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A Study on Evaluation of Spot Urine Microalbumin and Protein Creatinine Ratio in Newly Diagnosed Type 2 Diabetic Patients

Anu Singh[1], Dr. Savita Rathore[2]*

[1]Research Scholar Department of Biochemistry

ORCID ID: 0009-0008-6995-3922

2*Professor & Head Department of Biochemistry Malwanchal University

Email id:singhmaanu001@gmail.com

ABSTRACT

Background

Type 2 Diabetes Mellitus (T2DM) is a rapidly increasing global health problem associated with significant microvascular complications, particularly diabetic nephropathy. Early detection of renal involvement is essential to prevent progression to chronic kidney disease. Microalbuminuria and protein-creatinine ratio (PCR) in spot urine samples have emerged as simple and non-invasive biomarkers for early detection. However, their comparative diagnostic utility in newly diagnosed T2DM patients remains an area of ongoing research.

Methods

This case-control study was conducted in the Department of Biochemistry, Malwanchal University, Indore. A total of 636 participants were included, comprising 318 newly diagnosed T2DM patients (cases) and 318 non-diabetic individuals (controls). Clinical, anthropometric, biochemical, and lifestyle-related data were collected. Spot urine samples were analyzed for microalbuminuria and protein-creatinine ratio (PCR), while blood samples were evaluated for fasting blood sugar and HbA1c. Lipid profile and urinary parameters were also assessed. Statistical analysis was performed using appropriate tests, with $p < 0.05$ considered statistically significant.

Results

Diabetic patients demonstrated significantly higher fasting blood sugar (264.6 ± 81.58 mg/dL vs 158.6 ± 52.38 mg/dL) and HbA1c levels (10.2 ± 2.01% vs 8.42 ± 2.07%) compared to controls ($p < 0.001$). Lipid profile abnormalities, including elevated triglycerides, LDL, and VLDL, along with reduced HDL levels, were also statistically significant ($p < 0.001$). The protein-creatinine ratio was significantly higher in cases (2.46 ± 1.39) compared to controls (1.82 ± 0.99) ($t = 6.72$, $p < 0.001$), indicating early renal involvement. In contrast, microalbuminuria did not show a statistically significant difference between groups ($p = 0.07$). Urinary findings such as glucosuria, ketonuria, hematuria, and increased urinary sediments were more prevalent in diabetic patients. Strong correlations were observed between glycemic parameters and lipid profile in the case group.

Conclusion

The study concludes that protein-creatinine ratio (PCR) is a more sensitive and reliable marker than microalbuminuria for early detection of renal impairment in newly diagnosed T2DM patients. Spot urine analysis provides a simple, non-invasive, and cost-effective screening tool suitable for routine clinical practice. Early identification of renal dysfunction, along with comprehensive metabolic control, can significantly reduce the progression of diabetic nephropathy and improve patient outcomes.

Keywords: *Type 2 Diabetes Mellitus, Microalbuminuria, Protein-Creatinine Ratio, Diabetic Nephropathy, Renal Biomarkers, Glycemic Control*

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1. INTRODUCTION

Diabetes mellitus (DM) is one of the oldest known diseases, with origins tracing back nearly 3,000 years. It is a chronic metabolic disorder characterized by persistent hyperglycemia and has remained a major focus of medical research due to its widespread impact on human health [1].

The understanding of diabetes significantly evolved in 1936 with its classification into Type 1 (T1DM) and Type 2 diabetes mellitus (T2DM)[2]. T1DM results from autoimmune destruction of pancreatic β-cells leading to absolute insulin deficiency, whereas T2DM is primarily associated with insulin resistance and relative insulin deficiency [3]. Later, in 1988, T2DM was recognized as a key component of metabolic syndrome, along with hypertension, dyslipidemia, and central obesity[4].

T2DM is the most common form of diabetes, accounting for approximately 90–95% of cases worldwide[5]. It develops due to a complex interaction of genetic predisposition, environmental influences, and lifestyle factors such as obesity, sedentary habits, poor diet, smoking, and alcohol consumption[6]. If uncontrolled, it leads to severe complications including cardiovascular disease, neuropathy, nephropathy, and retinopathy [7].

Globally, diabetes has reached epidemic proportions. The number of affected individuals has risen dramatically, from 366 million in 2011 to a projected 552 million by 2030, with nearly 80% of cases occurring in low- and middle-income countries [8]. India, in particular, represents a major burden, with approximately 77 million diabetics in 2019, expected to exceed 100 million by 2030 [9].

The pathophysiology of T2DM involves insulin resistance, β-cell dysfunction, and impaired glucose metabolism, along with abnormalities in incretin hormones such as GLP-1 and GIP. Additional mechanisms include mitochondrial dysfunction, adipokine imbalance, and increased fatty acid metabolism, which contribute to hyperglycemia and systemic complications. These metabolic disturbances also adversely affect cardiac function, leading to diabetic cardiomyopathy and reduced cardiac efficiency [10].

Diagnosis of diabetes is based on established criteria including fasting plasma glucose, oral glucose tolerance test (OGTT), and HbA1c levels, as recommended by ADA and WHO guidelines [11]. Early detection is critical, as many patients already exhibit complications at diagnosis.

Recent research highlights the role of skeletal muscle mass and serum creatinine as important indicators of insulin resistance and diabetes risk. Lower muscle mass and reduced serum creatinine levels are associated with increased risk of developing T2DM, making them potential cost-effective biomarkers for early screening [12].

2. NEED FOR THE STUDY

Diabetes Mellitus, particularly Type 2 Diabetes Mellitus (T2DM), has emerged as a global public health challenge with alarming rates of incidence and prevalence. The increasing burden of T2DM is especially significant in low- and middle-income countries, where limited healthcare infrastructure, delayed diagnosis, and inadequate awareness contribute to poorer health outcomes. While national-level data provide an overview of the diabetes epidemic in India, region-specific data are crucial to understand the localized dynamics of this chronic condition and to design effective, context-sensitive prevention and management strategies.

In the Indian context, the International Diabetes Federation (IDF) estimates that India had approximately 77 million individuals living with diabetes in 2019, with projections exceeding 100 million by 2030. Despite such high national figures, there remains a scarcity of regionally disaggregated data, particularly in states like Madhya Pradesh, which is characterized by a mix of urban, semi-urban, and rural populations, varied socioeconomic backgrounds, and disparities in healthcare access.

Epidemiological studies specific to Madhya Pradesh have reported concerning trends. According to the ICMR-INDIAB Phase I Study, the prevalence of diabetes in urban Madhya Pradesh was estimated to be around 7.5%, while rural areas showed a prevalence of approximately 2.3%. [24] More recent local surveys conducted in urban Bhopal and Indore have suggested rising trends in both obesity and T2DM, particularly among middle-aged adults and individuals with sedentary lifestyles. However, consistent statewide surveillance and updated population- based studies are still lacking.

Given the rapidly changing lifestyle patterns, increasing urbanization, rising obesity rates, and persistent lack of awareness in both urban and rural Madhya Pradesh, it becomes imperative to assess the current status of diabetes prevalence, associated risk factors, and biomarkers such as serum creatinine especially considering emerging evidence of its inverse relationship with T2DM risk.

Justification for the Study:

1. **Data Gap in Madhya Pradesh:** Despite its growing population and diverse socio-demographic profile, there is a lack of updated, large-scale epidemiological data on T2DM and its risk factors in Madhya Pradesh.
2. **Public Health Relevance:** Understanding regional prevalence patterns was aid in planning effective screening programs and healthcare resource allocation.
3. **Emerging Biomarkers:** As literature increasingly supports the role of serum creatinine as a surrogate for muscle mass and predictor for T2DM, this study may help validate its utility in early identification of at-risk individuals in a cost- effective manner.
4. **Targeted Interventions:** Identifying high-risk populations based on lifestyle, biochemical parameters, and demographic factors was facilitate personalized prevention and management approaches, especially in semi-urban and rural areas where access to diagnostics is limited.
5. **Policy Implications:** The findings can inform local and state-level health policy frameworks, including integration into NPCDCS (National Programme for Prevention and Control of Cancer, Diabetes, Cardiovascular Diseases & Stroke) and other public health initiatives.

