Dutta D, et al., J Toxicol Cur Res 2022, 6: 023
DOI: 10.24966/TCR-3735/100023

HSOA Journal of Toxicology: Current Research

Review Article

Correlation between Clinical Features and Laboratory Parameters and Outcome in Acute Copper Sulphate Poisoning: A Cross-Sectional Observational Study

Dutta D[1], Gomes RR[2]*, Hasan FMM[3], Saha S[2] and Newaz M[4]

[1]*Ad-din Women's Medical College Hospital, Dhaka, Bangladesh*

[2]*Jashore Medical College Hospital, Jashore, Bangladesh*

[3]*Ad-din Sakina Women's Medical College Hospital, Jashore, Bangladesh*

[4]*Indoor Medical Officer, Rajshahi Medical College Hospital, Rajhshahi. Bangladesh*

Abstract

Background: Ingestion of copper sulphate is a common mode of poisoning in the Indian subcontinent. Cases are mainly suicidal in nature. The clinical course of the copper sulphate intoxicated patient is often complex involving intravascular hemolysis, jaundice and renal failure. The treatment is mainly supportive. Mortality is quite high in severe cases if treatment is not adequate and prompt.

Methods and materials: This cross sectional, observational, prospective study was conducted on 50 patients with acute copper sulphate poisoning attended in Medicine department of Khulna Medical College Hospital from January 2020 to June 2020.

Results: Almost all patients of copper sulphate poisoning, presented with gastrointestinal symptom like nausea 50(100%), vomiting 49 (98%) and abdominal pain 26 (52%). Development of anemia 17 (34%) and signs of hepatotoxicity like jaundice 18(36%), signs of dehydration 7 (14%), features of shock 8 (16%) and chest findings like tachypnoea, crepitations 9 (18%) were common. On investigation, Hemoglobin percentage below 10 gm/dl in 12 (24%), neutrophillic leukocytosis 18 (36%), Platelet count below 100000/cu mm blood in 3 (6%). features of hemolysis 16 (32%), haemoglobinuria 17 (34%) and haematuria15 (30%), S. bilirubin was elevated in 23 (46%) of patients. Serum ALT level was elevated in 17 (34%) of patients of which 13 (26%) were found as moderate to severely raised. both serum creatinine and blood urea were elevated in 16 (32%) of patients.

Conclusion: Acute copper sulphate poisoning is common in our country which is mainly suicidal in nature. As death occurs due to multi organ failure, while gastrointestinal, intravascular hemolysis, hepatic and renal toxicities significantly contribute to it. Management in High dependency unit is also required.

Keywords: Hemoglobinuria; Hematuria; Hepatotoxicity; Intravascular hemolysis; Renal failure

Introduction

Copper sulphate forms bright blue crystals containing five molecules of water [CuSO4 .5H 2 O]. It is commonly known as "Blue Vitriol" or "Blue Stone". Usually the people of the southern region of Bangladesh uses copper sulphate as a fungicidal agent [1]. It is used chiefly for agricultural purposes as a pesticide and in leather industry. It was also being used as a precipitator in heavy metal poisoning and was used to treat gastric and topical exposure to phosphorous. It has a nauseous and metallic taste. Solutions are acid to litmus, freely soluble in water [2,3]. It is consumed mainly with suicidal intentions. Accidental poisonings have been reported from children as well [4,5].

Approximately 30% of ingested copper is absorbed from the gastrointestinal tract [6]. In blood, copper is initially albumin-bound and transported via the hepatic portal circulation to the liver where it is incorporated into ceruloplasmin. Copper is present in serum in two forms; 93% is tightly bound to ceruloplasmin and 7% is loosely bound to albumin [7]. The copper-albumin complex represents the toxicological active portion of the serum copper [2]. Systemic transport of copper from liver is primarily as ceruloplasmin, which appears to donate copper to tissues. Copper is distributed to all tissues with the highest concentrations in liver, heart, brain, kidneys and muscle. Intracellular copper is predominantly bound to metallothionein. Fecal and biliary excretion accounts for 80 percent of excreted copper. Approximately four percent is excreted in the urine [6].

