International Journal of Research in Engineering, Science and Management
Volume 5, Issue 7, July 2022
https://www.ijresm.com | ISSN (Online): 2581-5792

Kidney Function Test Prevalence for Pesticide Handlers and Non-Pesticide Handlers

K. K. Dhanushka[1*], M. Thiruselvi[2]
[1]III B.Sc., Dr. NGP Arts and Science College, Coimbatore, India
[2]Dr. NGP Arts and Science College, Coimbatore, India

***Abstract*: Kidney is major to excrete the waste product. Excreting is a major process that must to be done to avoid the accumulations, because accumulation of waste product will lead to the formations of certain disease. Accumulations, because accumulation of waste product will lead to the formations of certain disease. The mechanism of stone formation is a complex process which results from several physicochemical events including super saturation, constituents within tubular cells. These steps are modulated by an imbalance between factors that promote or inhibit urinary crystallization. It is also noted that cellular injury promotes retention of particles on renal papillary surfaces. The exposure of renal epithelial cells to oxalate causes a signaling cascade which leads to apoptosis by p38 mitogen-activated protein kinase pathways. Kidney disease is also happened due to exposure of certain pesticides. Some pesticides were able to cause diseases like Diabetes mellitus, obesity, cancers, End stage renal disease. Organophosphates is used as insecticide in world, it accounts for more than 40% of pesticide market, it inhibits cholinsterase value in Livers. With this HbA1C level also increased in pesticide handlers. 20 Samples were collect among them 10 are pesticide exposed person and another 10 are non-pesticide exposed person and the level of serum urea and creatinine levels are checked.**

***Keywords*: Kidney function test, Pesticide handlers.**

1. Introduction

The impact of long-term pesticide exposure on human kidney function remains largely unknown. Pesticide exposure has been linked to variety of adverse health outcomes, including (1) Diabetes mellitus, (2) Gestational diabetes, (3) Obesity, (4) Cancer, (5) respiratory disease, while pesticide is associated with diseases that may contribute End Stage Renal Disease (ESRD) [Lebov, 2014]. End Stage Renal Disease is the final stage of chronic kidney disease (CKD), which is often debilitating in later stages of disease. Kidney disease is characterized by kidney dysfunction, kidney damage or both. Kidney dysfunction is defined as excretory capacity of the kidney. Healthy kidneys filter blood for waste products, which are excreted in the urine. Kidney dysfunction is measurement of metabolic by products, including urea, creatinine in blood and urine. Serum creatinine is used to estimate GFR which is best indicator of overall renal function. Urea is a waste product of metabolism that is excreted by the kidney in urine [Devis, 2017]. Kidney disease is associated with reduced urea excretion and consequent rise in blood concentration. There are many non-renal causes that can be associated with such a rise and for some of these conditions, (E.) Heart failure, dehydration is common. By comparison with urea, creatinine measurement is no more sensitive but is more specific Exposure to pesticides may produce biochemical changes even before the clinical health manifestations that may appear in the sprayers. These biochemical changes may result from destructive and degenerative changes caused by pesticides in many organs including kidney. Various studies had indicated that farm workers exposed to pesticide showed significant increase in serum concentrations of urea, creatinine [Yassin and shanti, 2016]. The normally functioning kidney controls the concentration of body fluids. It accomplishes this by excreting excessive amounts of water in the urine if body fluids are too dilute or by excreting excessive solutes when body fluids are too concentrated. Another important function is acid-base balance. The kidney also has a hormonal role. They are in part responsible for the conversion of vitamin D to its active metabolite, which is important in the absorption of calcium from the intestine. Erythropoietin is manufactured by the kidney and stimulates the bone marrow to produce red blood cell [YJ Temple, 2011). Special cells in the kidney monitor the oxygen concentration in blood. If oxygen levels fail, erythropoietin levels rise and the body starts to manufacture more red blood cells. With renal failure there is decreased production of this hormone and anemia results. As failure progresses the kidney is less able to maintain a steady volume and concentration of body fluids. As waste products accumulate, patient may have problems with fatigue, headaches, nausea, vomiting and decreased appetite resulting in weight loss, Practically, most patients will start dialysis when the creatinine clearance (CRCI) is very low, 3-5 cc per minute (normal 100 cc per minute) and serum creatinine is greater than 12-14 mg/dl (normal 100 mg/dl). Acetylcholinestrease (AchE) inhibiting insecticides are the most widely used pesticide [John and wanigasuriya et. al., 2006]. There seems to be an association between organophosphate pesticide exposure and hyperglycemia with mechanistic linkages through disruption of glycogenesis, glycolysis, glycogenolysis, gluconeogenesis, hormonal disruption and oxidative stress. Organophosphates has become a largely used insecticide in the world, accounting