3. REVIEWED LITERATURE

The reviewed studies collectively demonstrate that type 2 diabetes mellitus (T2DM) is strongly associated with renal dysfunction, microalbuminuria, proteinuria, dyslipidemia, and poor glycemic control. A major recurring finding is that microalbuminuria and albumin/protein-creatinine ratios serve as early and reliable indicators of diabetic kidney involvement. Studies by Bakris et al.,[13] Kara et al.,[14] Warjuka et al.,[15] Tamrakar et al.,[16] and McGill et al.[17] emphasize that even when estimated glomerular filtration rate (eGFR) is preserved, the presence of microalbuminuria indicates a higher risk of chronic kidney disease progression and cardiovascular morbidity. These findings strongly support the importance of early renal screening in diabetic patients.

Another significant theme observed across the literature is the clinical utility of spot urine protein-creatinine ratio (PCR) as a practical substitute for 24-hour urinary protein estimation. Studies by Medina-Rosa et al.,[18] Karthikeyan et al.,[19] Kaminska et al.,[20] Sumida et al.,[21] Alam et al.,[22] and Chauhan et al.[23] consistently report good sensitivity, specificity, and strong correlation between spot PCR and standard 24-hour proteinuria measurements. These findings suggest that PCR is a simple, outpatient-friendly, and cost-effective tool for routine assessment of renal impairment and proteinuria in diabetic patients.

The literature also demonstrates a consistent association between poor glycemic control and abnormal lipid profile. Studies by Bhambhani et al.,[24] Baranwal et al.,[25] Athyros et al.,[26] Sudhakar et al.,[27] Gordon et al.,[28] and Cai et al.[29] show that diabetic patients frequently exhibit elevated total cholesterol, triglycerides, LDL levels, and reduced HDL levels. These abnormalities are more pronounced in T2DM and in patients with complications such as nephropathy. HbA1c has been repeatedly shown to correlate with lipid disturbances, indicating that glycemic and lipid control should be monitored concurrently in diabetes management.

Several interventional and review studies further suggest that adjunctive therapies may improve metabolic parameters. Panahi et al.[30] demonstrated that curcuminoids improved selected lipid fractions, while Shabani et al.[31] reported that garlic supplementation reduced major lipid and glycemic markers. Ko et al.[32] showed that combined DPP-4 and SGLT2 inhibition was superior to monotherapy in improving both glycemic status and renal outcomes in an experimental diabetes model. These findings indicate that combination and supportive therapies may provide added benefits in diabetes management, particularly in patients at risk of renal complications.

Some studies also expand the understanding of diabetes risk and progression through muscle mass and serum creatinine-related mechanisms. Evidence suggests that reduced muscle mass and lower serum creatinine levels may reflect altered metabolic status, although these findings are less directly linked compared to renal and lipid parameters. Nevertheless, they support the concept that comprehensive biochemical profiling beyond glucose measurement alone may aid in earlier detection of diabetes risk.

4. AIM AND OBJECTIVE

Aim:

To evaluate the diagnostic utility of spot urine microalbumin and protein creatinine ratio in detecting early renal impairment in newly diagnosed Type 2 diabetic patients, thereby facilitating timely management of diabetic nephropathy.

Objectives:

1. To determine the prevalence of microalbuminuria in newly diagnosed Type 2 diabetic patients using the spot urine microalbumin test.
2. To assess the protein creatinine ratio in spot urine samples of newly diagnosed Type 2 diabetic patients and its relationship with early kidney damage.
3. To establish the correlation between microalbuminuria, protein creatinine ratio, and other risk factors like duration of diabetes, blood pressure, and glycemic control (HbA1c levels).
4. To evaluate the efficacy of the spot urine test as a simple, non- invasive tool for early detection of diabetic nephropathy.
5. To compare the sensitivity and specificity of microalbumin and protein creatinine ratio in predicting kidney dysfunction in Type 2 diabetic patients.
6. To recommend guidelines for routine screening and early intervention based on the findings, aiming to reduce the progression of kidney disease in diabetic patients.

Hypothesis:

In individuals recently diagnosed with Type 2 diabetes, the analysis of spot urine microalbumin and protein creatinine ratio has the potential to serve as an early and non-invasive indicator of kidney impairment. This concept holds promise for facilitating prompt intervention to impede the progression of diabetic nephropathy.

This hypothesis suggests that identifying abnormal levels of microalbumin and protein-creatinine ratio in the urine at an early stage can be linked to the development of kidney damage in individuals with Type 2 diabetes. This emphasizes the necessity for additional research and confirmation of this approach, which could have a substantial impact on the treatment of diabetes- related complications.

Alternative Hypothesis (H_1):

There is a significant difference in the levels of spot urine microalbumin and protein creatinine ratio between newly diagnosed type 2 diabetic patients and non- diabetic individuals, indicating that these markers can effectively detect early renal impairment in diabetic patients.

Null Hypothesis (H_0): There is no significant difference in the levels of spot urine microalbumin and protein creatinine ratio between newly diagnosed type 2 diabetic patients and non-diabetic individuals. This implies that any observed differences in the study would be due to chance rather than a true difference in the evaluated metrics between the groups.

Purpose of this study

The purpose of this study is to assess the diagnostic value of spot urine microalbumin and protein-creatinine ratio in newly diagnosed type 2 diabetic patients. By evaluating these markers, the study seeks to:

1. Identify early renal impairment in diabetic patients before the onset of overt kidney disease.
2. Provide a simple, non-invasive method for early detection of diabetic nephropathy.
3. Determine if these urine tests can serve as predictors for future complications, including renal and cardiovascular disease.
4. Aid in early clinical decision-making to prevent the progression of kidney damage through timely intervention and management.

5. MATERIALS AND METHODS

Study Design

This study was be conducted as a case-control investigation to assess diabetic nephropathy in newly diagnosed patients with type 2 diabetes mellitus and to evaluate its association with selected clinical, biochemical, and lifestyle-related risk factors.

Study Area

The study was be carried out in the Department of Biochemistry, Malwanchal University, Indore, in collaboration with the outpatient department (OPD) of the index hospital catering to patients from Indore city and nearby areas.

Study Population and Sample Size

A total of 318 subjects was be included in the study. The sample size has been determined on the basis of results from a previously published study and statistical considerations relevant to the objectives of the present investigation.

The study population was include patients diagnosed with type 2 diabetes mellitus (T2DM) and suitable controls, as per the case-control design.

Selection of Participants

Participants was be recruited from the OPD of the index hospital and nearby healthcare facilities in Indore. Eligible individuals was be screened according to the predefined inclusion and exclusion criteria.

Inclusion Criteria

1. Patients with newly diagnosed type 2 diabetes mellitus, diagnosed within the last six months at the time of enrollment.
2. Patients wasing to participate and provide written informed consent.
3. Age- and sex-eligible participants fulfilling the study requirements.

Exclusion Criteria

1. Patients unwasing to participate in the study.
2. Patients with uncontrolled hypertension.
3. Patients with poor glycemic control.
4. Patients with urinary tract infection.
5. Patients with cardiac failure.
6. Patients suspected to have non-diabetic nephropathy, such as those with ultrasonography of kidney, ureter, and bladder (USG KUB) showing contracted kidney, cystic renal disease, or other structural renal abnormalities.
7. Patients with significant comorbid medical illnesses that may influence renal function.
8. Patients with known degenerative renal diseases unrelated to diabetes.

Study Variables

Diabetic nephropathy was be assessed in relation to various risk factors, including:

- Hypertriglyceridemia
- Dyslipidemia
- Smoking
- Body mass index (BMI)
- Hypertension
- Family history of diabetes
- History of renal disorders

Data Collection Method

Data collection was focus on clinical, laboratory, demographic, and lifestyle-related variables for all study participants.