In acute poisoning, albumin, rather than ceruloplasmin, binds the excess copper. The liver is the major site of deposition of copper following large ingestion. Lethal dose is about 10-20g [7]. The copper content in normal adult liver ranges from 18-45 mg/g dry weight. When the concentration of hepatic copper is greater than 50 mg/g dry weight, liver cell necrosis occurs with release of large amount of copper into the serum. This released copper is rapidly taken up by erythrocytes and results in oxidative damage and may result in hemolysis of RBCs [2]. It is proposed that free reduced copper in the cell binds to sulfhydryl groups and inactivates enzymes such as glucose-6-phosphate dehydrogenase and glutathione reductase [8]. Intravascular hemolysis appears 12-24h following ingestion of copper sulphate.

***Corresponding author:** Gomes RR, Jashore Medical College Hospital, Jashore, Bangladesh, E-mail: rrichi.dmc.k56@gmail.com

Citation: Dutta D, Gomes RR, Hasan FMM, Saha S, Newaz M (2022) Correlation between Clinical Features and Laboratory Parameters and Outcome in Acute Copper Sulphate Poisoning: A Cross-Sectional Observational Study. J Toxicol Cur Res 6: 023.

Received: May 03, 2022; **Accepted:** May 11, 2022; **Published:** May 18, 2022

Jaundice in copper sulphate poisoning is partly hepatic in origin in addition to hemolysis [9]. Jaundice appears on the second or third day following ingestion. Liver damage has been attributed to liver mitochondrial dysfunction due to oxidized state. Intravascular hemolysis plays a major role in the pathogenesis of renal failure [8-10]. The heam pigment released due to hemolysis and direct toxic effect of copper released from lysed red cells contribute to tubular epithelial damage of the kidney. Severe vomiting, diarrhoea, lack of replacement of fluid and gastrointestinal bleed, leading to hypotension could also contribute to renal failure [10]. Renal complications are usually seen on the third or the fourth day and onwards after the poisoning [11].

The incidence of copper sulphate poisoning varies at different geographical areas depending on the local use of copper sulphate and the availability of other suicidal poisons. Diagnosis is based on history of exposure. Mainstay of treatment is supportive, including careful fluid therapy and vasopressors as needed, blood transfusion if needed and methylene in symptomatic methemoglobinemia. Chelation therapy given in most cases. The role of dialysis is limited to the management of associated renal failure. High mortality is noted in severe cases due to methemoglobinemea, hepatotoxicity and renal failure.

Materials and Methods

This cross sectional, observational, prospective study was carried out in Medicine department of Khulna Medical College Hospital (KMCH) from Januray, 2020 to June 2020. Total 50 cases of acute copper sulphate poisoning were selected. Data were processed and analyzed using SPSS (Statistical Package for Social Science) 15.0.

Inclusion criteria

- All patients with acute copper sulphate poisoning admitted in Medicine wards of KMCH.
- Voluntarily given consent.

Exclusion criteria

- Not willing to give informed consent.
- Patient is suffering from Wilson's disease, chronic liver disease and chronic kidney disease.

Results

Table 1 shows that almost all patients of copper sulphate poisoning, presented with gastrointestinal symptom like abdominal pain 26 (52%), nausea 50 (100%), and vomiting 49 (98%) A significant proportion of patients presented with high coloured urine 20 (40%) which developed initially and subsequently.

Presenting Feature	No. of Patient	Percentage (%)
Nausea	50	100(%)
Vomiting	49	98(%)
Abdominal pain	26	52(%)
High coloured urine	20	40(%)
Jaundice	8	16(%)

Table1: Showing clinical presentation of the patients.

Table 2 shows that anemia 17 (34%) and jaundice 18 (36%) developed on 2nd or 3rd day following copper sulphate poisoning. Development of signs of dehydration 7 (14%), features of shock 8 (16%) and chest findings like tachypnoea, crepitations 9 (18%) were not uncommon.