*Corresponding author: dhanushkad34@gmail.com

for more than 40% of pesticide market, it inhibits the cholinesterase value in liver. With this HbA1c level also increased in the pesticide handlers [Velmurugan, et. al., 2017). The liver enzymes has broad substrate specificity including a variety of pesticide oxidations. It has been reported to be significantly increased in humans occupationally exposed to pesticides [Zeinalov and Gorkin, 1990]. Many researchers tried to correlate various enzymes with the harmful effects of pesticides, especially in case of ALT, AST, ALP, Acetylcholinestrease [Altuntes et. al., 2002]. The participants were divided into two groups those with direct pesticide exposure (Farmers) and the ones with indirect exposure (People not associated with agricultural activities). Individuals with family history of diabetes were not part of the study. 20 Blood samples were analysed for Glycosylated haemoglobin (A marker for determining the average blood glucose levels in last three months) Kidney function test and liver function test, The Prevalence percentage is compared with pesticide handlers and non-pesticide handlers.

A. Aim

To study the kidney function test prevalence between pesticide handlers and non-pesticide handlers.

B. Objectives

To assess the amount of serum urea and serum creatinine in farm workers with pesticide handlers and non-pesticide handlers from annur. Pesticides are widely used in agricultural field. The toxicity can vary greatly by, the type of pesticide, the length of exposure and route of exposure. Damage or injury to kidneys caused by exposure to pesticides. Symptoms may be acute, sub-acute or chronic depending on the severity of the toxicity. Lack of awareness in handling of pesticides will leads to lots of health effects. High exposure of pesticides in blood leads to kidney dysfunction or damage. Our study aims to shows, the prevalence of kidney function test increased in those who are handling with pesticides.

Evidence for Relationship Between Pesticide Exposure and ESRD:

Kidney receives 20-25% of cardiac output. This large blood flow results in high concentrations of toxicant delivery to the kidney. The kidney is the primary organ for secrete the xenobiotics compounds. The renal enzyme can concentrate and metabolize xenobiotic compound in the kidney. The urine concentrating ability of the kidney may further increase the concentration of toxicants to the kidney, its leads to the obstruction of tubular flow and damage to nephron and tubular cells

Pesticide poisoning case studies, animal model, and in vitro laboratory research provide evidence for damaging effect of both acute and chronic pesticide exposure on renal failure. The most commonly reported pesticides that affects the human renal damage are organophosphates, acute kidney injury (AKI), a sudden (with 48 hours) reduction in kidney function, is frequently observed outcome of pesticide poisoning.

Evidence suggests that pesticide exposure may harm the kidney through oxidative stress and resulting cell damage. Exposure to a variety of Pesticides is associated with the generation of reactive oxygen species (ROS) and altered activity in animal models and humans [Abdollahi et, al., 2004). In vitro condition the rats having increased antioxidant enzyme activity has been observed in pesticide exposure. Insecticide exposure among agricultural workers have observed a decreased in antioxidant enzyme activity. The decreased in these enzymes may indicate an inhibition of antioxidant enzymes resulting from binding of oxidant molecules produce during pesticide metabolism [Lopez et. al., 2007). Enzyme changes indicate the oxidative stress among the patients with chronic renal failure

This study provides evidence for an association between ESRD risk and chronic exposure to specific chemicals among pesticide applicators. This study suggests that pesticide exposure resulting in medical visits increase the risk of incident ESRD, raising concerns that multiple high-level pesticide exposures may contribute to irreversible kidney damage and resultant disease, caution should be taken in interpreting results, when diagnosis date or disease severity information is not available, because the healthy worker survivor effect may bias estimates towards the null (Lebov et al., 2016).