A. Clinical Data

1. Patient History

Detailed history was be obtained from each participant, including:

- Date of diagnosis of diabetes mellitus for diabetic participants
- Past medical history, especially history suggestive of pre-existing kidney disease
- Drug history, particularly medications that may affect kidney function or urinary protein excretion

2. Anthropometric Measurements

The following anthropometric and physiological parameters was be recorded:

- Age (in completed years)
- Sex
- Weight (in kilograms)
- Height (in centimeters)
- Body mass index (BMI), calculated as weight in kilograms divided by height in meters squared
- Blood pressure readings

B. Laboratory Data

1. Urine Sample Collection

A spot urine sample was be collected from each participant in a sterile container under aseptic precautions. Participants was be instructed to provide a midstream urine sample to minimize contamination.

2. Urine Testing Parameters

a. Microalbuminuria Measurement

- Urinary microalbumin was be measured using an appropriate immunoassay method or enzyme-linked immunosorbent assay (ELISA).
- The result was be expressed in mg/L or as per standard laboratory reporting format.

b. Protein-Creatinine Ratio (PCR)

- Urinary total protein and urinary creatinine was be measured in the collected sample.
- The protein-creatinine ratio (PCR) was be calculated by dividing urine protein concentration by urine creatinine concentration.
- The PCR was be expressed in mg/g or mg/mmol, depending on the reporting standard used.

Reference Values

- Normal urine microalbumin: <30 mg/g
- Normal protein-creatinine ratio: <150 mg/g

3. Blood Sample Collection

For diabetic participants, blood samples was be collected for:

- Fasting blood glucose
- HbA1c for assessment of long-term glycemic control

C. Demographic and Lifestyle Data

The following additional information was be collected:

- Smoking history (smoker/non-smoker)
- Alcohol consumption (yes/no)
- Physical activity level (sedentary/moderate/active)
- Family history of diabetes
- History of hypertension and renal disorders

Data Collection Procedure

1. Informed Consent

All participants was be informed about the purpose of the study, procedures involved, and possible risks and benefits. Written informed consent was be obtained prior to enrollment.

2. Recruitment

- Participants was be recruited from local hospitals, diabetes clinics, and general practice centers.
- In case-control comparison, control subjects was be selected from non-diabetic individuals attending health facilities or from the community, after ensuring they meet eligibility criteria.

3. Screening Visit

During the initial visit:

- Participants was be screened according to the inclusion and exclusion criteria.
- Eligible individuals was be enrolled in the study.
- Clinical and demographic details was be recorded.

4. Data Collection Visit

During the data collection visit:

- Anthropometric measurements including weight, height, and blood pressure was be recorded.
- Spot urine samples was be collected from all participants.
- Blood samples was be collected from diabetic participants.
- Information related to medical history and lifestyle habits was be documented.

5. Transportation and Storage of Samples

- Urine samples was be stored under appropriate refrigerated conditions immediately after collection.
- Samples was be transported to the laboratory preferably within 2 hours of collection to maintain sample integrity.
- Blood samples was also be processed according to standard laboratory protocols.

6. Data Recording and Storage

- All clinical, laboratory, demographic, and lifestyle-related data was be entered into standardized case record forms.
- The collected information was subsequently be transferred into a secure electronic database.
- Each participant was be assigned a unique identification number to ensure confidentiality.

Quality Control

To maintain the accuracy and reliability of the study data, the following quality control measures was be implemented:

1. **Training of Personnel**

Research assistants and laboratory staff was be trained in standardized procedures for participant interviewing, sample collection, anthropometric measurement, and data recording.

2. **Calibration of Equipment**

All equipment, including weighing scales, stadiometers, sphygmomanometers, and laboratory instruments, was be calibrated regularly.

3. **Repeat Testing**

A subset of samples may be re-analyzed to assess reproducibility and reliability of laboratory findings.

4. **Standardized Protocols**

Uniform protocols was be followed for collection, storage, transport, and analysis of samples.

Ethical Considerations

Confidentiality

Participant confidentiality was be maintained throughout the study. Personal identifiers was be removed, and all data was be coded using unique participant IDs.

Ethical Approval

Prior approval for the study was be obtained from the Institutional Ethics Committee/Institutional Review Board of Malwanchal University, Indore, before commencement of data collection.

Informed Consent

Written informed consent was be obtained from all participants after explaining the objectives, procedures, potential risks, and benefits of the study in understandable language.

Timeline

The study is expected to be completed over a period of six months, distributed as follows:

- **Months 1–2:** Participant recruitment and screening
- **Months 3–4:** Sample collection and data recording
- **Months 5–6:** Laboratory analysis, data cleaning, and preliminary statistical analysis

Data Entry and Management

- All data was be double-entered into a secure, password-protected electronic database to minimize data entry errors.
- Missing, incomplete, or inconsistent entries was be identified and verified through source documents wherever possible.
- Final cleaned data was be used for statistical analysis.

6. RESULTS

In our present study we take 636 Subjects in which 318 taken in control group and 318 for cases group as given in table number 1.

Table number 1 showing the number of control and cases group in subjects.

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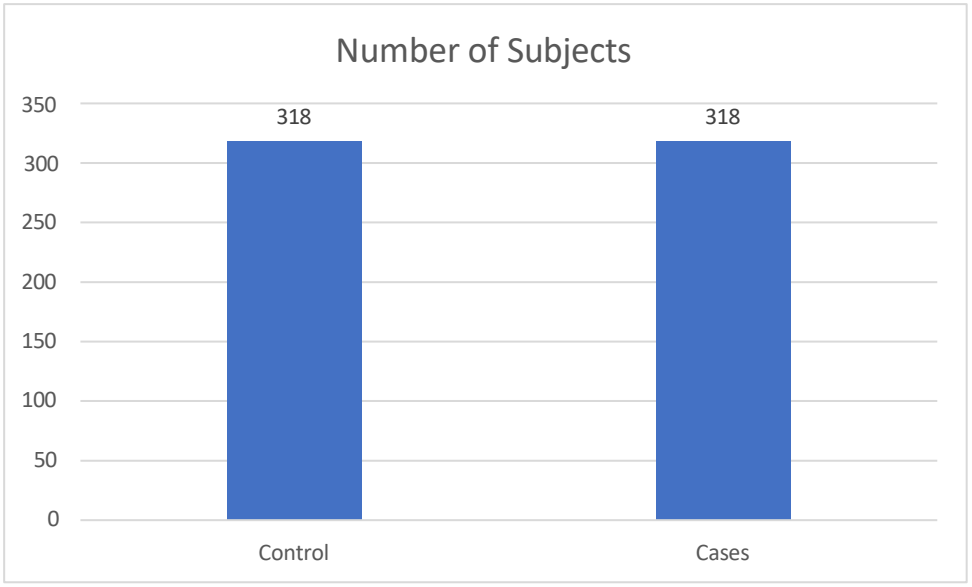
S. No	Number of Subjects
Control	318
Cases	318

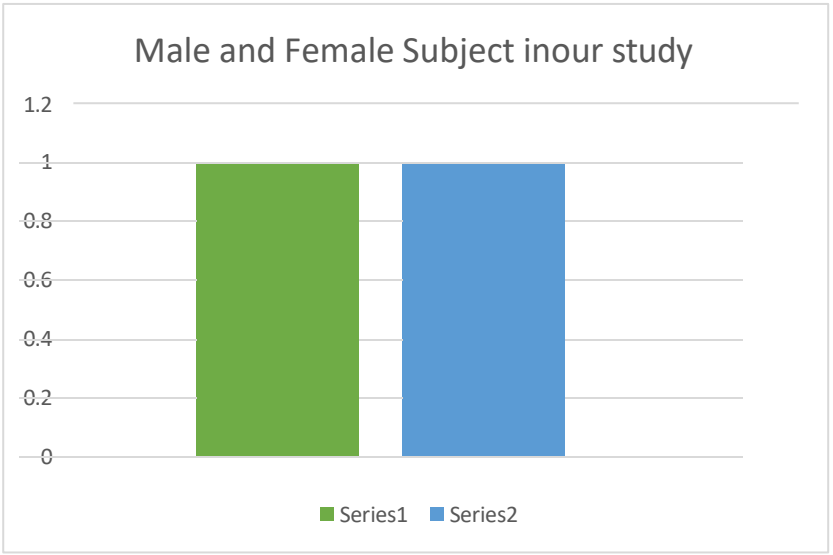


Bar chart number 1 showing the number of subjects in our study.