Physical findings on subsequent days	No. of patients	Percentage (%)
Anemia	17	34 %
Jaundice	18	36 %
Signs of dehydration	7	14 %
Features of shock	8	16 %
Chest findings(e,g - crepitations)	9	18 %

Table 2: Showing physical findings on subsequent days during hospital stay.

Table 3 shows that anemia is a common feature. Hemoglobin percentage was found below 10 gm/dl in 12 (24%) of patients. It is due to hemolysis or blood loss. Neutrophilic Leukocytosis were found on 18 (36%) of patients. Platelet count was found below 100000/cu mm blood in a minority patient 3 (6%).

Complete Blood Count		No. of patients	Percentage (%)
Percentage of hemoglobin	<10 gm/dl	12	24.0 %
	>10 gm/ dl	38	76.0 %
TC of WBC	>11000/cu mm	18	36.0 %
	<11000/ cu mm	32	64.0 %
DC of WBC (Percentage of Neutrophil)	>70%	18	36.0 %
	<70%	32	64.0 %
Platelet count	<100000 /cu mm	3	6.0 %
	>100000/cu mm	47	94.0 %

Table 3: Complete Blood Count among the patients.

Table 4 shows that features of hemolysis was present on 16 (32%) of patient.

PBF findings	Frequency	Percent
Features of hemolysis	16	32.0%
Normal study	34	68.0%
Total	50	100.0%

Table 4: Peripheral blood film among the patients.

Table 5 shows that hemoglobinuria and hematuria developed in 17 (34%) and 15 (30%) of the patients respectively.

Urine Analysis		No. of patients	Percentage (%)
Albuminuria	present	5	10.0 %
	Absent	45	90.0 %
Hemoglobinuria	present	17	34.0 %
	Absent	33	66.0 %
Hematuria	present	15	30.0 %
	absent	35	70.0 %

Table 5: Urine analysis among the patients.

Table 6 shows that serum bilirubin was elevated in 23 (46%) of patients. Serum ALT level was elevated in 17 (34%) of patients of which 26% were found as moderate to severely raised. LFT were done at the 4th day of copper sulphate ingestion.

Liver Function Test		No. of patients	Percentage (%)
Serum bilirubin	Increased (>1.1 mg/dl)	23	46.0
	Normal (up to 1.1 mg/dl)	27	54.0
Serum alanine aminotransferase (ALT)	Moderate to severely raised (>80 u/l)	13	26.0
	Mildly raised (41-80 u/l)	4	8.0
	Normal (up to 40u/l)	33	66.0

Table 6: Liver function test among the patients.

Table 7 shows that both serum creatinine and blood urea were elevated in 16 (32%) of patients. RFT were done at the 4th day of copper sulphate ingestion.

Renal Function Test		No. of patients	Percentage (%)
Serum creatinine	Increased (>1.2 mg/dl)	16	32.0 %
	Normal (up to 1.2 mg/ dl)	34	68.0 %
Blood urea	Increased (>1.2 mg/dl)	16	32.0 %
	Normal (upto 1.2 mg/ dl)	34	68.0 %

Table 7: Renal function test among the patients.

Among 16 patients of raised serum creatinine, 14 patients improved and 2 patients died. Among 34 patients of normal serum creatinine, all of them (100%) improved. There is statistically significant association (p-value=0.035) between raised serum creatinine and outcome of treatment of patients in acute copper sulphate poisoning (Table 8).

	Serum creatinine			
Outcome of treatment of patient	Increased (>1.2 mg/dl)	Normal (upto 1.2 mg/ dl)	Total	P-Value
Improved	14	34	48	
Death	2	0	2	0.035
Total	16	34	50	

Table 8: Co-relation between serum creatinine and outcome of treatment.

