome which may be due to a variety of causes. This results at time in a state of haemoconcentration as evidenced by high haematocrit values and red blood cells counts and excessive water loss during vomiting, diarrhoea, intestinal fistula, pancreatic and biliary fistula (Latner, 1975).

Iron is an absolutely essential mineral both for transport of oxygen to the tissues and for

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maintenance of oxidative systems within the tissue cells without which life would cease within a few seconds (Sukkar et al., 1993).

The present study was designed to investigate haematological changes, serum electrolytes and iron concentration in heroin addicts.

Materials and Methods

Patient selection criteria

The present investigations were carried out in two groups

of subjects:

1. Heroin addict group: One hundred healthy males 20 to 45 years of age were selected with at least one year history of heroin dependence and in the absence of any significant physical illness. All the subjects were interviewed and were found to have started heroin use by smoking and subsequently began to inhale heroin vapours by "Panni method" (Javed and Ahmad, 1993). History of the individuals including their age, period of contact with heroin, daily heroin intake and major complaints were recorded. All the subjects had detailed clinical evaluation and identification of heroin was performed in the urine of each addict by thin layer chromatography (Rasheed et al., 1992). All the subjects were selected from the patients of Drug Abuse, Rehabilitation and Counselling Center, Psychiatry Department, King Edward Medical College, Mayo Hospital, Lahore, Pakistan.

2. Control group: Fifty healthy males 20 to 45 years of age were selected among the friends and relatives comparable with the same socioeconomic status of the addicts. All the control subjects had no present or past history of significant physical illness and they were not addicted to any other drug.

Sample collection

The blood and urine samples from subjects of all the heroin addicts and control group were collected when they came to the Outpatient Department of Drug Abuse Treatment Center, Psychiatry Department, Mayo Hospital, Lahore, Pakistan.

Apparatus used

Flame Photometer (Corning 410, Gallen and Camp, England), Photometer (Campur Model M 2000 CS, Diagnostic Merck), Haematology Analyser (Sequoia Cell-Dyn 800, Semiautomatic Haematology Analyser, Sequoia-Turner Corporation, California, USA).

All the reagents were supplied by Boehringer Mannheim GmbH, Diagnostica, Germany, in a ready use state.

Haematological investigations

Haematology Analyser was used to analyse the following parameters: Red Blood Cell Count (RBC), White Blood Cell Count (WBC), Haemoglobin (Hb), Haemeatocrit (Hct), Platelet Count (Plt), Mean Corpuscular Haemoglobin (MCH), Mean Corpusular Volume (MCV), Mean Corpuscular Haemoglobin Concentration, (MCHC).

Routine methods adopted by Dacie and Lewis (1984) were used for the determination of: Differential Leucocyte Count (DLC), Erythrocyte Sedimentation Rate (ESR) and Reticulocyte Count (RCC).

Serum electrolytes determination

Serum sodium and potassium concentrations were determined by a flame photometric method (Amdisen, 1967).

Serum iron determination

Determination of iron and total iron binding capacity (TIBC) were carried out by the methods performed by Trinder (1956) and Ramsay (1958) respectively. Routine urine examination was also carried out in all of the subjects.

Biostatistical analysis

Results were expressed as Mean±Standard Error of Mean (M±SEM). While two-tailed Student's t-test was applied to calculate significance of difference (Daly et al., 1991).

Results and Discussion

Major complications of heroin abuse include hepatitis, endocarditis, pulmonary disease and overdosage. Adams et al. (1978) described five patients with acute thrombocytopenia apparently due to intravenous heroin use. This syndrome was probably due to some adulterant in street heroin.

The clinical manifestations were characteristic of drug induced immune thrombocytopenia in all patients. Numerous causes exists for the development of thrombocytopenic purpura. These causes may be due to increased peripheral destruction of platelets and bone marrow depression. Infections and collagen vascular disease are frequent causes of this type of thrombocytopenic purpura. The pathogenesis appears to involve idiosyncratic production of IgG antibody directed against the drug. Quinine induced thrombocytopenic described. well has been purpura Thrombocytopenia was due to drug related

Table 1. Haematoloical status in heroin dependents and control subjects

S. No	Haematological Parameters	Heroin dependents* (N=100)	Control subjects* (N=50)	P values	Normal values
1	Haemoglobin (g/dl)	11.2±0.14	14.90±0.09	< 0.001	14-16
2 3	Total Leucocyte Count (103/ul) Differential Leucocyte Count	9.12±0.73	7.74±0.15	<0.001	4-9
	Polymorphs (%)	61.30±1.12	63.20±1.24	Not significant	55-70
	Lymphocytes (%)	34.50±0.87	34.20±0.83	Not significant	25-40
	Eosinophils (%)	2.30±0.13	1.70±0.07	< 0.001	2-4
	Monocytes (%)	2.20±0.17	1.50±0.11	<0.001	2-6
4	Red Blood Cell Morphology	Normocytic Hypochromic Normochromic	Normocytic Normochromic	 	
5	Erythrocyte sedimentation rate				
	(mm/1 st hr)	17.90±1.06	4.60±0.18	<0.001	3-7
6	Reticulocyte Count (%)	0.50±0.04	0.70±0.18	Not significant	1-2
7	Platelet Count (106/ul)	144.3±2.41	168.5±3.35	< 0.001	150-400
8	Red Blood Cell Count (106/ul)	4.60±0.05	5.20±0.04	< 0.001	4.5-5.5
9 10	Haematocrit (%) Erythrocyte Constants	42.50±0.52	45.80±0.33	<0.001	40-50
	Mean Cell Volume (fl)	93.0±1.05	88.90±0.55	<0.001	85-95
	Mean Cell Haemoglobin (pg)	24.70±0.31	28.97±0.20	<0.001	28-32
	Mean Cell Haemoglobin Conc (g/dl)	26.50±0.32	32.57±0.24	<0.001	32-36