In our study we taken 160 male and 158 female in control group and 153 male and 165 female in cases group as show in in table number 2 given below.

Table number 2 showing male in female subjects in our study.

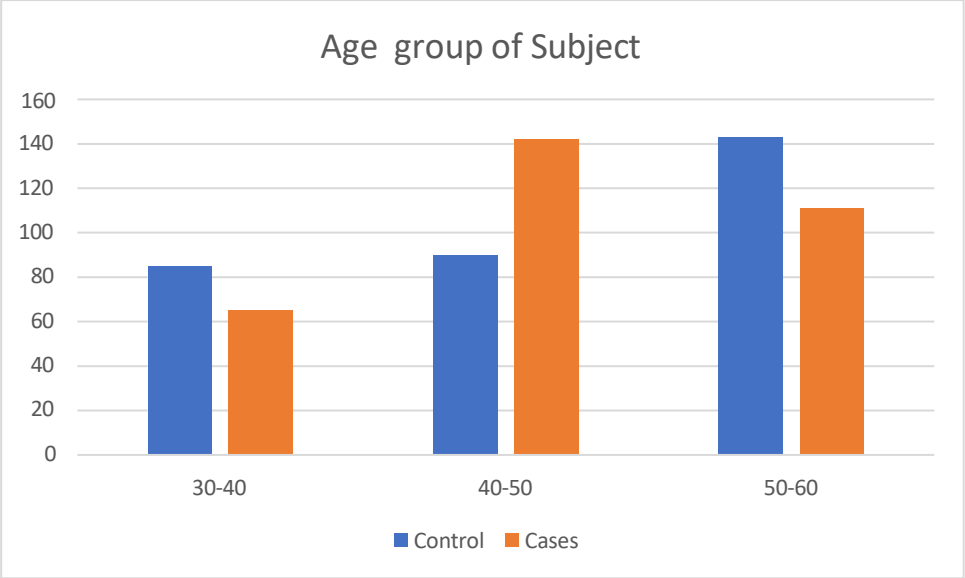
S. No	Subjects	Male	Female
1	Control	160	158
2	Cases	153	165



Bar chart number 2 showing the number of Number of Male and female in our Study.

In our study we observed 85 control subject and 65 in cases group which age belong between 30-40 year, 90 subjects in control and 142 in cases group belong 40-50 year 143 in control and 111 belonging to 50-60 year table number 3.

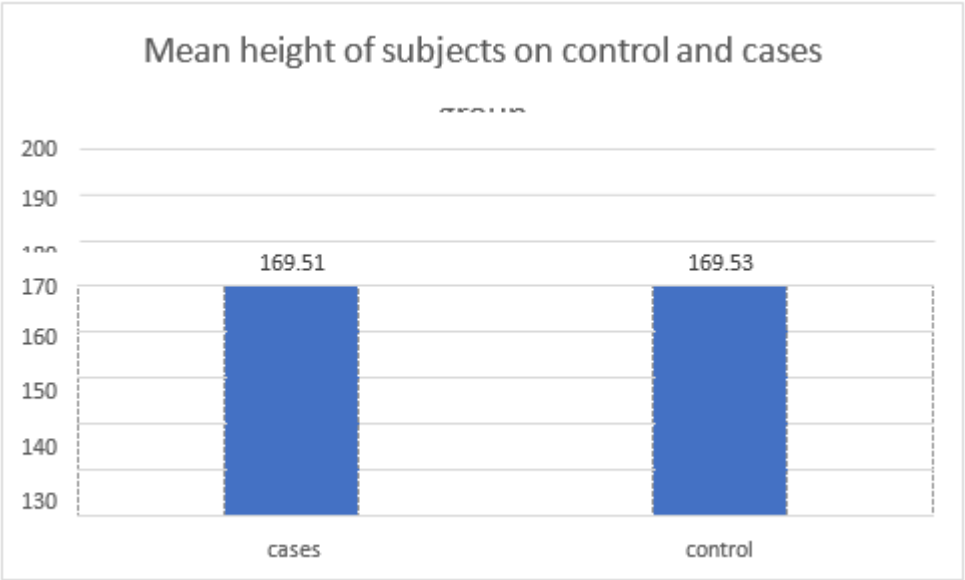
Subjects	30-40	40-50	50-60
Control	85	90	143
Cases	65	142	111



Bar chart number 3 showing the number of Number of subjects in control and cases group.

In our study we observed the mean height of control group having 169.51 cm and 169.53 cm in cases group having P Value 0.98 and T value of -0.03 as given in table number 4

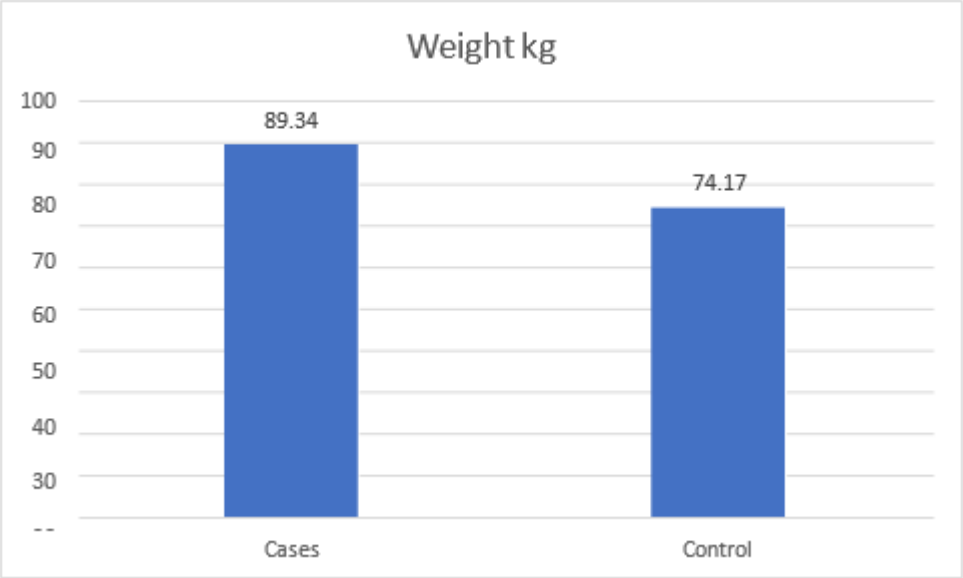
Height cm	Mean	Standard Deviation	Standard Error	T-Value	P-Value
Cases	169.51	11.13	0.62	-0.03	0.98
Control	169.53	11.41	0.64		



Bar chart Number 4 showing the, mean height of subjects on control and cases group

In our study we observed that the mean wight of control group having 74.17 kg and in cases group it was 89.34 kg as given in table number 5 .

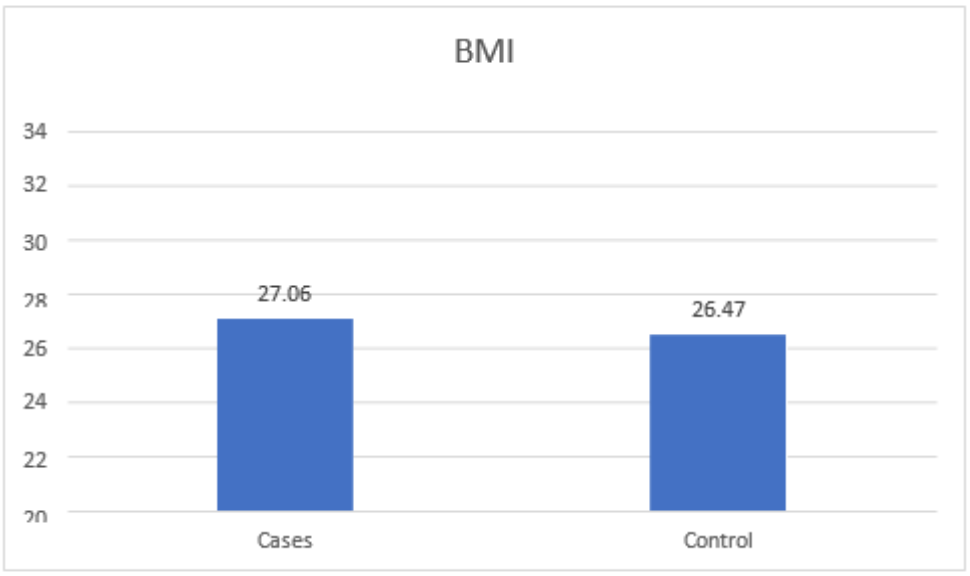
Weight kg	Mean	Standard Deviation	Standard Error	T-Value	P-Value
Cases	89.34	17.98	1.01		
Control	74.17	14.58	0.82	11.69	0.00



Bar chart Number 5 showing the, mean weight of subjects on control and cases group

In our study we observed that the mean BMI of control group having 27.06 and in cases group it was 26.47 as given in table number 6.