Chi-square (χ^2) Test was employed to analyse the data. P-value <0.05 was considered statistically significant. Among 13 patients of Moderate to severely raised (>80 u/l) serum ALT, 11 patients had improved and 2 patients had died. Among 4 patients of mildly raised (41-80 u/l) serum ALT and 33 patients of normal serum creatinine, all of them (100%) had improved. There is statistically significant association (p-value=0.05) between raised serum ALT and outcome of patients in acute copper sulphate poisoning (Table 9).

Chi-square (χ^2) Test was employed to analyze the data. P-value <0.05 was considered statistically significant. Among 16 patients with features of hemolysis on peripheral blood film, 14 patients improved and 2 patients died. Among 34 patients with normal peripheral blood film, all patients (100%) improved. There is statistically significant association (p-value=0.035) between presence of features of hemolysis on peripheral blood film and outcome of patients in acute copper sulphate poisoning (Table 10).

	Outcome of patient			
Serum alanine aminotransferase (ALT)	Improved	Death	Total	P-Value
Moderate to severely raised (>80 u/l)	11	2	13	
Mildly raised (41-80 u/l)	4	0	4	0.05
Normal (up to 40u/l)	33	0	33	
Total	48	2	50	

Table 9: Co-relation between serum alanine aminotransferase (ALT) and outcome of treatment of patient.

	Outcome of patient			
Peripheral blood picture	**Improved**	**Death**	**Total**	**P-Value**
Features of hemolysis	14	2	16	
Normal study	34	0	34	0.035
Total	48	2	50	

Table 10: Co-relation between features on peripheral blood picture and outcome of patient.

Chi-square (χ2) Test was employed to analyze the data. P-value <0.05 was considered statistically significant.

Discussion

Acute copper sulphate poisoning is a common mode of poisoning in southern region of Bangladesh which are mainly suicidal in nature [1] usually the people of this region use copper sulphate as a fungicidal agent. The incidence of acute copper sulphate poisoning varies at different geographical areas depending on the local use of copper sulphate and the availability of other suicidal poisons. The lowest dose copper sulphate that is toxic when ingested is 11mg /kg [12]. Akintonwa et al., 1996 [13] claimed 10-20 gm. copper sulphate to be a "definitely fatal" dose. In a review of 123 cases Ahsan et al., 1994 [1] observed an "unpredictable" outcome in those consuming less than 50 gm while 100 gm was "invariably" fatal. Following ingestion of copper sulphate gastrointestinal symptoms appear first and is always present. In present study, gastrointestinal symptoms like nausea 50 (100%), and vomiting 49 (98%), abdominal pain 26 (52%) were observed in patients, which is consistent with the previous studies [SH] Mollick et al., 2011 [14], Chowdhury et al., 1961 [15], singh and singh et al., 1968 [9],Chugh KS et al ., 1977 [10].

Jaundice developed on 2nd or 3rd day following ingestion of copper sulphate. Clinically it is detected in 36% patients (18 out of 50). Jaundice may be hemolytic or hepatocellular or mixed. Serum bilirubin was elevated in 23 (46%) of patients. In previous study, Wahal et al., 1963 [16] jaundice was found on 36% of patient and Agarwal SK et al., 1993 [17] jaundice was found 58% of patients. Serum ALT level was elevated in 17 (34%) of patients of which 13 were found as moderate to severely raised which is consistent with Singh M et al., 1968 [9], Ashraf I et al., 1970 [18], Wahal PK et al., 1976 [16]. In a study, SH Mollick et al., 2011 [14] stated Serum ALT & serum bilirubin were elevated in 40% and 37% patients respectively. Among

13 patients of Moderate to severely raised (>80 u/l) serum ALT, 11 patients improved and 2 patients died. Among 4 patients with mildly raised (41-80 u/l) serum ALT and 33 patients with normal serum ALT, all of them (100%) had improved. There is statistically significant association (p-value=0.05) between raised serum ALT and outcome of patients in acute copper sulphate poisoning (Table 9).