Effect of Pesticide on Kidney Function Test:

The study population comprised with farm workers using pesticide and non-exposed controls. Effect of pesticides on serum urea, creatinine and uric acid were significantly increased in farm workers compared to controls. Total protein, albumin and globulin of pesticides-exposed farm workers were significantly higher than those controls. The significant increase was observed for albumin but in farm workers groups aged less than 30 years and 31-45 years. Impaired glomerular filtration, urinary tract obstruction and kidney damage were seen in those who are exposed to pesticides.

Pesticides through impairment of protein synthesis by hepatocytes and disturbance of kidney function. Elevated serum urea observed in the study in response to pesticides exposure may explain by: (1) Impairment in it synthesis as a result of impaired hepatic function. (2) disturbance in protein metabolism as found in the results. (3) decrease in the filtration rate of kidney, it was documented that excessive exposure to pesticides caused cytototoxic changes in hepatic and biochemical markers.

Elevation of urea and creatinine in pesticide exposed workers on the basis of tubular renal insufficiency, impaired glomerular filtration, urinary tract obstruction and kidney damage [Yassin and shanti, 2016].

Exposure to Ache – Inhibiting Pesticides and Chronic Renal Failure

In this study, Chronic Renal Failure patients (both exposed and unexposed) were taken. Exposure to AChE- inhibiting pesticides was estimated by measuring hemoglobin correlated red cell AChE levels using cholinestrease test system. AChE levels among farmers occupationally exposed to pesticides were significantly lower than the unexposed controls. Red cell ACHE levels were lower in Chronic Renal Failure (Exposed and unexposed groups), as compared to the exposed non-Chronic Renal Failure farmers and the controls. The patients

with chronic Renal Failure, red cell AChE levels were lower in the exposed group, as compared to the unexposed group.

The ratio were taken between the exposed-non Chronic Renal Failure to the exposed Chronic Renal Failure patients. Non-Chronic Renal Failure exposed farmers having long term low-level exposure to organophosphate had significantly lower levels of compared to the controls [John et. al., 2006].

Chronic Exposure to Commonly Used Insecticide Causes Diabetes:

This study clearly shows the prevalence of diabetic conditions mediated by microbial degradation of pesticide in humans. The researchers found evidence that chronic exposure to organophosphate insecticides induces diabetes and impaired glucose tolerance in both humans and mice. The researchers found that organophosphate induced diabetes was mediated by gut bacteria. In this study. 3000 peoples were taken and the prevalence of diabetes was higher in people who are directly exposed to the pesticides when compared to non-exposed peoples

Chronic exposure of organophosphate led to diabetes, the researchers treated the mice with organophosphate, then they saw an increase in blood sugar but they didn't see any correlation between insecticide and neurotransmitter. Acetylcholinestrease level in mice treated with pesticide. "The neurotransmitter is the main target of pesticides". In this, they were confident that the pesticide inducing diabetes through a new route. So, the researchers collected faecal material from mice for 180 days and transplanted it to a new set of mice. The new set of mice developed diabetes in just one week. While, the control did not. "Then they concluded that organophosphate induced diabetes was mediated by gut bacteria". So, they focused on finding the pathway involved in the degradation of the pesticide.

The Pathway:

The gluconeogenesis pathway-where glucose is generated from non-carbohydrate sources [Highly expressed]. In mice, blood sugar level is administrated by sodium acetate orally and through rectal route. With this, the rectal route led to more blood sugar increase than the oral route.

Reconfirmed in Humans:

"The role of gut bacteria in mediating pesticide induced diabetes was confirmed in humans by studying the faeces of diabetes"[Velmurugan et al., 2017).

Serum Urea, Uric Acid, Creatinine Levels in Diabetic Mellitus Patients:

This research evaluated and compared the results of the level of serum uric acid, urea, and creatinine levels in diabetic and non-diabetic mellitus patients. It was observed that there was a significant increase in the level of urea and creatinine in diabetic patients.

The serum uric acid concentration was higher in the diabetics and there was no significant difference between them compared to the control. The increase in the serum urea, uric acid and creatinine level may be implication of renal insufficiency which can cause increased creatinine and uric acid level as a result of reduction of blood flow to the kidneys. The level of serum urea and creatinine increased in diabetic subjects [Male and female]. The clinical diagnosis and estimation of serum urea and creatinine to help in the management of diabetes mellitus patients Richard et al. 2017].