BMI	Mean	Standard Deviation	Standard Error	T-Value	P-Value
Cases	27.06	4.78	0.27	-1.42	0.16
Control	26.47	5.66	0.32		

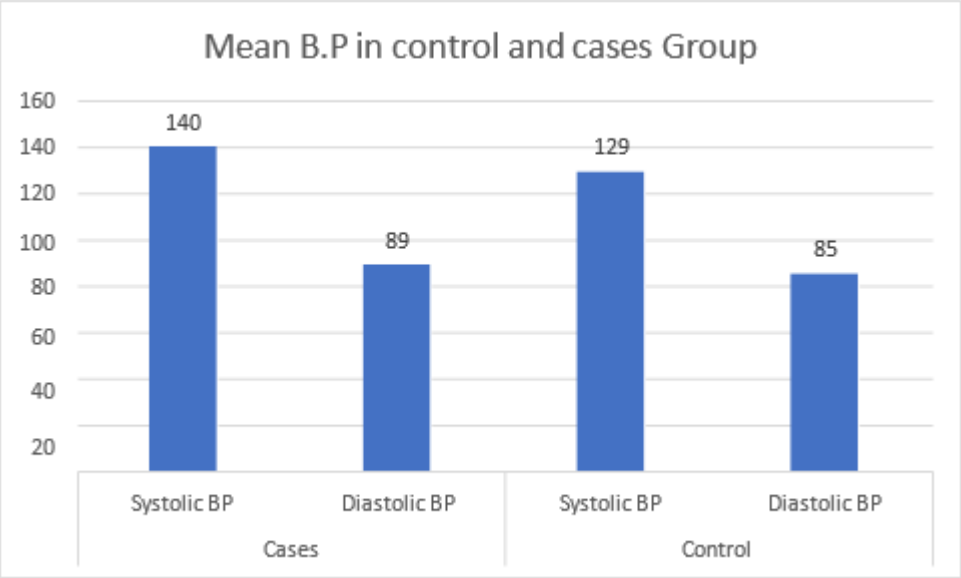


Bar chart Number 6 showing the, mean BMI of subjects on control and cases group

In our Present study we observed that the Systolic BP was 140 mmHG Diastolic BP was 89 mmHG and in case group 129 mmHG and 85 Diastolic BP given in table number 7.

Table number 7 showing the BP in control and cases group

	Cases		Control	
	Systolic BP	**Diastolic BP**	**Systolic BP**	**Diastolic BP**
Mean	140	89	129	85
Standard Deviation	11.1	8.61	11.49	8.95
Standard Error	0.62	0.48	0.61	0.5
T-Value	12.6		5.7	
P-Value	0		0	

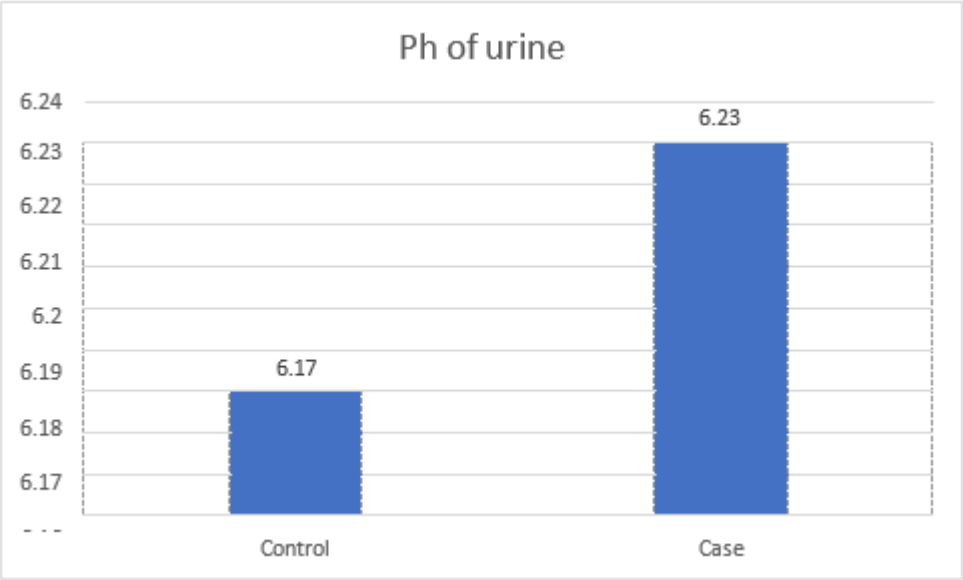


Bar chart Number 7 showing the, mean BP of subjects on control and cases group

In our observation we found the mean Ph of urine was 6.17 in control and 6.23 in cases statical calculation given in table number 8 given below.

Table number 8 showing the Mean Ph of urine with there statical value

	Mean	**Standard deviation**	**Standard Error**	**T value**	**P Value**
Control	6.17	0.99	0.06	0.81	0.42
Case	6.23	1.03	0.06		

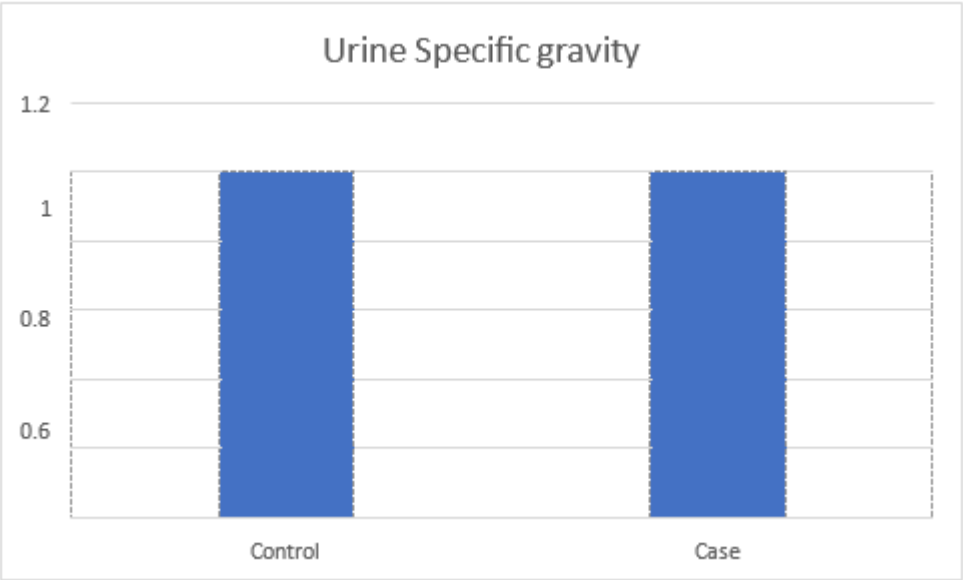


Bar chart Number 8 showing the mean **difference** of Urine Ph in both control and cases

In our finding we observed the mean specific gravity of urine was 1.02 in control and cases also it was 1.02 the statical calculation given below in table number 9.