Intravascular hemolysis can start as early as within the first 24 hours since ingestion and is due to the direct oxidative damage to erythrocyte membranes. The hemolysis can be rapid and severe with drastic drops in the hemoglobin level. In this study, Hemoglobin percentage was found below 10 gm/dl in 12 (24%) of patients. Which may be due to copper induced hemolysis (increased total bilirubin and evidence from blood picture). A significant proportion of patients developed high coloured urine 20 (40%). which may be due to hemolysis or jaundice. Hemoglobinuria, hematuria and albuminuria were developed in 17 (34%), 15 (30%) and 5 (10%) of the patients respectively. In previous study, Chugh KS et al., 1977 [14] and Agarwal SK et al., 1993 [17] found intravascular hemolysis ranged from 47-65% in two case series. Among 16 patients with features of hemolysis on peripheral blood film, 14 patients improved and 2 patients died. Among 34 patients of normal peripheral blood film, all of them (100%) improved. There is statistically significant association (p-value=0.035) between presence of features of hemolysis on peripheral blood film and outcome of patients in acute copper sulphate poisoning (Table 10).

Renal complications were observed usually after 48hours. The possible mechanisms of kidney damage include; pre-renal failure due to dehydration (vomiting, diarrhea, reduced fluid intake), hemoglobinuria, sepsis, rhabdomyolysis, direct copper toxicity on proximal tubules and secondary effects of multi organ dysfunction. In this study, serum creatinine and blood urea were elevated in 16 (32%) patients. High coloured urine developed in 20 (40%) patients (it may be due to hematuria and/or hemoglobinuria). Hemoglobinuria, hematuria and albuminuria developed in 17 (34%), 15(30%) and 5 (10%) patients respectively.

In some previous studies, Dash SC et al., 1989 [8] , Chugh KS et al., 1977 [10] and Mehta A et al., 1985 [11] described that acute renal failure developed in 20-40% of patients with acute copper sulphate poisoning and urinary abnormalities detected are oliguria, anuria, albuminuria, hemoglobinuria and hematuria. Among 16 patients with raised serum creatinine, 14 patients improved and 2 patients died. Among 34 patients with normal serum creatinine, all of them (100%) improved. There is statistically significant association (p-value=0.035) between raised serum creatinine and outcome of patients in acute copper sulphate poisoning (Table 8).

Conclusion

Copper sulphate is a common form of suicidal agent with significant mortality. It is a readily available substance can be taken easily. Acute copper sulphate poisoning is a rare event and uncommon worldwide except Indian subcontinents. It is most prevalent in southern region of Bangladesh. Oral copper sulphate ingestion has significant effects on gastrointestinal, hematological, hepatic and renal system. Gastrointestinal symptoms like nausea, vomiting, abdominal pain frequently occurs and appears .Intravascular hemolysis usually developed within 12 - 24 hours of ingestion. Appearance of jaundice is common. Most common renal toxicities are hematuria, hemoglobinuria, albuminuria, elevated blood urea, elevated serum creatinine level and acute renal failure. Although death occurs due to multi organ failure, gastrointestinal, intravascular hemolysis, hepatic and renal toxicities significantly contributes to it.

Limitations of Sudy

The sample size was relatively small. No follow-up could be carried out to repeat the investigations. Actual measurement of dose of copper sulphate is not possible. Advanced investigation facilities are limited. No advanced life supports were available.

Funding

Self-funded.

Conflict of Interests

None declared.