Pesticide Level in Patients with Chronic Kidney Disease:

They detected nine organochlorine pesticides in the blood samples. The quantum of organochlorine pesticide present in the chronic kidney disease unknown etiology patients are higher as compared to that present in chronic kidney disease known etiology patients. It appears that chronic kidney disease patients tend to have higher blood level of pesticides as compared to normal healthy subjects. The finding of increased levels of organochlorine pesticides in chronic kidney disease patients suggest involvement of organochlorine pesticides with deranged renal function and of chronic kidney disease of unknown etiology.

Organochlorine pesticides are known inducer of oxidative stress. Oxidative stress is prevalent in chronic kidney disease patients and is considered to be an important pathogenic mechanism. With this, it is possible that accumulated pesticides may induce oxidative stress leading to development of chronic kidney disease. This study reveals the knowledge among urban population of Delhi who are not directly involved in agricultural activity or manufacture of pesticides and are probably exposed through environmental contamination. Prolonged cumulative exposure in conjugation with other contributing factors possibly lead to renal dysfunction and chronic kidney disease of unknown etiology [Ghosh et al., 2017).

2. Methodology

Farmers with pesticide handlers (10), and others with non-pesticide handlers (10) were taken for the comparative study. Studies suggests that pesticide exposed farmers have higher concentration of serum urea and serum creatinine when compared to non-pesticide exposed farmers [Yassin and shanty, 2016].

A questionnaire was administrated to pesticide exposed farmers and non-pesticide exposed farmers. The individuals with diabetes were not taken for the kidney function test. Because the level of serum urea and creatinine increased in individuals with diabetes. Serum area and creatinine concentrations have an important role in the management of diabetes mellitus Richard et al, 2017). Colorimetric determination of serum urea and creatinine level is estimated for kidney function test

Sample Collection:

The age should be considered between 30-50 years. Gender is not significant. The person with family history of diabetes and medical history of any other diseases should be excluded. The upper arm tied with tourniquet and vigorously cleaned with 75% alcohol and allowed to dry. Using a sterile syringe and needle, blood was drawn from suitable vein in the arm. The needle and the syringe were removed and collected blood sample transferred to a sterile red tube and red in refrigerator at a temperature of 20 degree Celsius.

Estimation of Urea by Dam-TSC Method:

Principal:

Urea directly reacts with diacetyl monoxime, in the presence

of thio-semi carbazide to form a pink colored complex which is measured at 540 nm [Yellow and green filters]. The intensity of pink colored complex formed gives quantitative estimation of urea present in the samples

Reagents:

1) Diacetyl monoxime (DAM):

1.56 g of Diacetyl monoxime is dissolved in 250 ml of distilled water

2) Thio-semi Carbaszide (TSC):

.041 g of thio-semi Carbaszide is dissolved in 250 ml of distilled water. Stored at brown bottle

3) Ferric chloride reagent:

0.324 gram of ferric chloride is dissolved in 10 ml of 85% orthophosphoric acid. Stored in a brown bottle.

4) 20% H_2So_4

5) Acid reagent:

1.0 ml of ferric chloride is mixed with 100 ml of distilled water.

6) Stock Standard solution:

Dissolved 0.1 gram of urea in 100 ml of distilled water.

7) Working Standard solution:

2 ml of stock standard solution was diluted to 100 ml with distilled water.

Procedure:

Mixed 1.8 ml of 10% of TCA with 0.2 ml of serum. After 10 minutes centrifuged and 0.5 ml of the supernatant was pipetted out into test tubes. Into the serious of test tubes, taken 0.5-2.5 ml of standard urea solution corresponding to the value 10-50 µg respectively. The volume was made upto 3 ml with distilled water in all test tubes. I ml of DAM, added 1 ml of TSC, 3 ml of acid reagent, mixed well and heated in a vigorously boiling water bath for 20 minutes. A blank was also conducted.

Remove the tubes and cooled. Reading was taken against a reagent blank at 540 nm in a colorimeter. A standard graph was drawn by plotting the concentration of urea on X axis and optical density along Y axis. From this, the amount of urea present in the serum is calculated.

Normal Value:

14-40 mg/dl [Pandey, 2012].