^{* (}M±SEM)

N= Number of heroin dependents and control subjects

Table 2. Serum electrolytes in herion dependents and control subjects

/	Serum sodium (mmol/l)*	Serum potassium (mmol/l)*
Heroin dependents (N=100)	139.22±0.54	3.98±0.05
Control subjects (N=50)	139.64±0.69	4.96±0.05
P values	Not significant	<0.001
Normal values	136-148	3.8-5

^{* (}M SEM)

N= Number of heroin dependents and control subjects

Table 3. Serum iron and total iron binding capacity in heroin dependents and control subjects

Percent saturation %	Serum iron (g/dl)*	Total iron binding capacity (TIBC) (g/dl)*	Transferrin % saturation
Heroin dependents N=100	81.9±2.60	276.8±5.19	29.59
Control subjects N=50	111±2.84	345.1±7.05	32.16
P values	<0.001	<0.001	<0.01
Normal values	60-150	260-440	

^{* (}M±SEM)

N= Number of heroin dependents and control subjects

immunologic mechanism that resulted in peripheral platelet destruction. Epidemiologic considerations suggest that the common agent involved in this illness was present in the heroin they used. The use of brown heroin may be the novel event pathologically associated with the development of thrombocytopenia. Platelet count severely depressed mucocutaneous bleeding was a prominent feature of chronic hepatitis, common among heroin users has been reported associated with thrombocytopenia and anti-platelet antibody (Karpatkin et al., 1977). It is possible that chronic antigenic stimulation by i.v. heroin injection caused autoimmune platelet destruction. Immunologic distrubances have been described in heroin users including the presence of smooth muscle antibody and possibly an nonimmunologic nephropathy (Husby et al., 1975).

There may be two reasons for initially suspecting a toxic basis for thrombocytopenia, suggesting an exposure to some toxic element or recovery from reversible bone marrow injury. All the patients had both leucocytosis and reticulocytosis during the period of observati-

on.

The present study showed a statistically significant (<0.001) decrease in haemoglobin, haematocrit, red blood cell and platelet count in heroin dependents as compared to control subjects (Table 1). Sapira (1968) also observed a decrease up to 3 g/dl in haemoglobin concentration in morphine addicts which is comparable to 3.7 g/dl as observed in heroin adicts during the course of investigation. A similar observation has been reported by Paston et al., (1977) indicating low level of haemoglobin and haematocrit. Isbell (1947) and Sapira (1968) have observed the same types of results and had shown normocytic normochromic anemia in opiate addicts. There was a significant (<0.001) increase in the mean leukocyte count and erythrocyte sedimentation rate in heroin dependents as compared to control subjects. The other haematological features showed presence of a typical lymphocytes, toxic granulation of neutrophils in peripheral blood and reticulocytosis with a syndrome resembling acute immune thrombocytopenic purpura (Adams et al., 1978). Louria et al., (1967) observed leucocytosis and fever without a detectable infective lesion in heroin dependents.

Paston et al. (1977) noted 8200 x 109/L as a mean leucocyte count and 58 mmfirst hr ESR in heroin addicts. These observations lead to the conclusion that malnutrition, inadequate diet, low haemoglobin level and blood donation can cause anemia and infection in heroin addicts. Serum potassium level was significantly (<0.001) low in heroin dependents as compared to control subjects (Table 2). Pearce and Cox (1980) reported hyperkalemia in heroin addicts due to leakage of potassium ions from muscles to the extra-cellular fluid.

Tuller (1971) observed wide and rapid fluctuations in serum potassium of heroin addicts. The observed comparatively low serum potassium level may be due to an increased loss of potassium through vomiting and diarrhoea during withdrawal symptoms or it may be caused by low potassium intake due to malnutition.

Serum iron and total iron binding capacity (TIBC) significantly (<0.001) decresased in heroin dependents (Table 3) as compared to control subjects which may be due to decreased synthesis of transferrins and donating blood. Abraham (1973) also noticed severe iron deficiency in heroin addicts caused by donating excessive amount of blood.

These observations lead to the conclusion that poor nutrition, decreased level of haemoglobin and blood donation may be the cause of iron deficiency in heroin addicts.

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