References

1. Ahasan HA, Chowdhury MA, Azhar MA, Rafiqueuddin AK (1994) Copper sulphate poisoning. Trop Doct 24: 52-53.
2. Barceloux DG (1999) Copper. J Toxicol Clin Toxicol 37: 217-230.
3. Gupta PS, Bhargava SP, Sharma ML (1962) Acute copper sulphate poisoning with special reference to its management with corticosteroid therapy. J Assoc Physicians India 10: 287-292.
4. Sharma NL, Singh RN, Natu NK (1967) Accidental poisoning in infancy and childhood. J Indian Med Assoc 48: 20-25.
5. Blundell S, Curtin J, Fitzgerald D (2003) Blue lips, coma and haemolysis. J Paediatr Child Health 39: 67-68.
6. Dowdy RP (1969) Copper metabolism. Am J ClinNutr 22: 887-892.
7. Ellenhorn MJ (1997) Ellenhorn's Medical Toxicology: Metals and Related Compounds. In : Diagnosis and treatment of human poisoning 2: 1554-1556.
8. Singh M, Singh G (1968) Biochemical changes in Blood in cases of acute copper sulphate poisoning. J Indian Med Assoc 50: 549-554.
9. Chugh KS, Sharma BK, Singhal PC (1977) Acute renal failure following copper sulphate intoxication. Postgrad Med J 53: 18-23.
10. Mehta A, Patney NL, Bhati DP, Singh SP (1985) Copper sulphate poisoning - Its impact on Kidneys. J Indian Med Assoc 83: 108-110.
11. Mehta A, Patney NL, Bhati DP,Singh SP (1985) Copper sulphate poisoning - Its impact on Kidneys. J Indian Med Assoc 83:108-110.
12. http://extoxnet.orst.edu/pips/coppersu.htm
13. Akintonwa A, MebadejeAFB, Odutola TA (1989) Fatal poisonings by copper sulphate ingested from "spiritualwater". Vet Hum Toxical 31: 453-454.
14. Mollick SH, Mollick KA, Bakar A, Miah M (2011) Burden and outcome of acutecoppersulphate poisoning in a teaching hospital Bang Med J (Khulna) 44 : 7-10.
15. Chowdhury AK, Ghosh S, Pal D (1961) Acute copper sulphate poisoning. J Indian Med Assoc 36: 330-336.
16. WahalPK, Lahiri B, Mathur KS, Kehar U, Wahi PN (1963) Acute copper sulphate poisoning. J Assoc Physicians India 11: 93-103.
17. Agarwal SK, Tiwari SC, Dash SC (1993) Spectrum of poisoning requiring hemodialysis in tertiary care hospital in India. Int J Artif Organs16: 20-22.
18. Ashraf I (1970) Hepatic derangements (biochemical) in acute copper sulphate poisoning. J Indian Med Assoc 55: 341-342.