Clinical significance:

Elevated serum urea levels may be due to pre-renal, renal or post-renal etiology. Pre-renal causes could be cardiac related or due to increased protein catabolism and dehydration. Renal causes include glomerular nephritis, chronic nephritis, nephritic syndrome and other kidney disease. Post renal causes include obstruction of urinary tract.

Decreased serum urea levels could be due to pregnancy, low protein intake, severe liver diseases, inborn errors of urea cycle and SIADH [Syndrome of In-appropriate ADH secretion].

Estimation of creatinine in serum by Jaffe's method:

Principal:

To quantitative estimation of creatinine is based on jaffe's method in which creatinine reacts with alkaline picric acid to give orange red colored compound of creatinine picric acid.

Reagents:

a) Picric acid (0.04 mol/litre)

9.16 gram of picric acid crystals is dissolved in 100 ml of distilled water

b) Sodium hydroxide (0.75 N)

Three gram of sodium hydroxide in 100 ml of distilled water

c) Stock Standard solution

0.1 gram of creatinine is dissolved in 0.1 N of HCL and the volume is made up to 100 ml with distilled water

d) Working Standard solution

1 ml of stock standard solution is accurately pipetted out into 100 ml standard flask and made up with the distilled water

Procedure:

Tubes containing 0.5-2.5 ml of the working standard solution corresponding to the value 10-50 µg. The volume was made up to 3.0 ml of with water. For blank, added 3.0 ml of distilled water. 1.0 ml of serum was taken for the test, to all the test tubes, added 1 ml of picric acid solution and 1 ml of NaOH and allow to stand for developed 20 minutes. The color developed was read in a colorimetrically at 540 nm. Against the reagent blank.

A graph was drawn by plotting colorimetrically the reading is on the Y axis and concentration of creatinine on X axis.

Normal Value:

0.5-1.4 mg/dl[Pandey, 2012].

Clinical significance:

Elevated creatinine levels are associated with

(1) Renal failure

(2) Obstruction of urinary tract

(3) Chronic nephritis

(4) Muscular disease like gigantism and acromegaly.

Lower levels are associated with,

(1) Muscular dystrophy.

3. Result and Discussion

The Farmers (n=20) were joined in this comparative study, which is divided into 2 groups, pesticide handlers (n=10) and non-pesticide handlers (n=10). The following parameters are Gender, age, BMI, Years of exposure, Serum urea and creatinine for both pesticide and non-pesticide handlers were tabulated in table 1 and table 2.

Table 1

Pesticide handlers

PESTICIDE HANDLERS										
Parameters	PH-1	PH-2	PH-3	PH-4	PH-5	PH-6	PH-7	PH-8	PH-9	PH-10
Gender	Male	Male	Male	Male	Male	Female	Male	Male	Male	Male
Age	49	47	36	48	50	43	42	50	46	35
BMI	23.1	28	22.3	19.9	25.3	24.8	20.6	22.9	23.5	25
Years of exposure	18	15	12	20	22	13	15	22	14	13
Urea mg/dl	27	35	32	31	27	26	29	32	36	30
Creatinine mg/dl	1.2	1.4	0.9	1	1.3	0.9	1	1.1	1.2	1.3

Table 2
Non-Pesticide handlers

NON PESTICIDE HANDLERS										
Parameters	NPH -1	NPH-2	NPH-3	NPH -4	NPH -5	NPH -6	NPH-7	NPH-8	NPH-9	NPH -10
Gender	Male	Male	Male	Male	Male	Male	Female	Female	Male	Male
Age	46	33	33	45	42	35	43	35	45	44
BMI	23.5	18.8	18.9	24.2	24	20.5	22.4	26.6	23.1	19.9
Urea mg/dl	20	21	19	25	17	22	24	21	18	17
Creatinine in mg/dl	0.6	o.7	0.5	0.7	0.5	0.6	0.5	0.5	0.8	0.7

Table 1 and 2 shows the data for pesticide handlers and non-pesticide handlers. Pesticide handlers having increased level of serum urea and creatinine concentrations when compared to non-pesticide handlers. With this exposure level in pesticides increases the concentration of serum urea and creatinine.

Mean ± standard deviation for parameters with age, BMI, urea, creatinine for both pesticide handlers and non-pesticide handlers were tabulated in Table 3.