Table number 9 showing the statical calculation of urine specific gravity

	Mean	**Standard deviation**	**Standard Error**	**T value**	**P Value**
Control	1.02	0.01	0	1.24	0.22
Case	1.02	0.01	0		

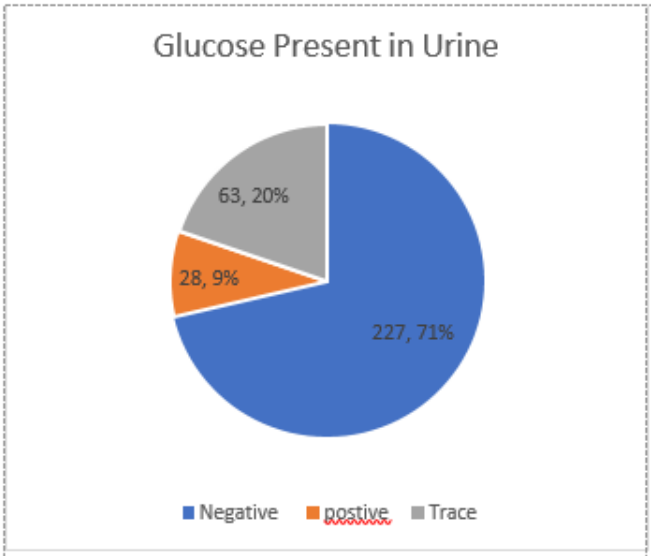


Bar chart 9 Showing the mean difference of specific gravity of urine in control and cases.

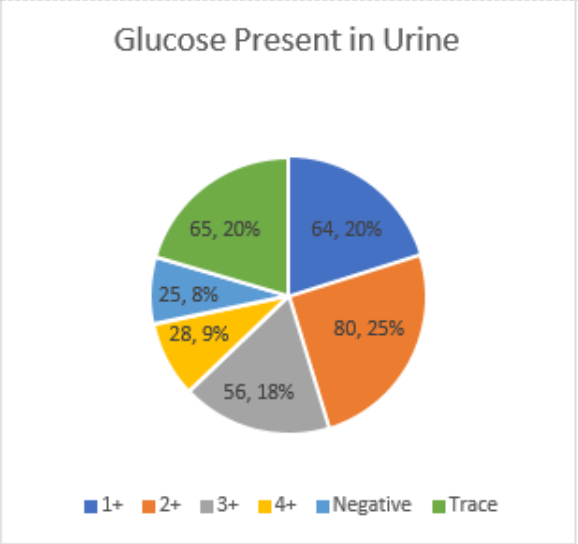
In our finding we observed that the presence of glucose in our subjects was given as follow 277, 28 and 63 found as negative, positive and trace amount as in cpntrol group where as in cases group it was 64,80,56,28,25 and 65 subjects sample showing as 1+,2+3+,4+ Negative and trace amount found in control group as show in table number 10.

Table number 10 showing present of glucose in control and cases group.

S. No	Control	Number of Subject	Percentage	S. No	Cases group	Number of Subjects	percentage
1	Negative	227	71	1	1+	64	20
2	positive	28	9	2	2+	80	25
3	Trace	63	20	3	3+	56	18
				4	4+	28	9
				5	Negative	25	8
				6	Trace	65	20



Pie Chart Number 1 Showing the Presence of glucose in urine in our control group



Pie Chart Number 2 Showing the Presence of glucose in urine in our cases group

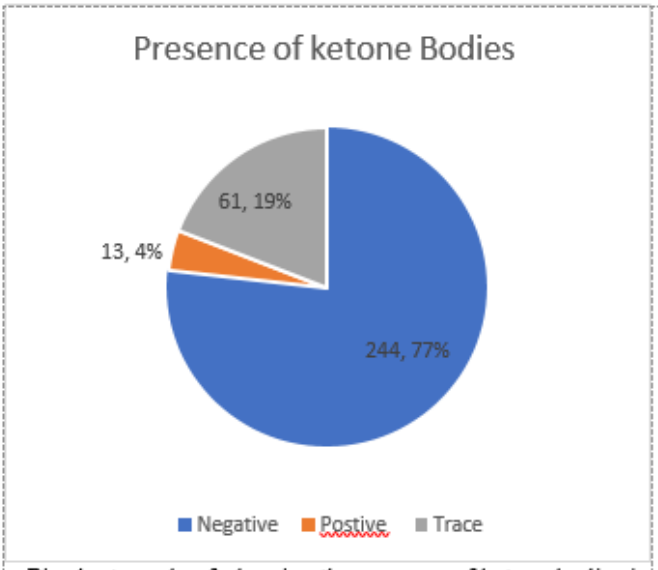
In our present study we observed that the presence of ketone bodies in our study was followed like positive in 61 trace amount in 13 and rest subjective 244

having negative where as on cases group it was 56,16,18,175,53 as 1+,2+,3+ negative and trace amount as followed in given in table number 11 given below.

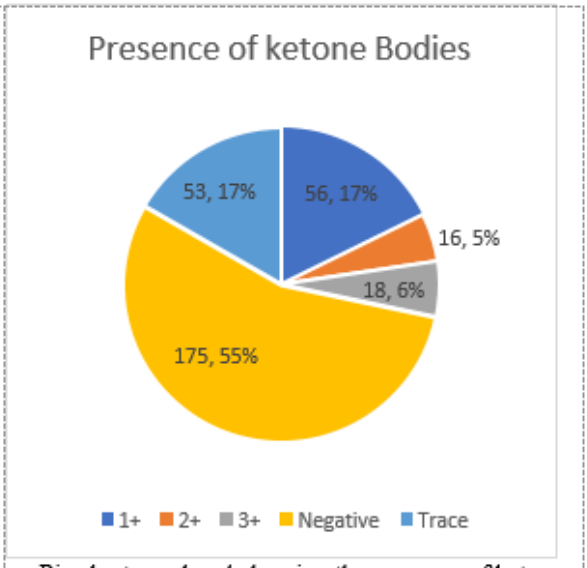
Table number of 11showing present of ketone bodies in control and cases group

S. No	Parameter	Number of subject in control	percentage	S. No	Parameter	Number of subject in control	percentage
1	Positive	61	19	1	1+	56	17
2	Trace	13	4	2	2+	16	5
3	Negative	244	77	3	3+	18	6
				4	Negative	175	54
				5	Trace	53	16

Pie chart number 3 showing the presence of ketone bodies in our control group.



Pie chart number 3 showing the presence of ketone bodies in our control group.

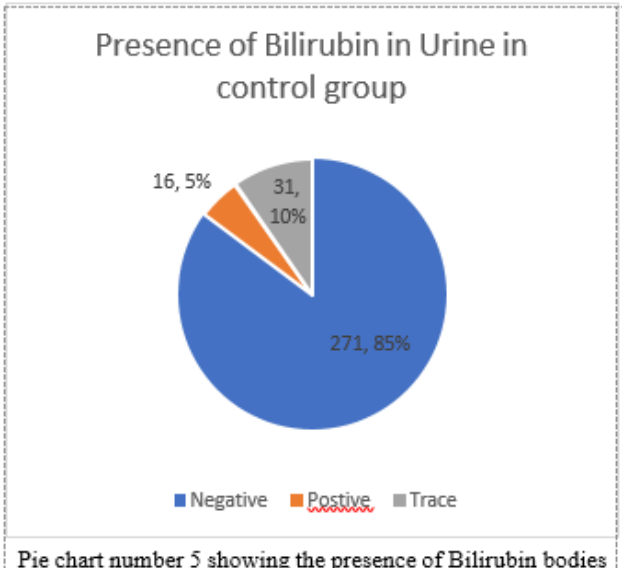


Pie chart number 4 showing the presence of ketone bodies in our cases group.