Advances In Industrial Biotechnology | ISSN: 2639-5665

Advances In Microbiology Research | ISSN: 2689-694X

Archives Of Surgery And Surgical Education | ISSN: 2689-3126

Archives Of Urology

Archives Of Zoological Studies | ISSN: 2640-7779

Current Trends Medical And Biological Engineering

International Journal Of Case Reports And Therapeutic Studies | ISSN: 2689-310X

Journal Of Addiction & Addictive Disorders | ISSN: 2578-7276

Journal Of Agronomy & Agricultural Science | ISSN: 2689-8292

Journal Of AIDS Clinical Research & STDs | ISSN: 2572-7370

Journal Of Alcoholism Drug Abuse & Substance Dependence | ISSN: 2572-9594

Journal Of Allergy Disorders & Therapy | ISSN: 2470-749X

Journal Of Alternative Complementary & Integrative Medicine | ISSN: 2470-7562

Journal Of Alzheimers & Neurodegenerative Diseases | ISSN: 2572-9608

Journal Of Anesthesia & Clinical Care | ISSN: 2378-8879

Journal Of Angiology & Vascular Surgery | ISSN: 2572-7397

Journal Of Animal Research & Veterinary Science | ISSN: 2639-3751

Journal Of Aquaculture & Fisheries | ISSN: 2576-5523

Journal Of Atmospheric & Earth Sciences | ISSN: 2689-8780

Journal Of Biotech Research & Biochemistry

Journal Of Brain & Neuroscience Research

Journal Of Cancer Biology & Treatment | ISSN: 2470-7546

Journal Of Cardiology Study & Research | ISSN: 2640-768X

Journal Of Cell Biology & Cell Metabolism | ISSN: 2381-1943

Journal Of Clinical Dermatology & Therapy | ISSN: 2378-8771

Journal Of Clinical Immunology & Immunotherapy | ISSN: 2378-8844

Journal Of Clinical Studies & Medical Case Reports | ISSN: 2378-8801

Journal Of Community Medicine & Public Health Care | ISSN: 2381-1978

Journal Of Cytology & Tissue Biology | ISSN: 2378-9107

Journal Of Dairy Research & Technology | ISSN: 2688-9315

Journal Of Dentistry Oral Health & Cosmesis | ISSN: 2473-6783

Journal Of Diabetes & Metabolic Disorders | ISSN: 2381-201X

Journal Of Emergency Medicine Trauma & Surgical Care | ISSN: 2378-8798

Journal Of Environmental Science Current Research | ISSN: 2643-5020

Journal Of Food Science & Nutrition | ISSN: 2470-1076

Journal Of Forensic Legal & Investigative Sciences | ISSN: 2473-733X

Journal Of Gastroenterology & Hepatology Research | ISSN: 2574-2566

Journal Of Genetics & Genomic Sciences | ISSN: 2574-2485

Journal Of Gerontology & Geriatric Medicine | ISSN: 2381-8662

Journal Of Hematology Blood Transfusion & Disorders | ISSN: 2572-2999

Journal Of Hospice & Palliative Medical Care

Journal Of Human Endocrinology | ISSN: 2572-9640

Journal Of Infectious & Non Infectious Diseases | ISSN: 2381-8654

Journal Of Internal Medicine & Primary Healthcare | ISSN: 2574-2493

Journal Of Light & Laser Current Trends

Journal Of Medicine Study & Research | ISSN: 2639-5657

Journal Of Modern Chemical Sciences

Journal Of Nanotechnology Nanomedicine & Nanobiotechnology | ISSN: 2381-2044

Journal Of Neonatology & Clinical Pediatrics | ISSN: 2378-878X

Journal Of Nephrology & Renal Therapy | ISSN: 2473-7313

Journal Of Non Invasive Vascular Investigation | ISSN: 2572-7400

Journal Of Nuclear Medicine Radiology & Radiation Therapy | ISSN: 2572-7419

Journal Of Obesity & Weight Loss | ISSN: 2473-7372

Journal Of Ophthalmology & Clinical Research | ISSN: 2378-8887

Journal Of Orthopedic Research & Physiotherapy | ISSN: 2381-2052

Journal Of Otolaryngology Head & Neck Surgery | ISSN: 2573-010X

Journal Of Pathology Clinical & Medical Research

Journal Of Pharmacology Pharmaceutics & Pharmacovigilance | ISSN: 2639-5649

Journal Of Physical Medicine Rehabilitation & Disabilities | ISSN: 2381-8670

Journal Of Plant Science Current Research | ISSN: 2639-3743

Journal Of Practical & Professional Nursing | ISSN: 2639-5681

Journal Of Protein Research & Bioinformatics

Journal Of Psychiatry Depression & Anxiety | ISSN: 2573-0150

Journal Of Pulmonary Medicine & Respiratory Research | ISSN: 2573-0177

Journal Of Reproductive Medicine Gynaecology & Obstetrics | ISSN: 2574-2574

Journal Of Stem Cells Research Development & Therapy | ISSN: 2381-2060

Journal Of Surgery Current Trends & Innovations | ISSN: 2578-7284

Journal Of Toxicology Current Research | ISSN: 2639-3735

Journal Of Translational Science And Research

Journal Of Vaccines Research & Vaccination | ISSN: 2573-0193

Journal Of Virology & Antivirals

Sports Medicine And Injury Care Journal | ISSN: 2689-8829

Trends In Anatomy & Physiology | ISSN: 2640-7752

Submit Your Manuscript: https://www.heraldopenaccess.us/submit-manuscript

Herald Scholarly Open Access, 2561 Cornelia Rd, #205, Herndon, VA 20171, USA.
Tel: +1 202-499-9679; E-mail: info@heraldsopenaccess.us
http://www.heraldopenaccess.us/