Table 3

Parameters	Pesticide handlers [mean ± standard deviation]	Non pesticide handlers [mean ± standard deviation]
Age	44.6 ± 5.5015	40.1 ± 5.1273
BMI	23.54 ± 2.5326	22.19 ± 2.4447
Urea	30.5 ± 3..2015	20.4 ± 2.6153
Creatinine	1.13 ± 0.1676	0.61 ± 0.1044

Table 3, When compared to non-pesticide handlers there is an increase in mean and standard deviation for pesticide handlers.

The body stores many pesticides in fat before they are removed from the body by the liver or kidneys. Pesticides that are stored in fat can build up in larger quantities in the bodies of pesticide handlers [National pesticide information center, 2011]. The researchers found that diabetes and obesity are major contributions to End Stage Renal Disease, but not everyone with these conditions develop kidney failure. It is possible that exposure such as pesticides may account for some of that difference [Aymes, 2015].

Comparison of serum urea concentration in pesticide handlers and non-pesticide handlers were tabulated in table 4.

From Table 4, increased serum urea concentration is increased in pesticide handlers. Fig. 1 shows the graphical chart for serum urea in pesticide handlers and non-pesticide handlers, concentrations as mg/dl. Increased serum urea may be due protein catabolism and dehydration. Renal causes include glomerular nephritis, chronic nephritis, obstruction of urinary tract and other kidney disease. Decreased serum urea levels could be due to pregnancy and low protein intake, severe liver diseases, inborn errors of urea cycle. Urea is the major end product of protein catabolism in humans. The estimation of urea is done by DAM-TSC method, colorimetrically at 540nm [Pandey, 2012].

Table 4

Urea mg/dl in pesticide handler	Urea mg/dl in non pesticide handler
27	20
35	21
32	19
31	25
27	17
26	22
29	24
32	21
36	18
30	17

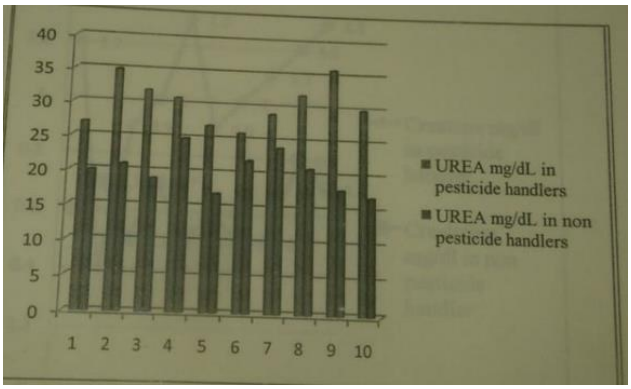
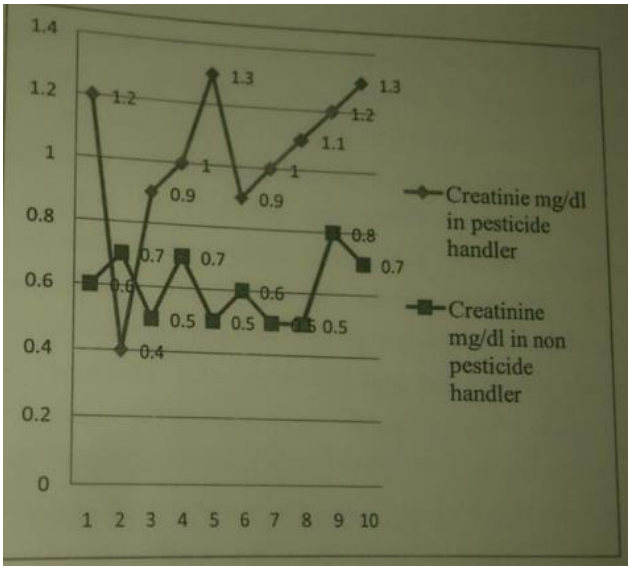


Fig. 1.

Comparison of serum creatinine concentration for pesticide handlers and non-pesticide handlers were tabulated in table 5.

Table 5

Creatinine mg/dl in pesticide handler	Creatinine mg/dl in non pesticide handler
1.2	0.6
0.4	0.7
0.9	0.5
1	0.7
1.3	0.5
0.9	0.6
1	0.5
1.1	0.5
1.2	0.8
1.3	0.7



From Table 5 also shows the increased serum creatinine concentrations in pesticide handlers. Fig. 2 shows the graphical line for serum creatinine in both pesticide handlers and non-pesticide handlers, concentrations as mg/dl. Increased serum creatinine concentrations are associated with renal failure, destruction of urinary tract, chronic nephritis, muscular disease like acromegaly. Decreased serum creatinine concentrations are seen in muscular dystrophy. The method of serum creatinine is estimated by jaffe's method colorimetrically at 540nm [Pandey, 2012].