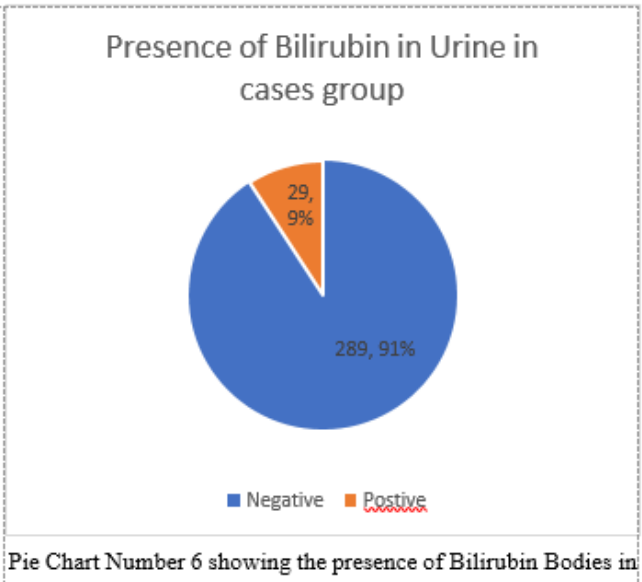
In our finding we observed that the presence Bilirubin of in control group was followed as Negative, Positive and trace amount as followed 271,16, and 31where as in control group it was as positive in 29 subjects and in rest 289 it was negative as given in table number 12 below

Table number 12 showing the presence of bilirubin in our control and cases group

S.N	Amount of Bilirubin Bodies	Number of Subjects	Percentage	S.No	Amount of Bilirubin Bodies	Number of Subjects	Percentage	Chi-Square	P-Value	Degree of Freedom
1	Negative	271	85	1	Negative	289	91	2.10	0.35	2
2	Positive	16	5	2	Positive	29	9			
3	Trace	31	31							



Pie chart number 5 showing the presence of Bilirubin bodies in our control group

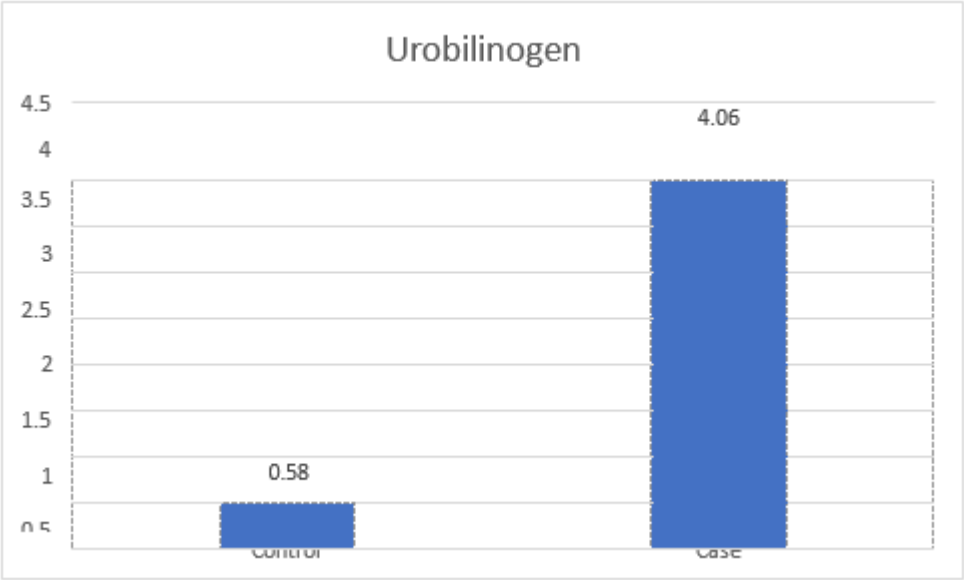


Pie Chart Number 6 showing the presence of Bilirubin Bodies in cases group

In our present finding we observed that the mean value of urobilinogen value in urine as followed 0.58 in control group and 4.06 in cases group completed static calculation showing in table number 13 given below

Table number 13 showing presence of urobilinogen static calculation

	Mean	Standard deviation	Standard Error	T value	P Value
Control	0.58	0.25	0.01	26.45	0
Case	4.06	2.33	0.13		

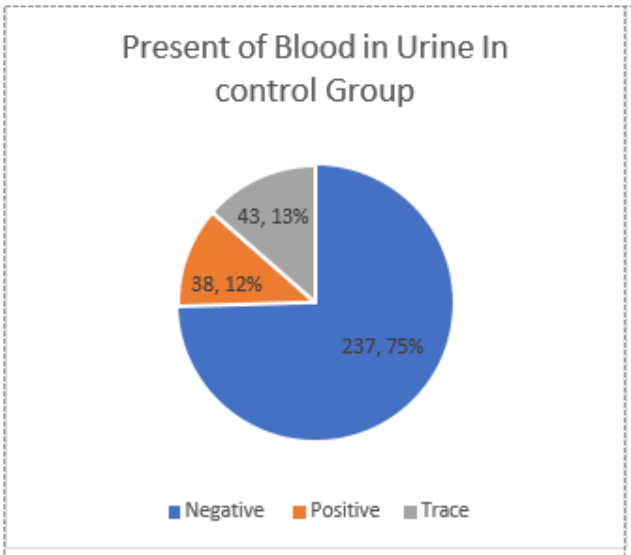


Bar Graph 10 showing the mean Urobilinogen value in control and cases group

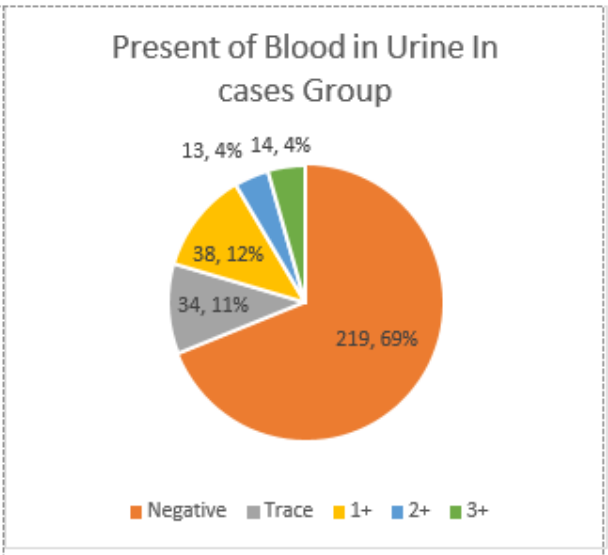
In our study we observed blood in urine as followed negative positive and trace amount as followed 237,38 and 43 in control group and in cases group it is followed as negative, trace,1+,2+ and 3+ followed as 219,34,38,13 and 14 as given below table number 14

Table number 14 showing the presence of blood in urine in control and cases group.

S. No	Amount of blood in urine	Number of Subjects in control group	percentage	S. No	Amount of blood in urine	Number of Subjects in cases group	percentage	Chi-Square	P-Value	Degree of Freedom
1	Negative	237	75	1	Negative	219	69	18.30	0.02	8
2	Positive	38	12	2	Trace	34	22			
2	Trace	43	13	2	1+	38	12			
				4	2+	13	4			
				5	3+	14	4			



Pie Chart Number 7 showing the presence of Blood in urine in Control group

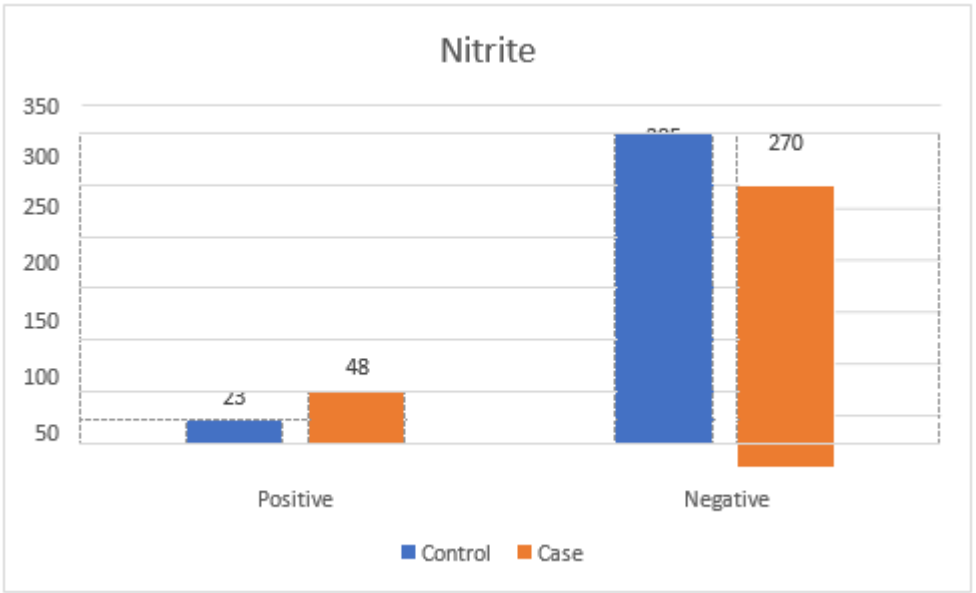


Pie Chart Number 8 showing the presence of Blood in urine in cases group

In our present study we observed that the present of Nitrite in urine as followed positive in 23 and negative in 295 in control region and in cases region it was 48 positive and 270 negative as given in table number 15 given below.