The method of serum creatinine is estimated by jaffe's method colorimetrically at 540nm with this we can conclude that exposure of pesticides cause decreased in serum, concentrations.

4. Summary and Conclusion

Pesticide exposure has been linked to variety of health outcomes. Kidney disease is characterized by kidney damage and kidney dysfunction. Kidney disease are associated with reduced urea excretion and consequent rise in blood concentration. Increase of pesticide exposure cause decreased activity of acetylcholinestrease. Certainly, it alters the HbA1c concentrations. Acetylcholine is the main target of pesticides. Organophosphates is the one example for inhibiting the activity of acetylcholinestrease. Exposure to acetylcholinesterase inhibiting pesticides was estimated by measuring haemoglobin correlated red cell acetylcholinesterase levels using cholinesterase test [John et. al.., 2006]. Our study on, kidney faction test between pesticide handlers and non-pesticide handlers shows the increased prevalence in those who were working with pesticides. The participants were taken in annur village (n-20). Chronic kidney disease is the final stage of End Stage Renal Disease. Evidence shows that pesticide exposure may harm the kidney through oxidative stress and resulting cell damage (Abdollahi et al, 2004). Pesticide exposure cause the impairment of protein synthesis by hepatocytes and disturbance of kidney function. Elevation of serum urea and creatinine in blood due to pesticide exposure on the basis of renal insufficiency, impaired glomerular filtration, urinary tract obstruction and kidney disease [Yassin and shanti, 2016).

Chronic exposure of organophosphate led to diabetes. Organophosphate induced diabetes was mediated by gut bacteria. Gluconeogenesis pathway is involved in the gut microbial degradation of pesticides, where the glucose is highly expressed (Velmurugan et al, 2017]. The clinical diagnosis of serum urea, creatinine, uric acid to help in the management of diabetes mellitus patients [Richard et al. 2017]. Our study taken between pesticide handlers (n=10) and non-pesticide handlers (-10). Studies shows that pesticide exposed farmers have higher concentration of serum urea and creatinine when compared to non-pesticide exposed farmers (Yassin and shanti, 2016].

A questionnaire was administrated to pesticide handlers and non-pesticide handlers. The subjects with family history of diabetes and having medical history of other diseases were not part of the study. Serum creatinine is estimated by jaffe's method, colorimetrically at 540 nm and serum urea is estimated by DAM-TSC method, colorimetrically at 540 nm. Results shows that increased mean ± standard deviation for pesticide handlers with urea[30.5 +3.2015] and creatinine[1.13 ± 0.1676], mean ± standard deviation of pesticide handlers BMI also increased it is tabulated in table 3. The amount of urea and creatinine elevated level in serum is significantly increased in pesticide handlers is tabulated in table 1 and 2. The graph shows the comparison between pesticide handlers and non-pesticide handlers in fig. 1 and fig. 2. The curve shows that increased level of serum urea and creatinine concentrations when compared to non-pesticide handlers, the prevalence of kidney function is increased in those who are working in agricultural field [Pesticide handlers].

References

[1] Stevens L, Levey A. Frequently asked questions about GFR estimates. New York: National Kidney Foundation. 2004.
[2] Vupputuri S. Lifestyle risk factors and chronic kidney disease. Annals of epidemiology. 2003;13(10):712-20.
[3] Sandler DP, Smith JC, Weinberg CR, Buckalew VM, Jr., Dennis VW, Blythe WB, et al. Analgesic use and chronic renal disease. N Engl J Med. 1989;320(19):1238-43.
[4] , Bunini, et al., Self-reported Health Effects among Short and Long-term Pesticide Sprayers in Arusha, Northern Tanzania: A cross Sectional Study Occup Med Health Aff 2015,3:6.
[5] Velmurugan et al., Gut microbial degradation of organophosphate insecticides-induces glucose intolerance via gluconeogenesis, Genome Biology, 2017, 18:8.