Table number 15 showing the Nitrite in urine sample in control and cases group.

S. No	Nitrite present is study	Control	Case	Chi-Square	p-value	Degrees of Freedom
1	Positive	23	48	0	1	1
2	Negative	295	270			

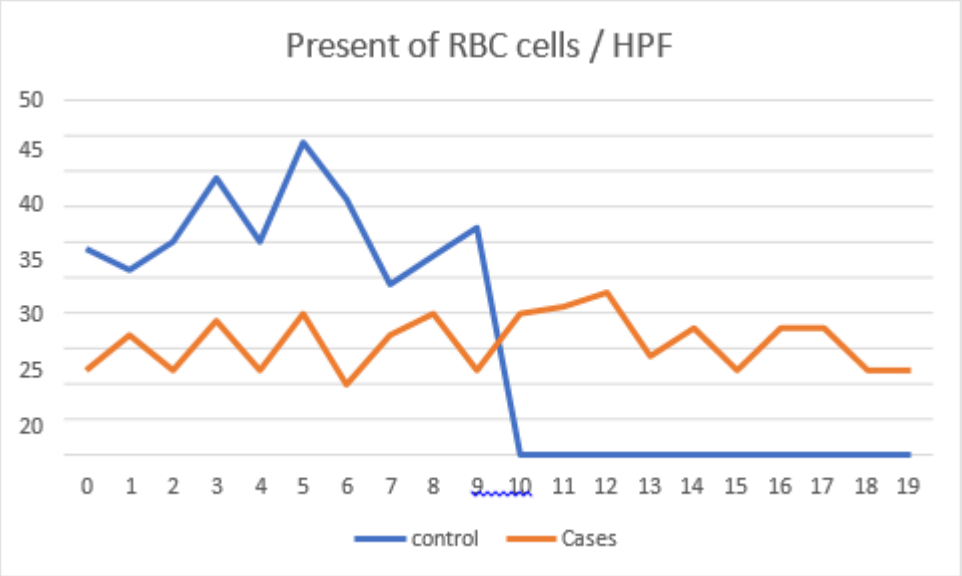


Bar Graph 11 showing the presence of nitrite in urine in control and cases group.

In present study we observed that the present of RBC cells / HPF in urine sample was most high in cases group which was 19 cells in 12 subject and where as in control group it was 9 cells in 32 control as given in table number 16 given below.

Table number 16 showing the present of RBC cells/ HPF found in control and cases group.

Present of RBC cells / HPF	control	Cases
0	29	12
1	26	17
2	30	12
3	39	19
4	30	12
5	44	20
6	36	10
7	24	17
8	28	20
9	32	12
10	0	20
11	0	21
12	0	23
13	0	14
14	0	18
15	0	12
16	0	18
17	0	18
18	0	12
19	0	12

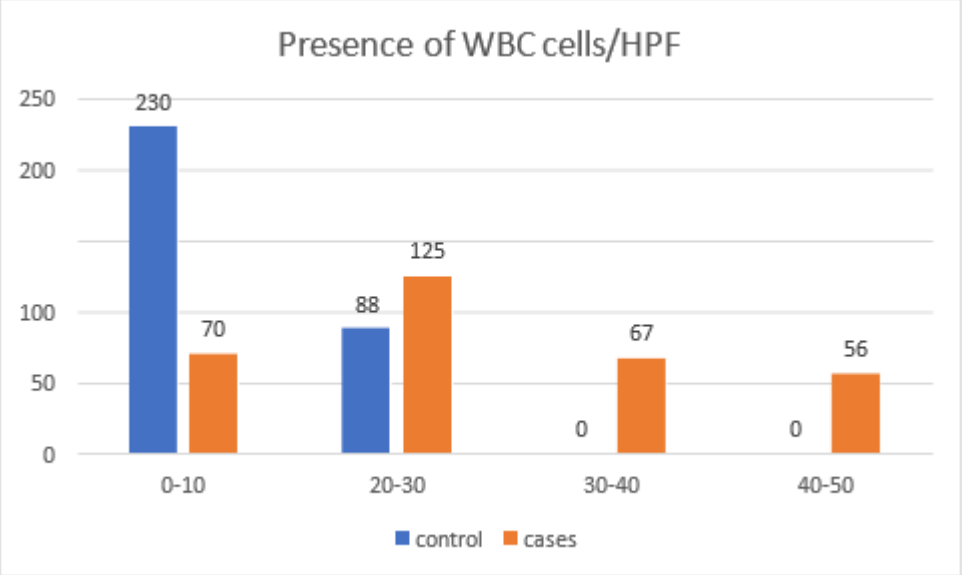


Line chart 1 showing the present of RBC cells in our both control and cases group.

In current study we observed that the presence of WBC cells/ HPF in control group it was 0-10 in 230 cases and in case group 20-30 in 125 subjects rest show in table number 17 given below.

Table number 17 showing the presence of WBC cells/ HPF in cases and control

Presence of WBC cells/HPF	control	cases
0-10	230	70
20-30	88	125
30-40	0	67
40-50	0	56

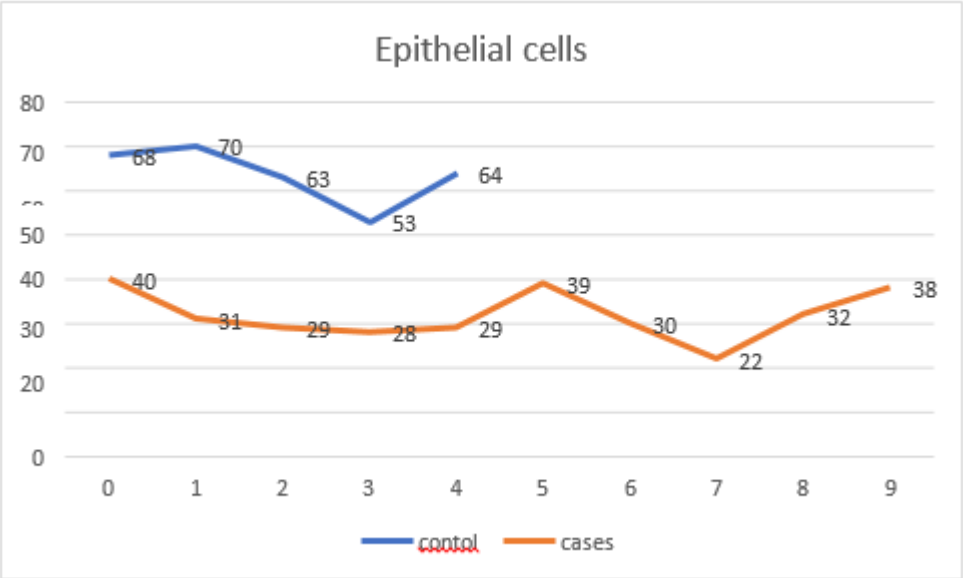


Bar graphs number 12 showing Presence of WBC cells/ HPF in control and cases group.

In our present study we observed that the presence of epithelial cells in urine as followed in control group it was 0,1,2,3,4, cells in 68,70,63,53,64 where as in cases group it was 40,31,29,28,29,39,30,22,32and 38 subjects having cells number as followed 0,1,2,3,4,5,6,7,8 and 9 given below in table number 18.

Table number 18. Showing the presence of epithelial cells in urine sample in both control and cases group.

Epithelial cells	control	cases
0	68	40
1	70	31
2	63	29
3	53	28
4	64	29
5		39
6		30
7		22
8		32
9		38

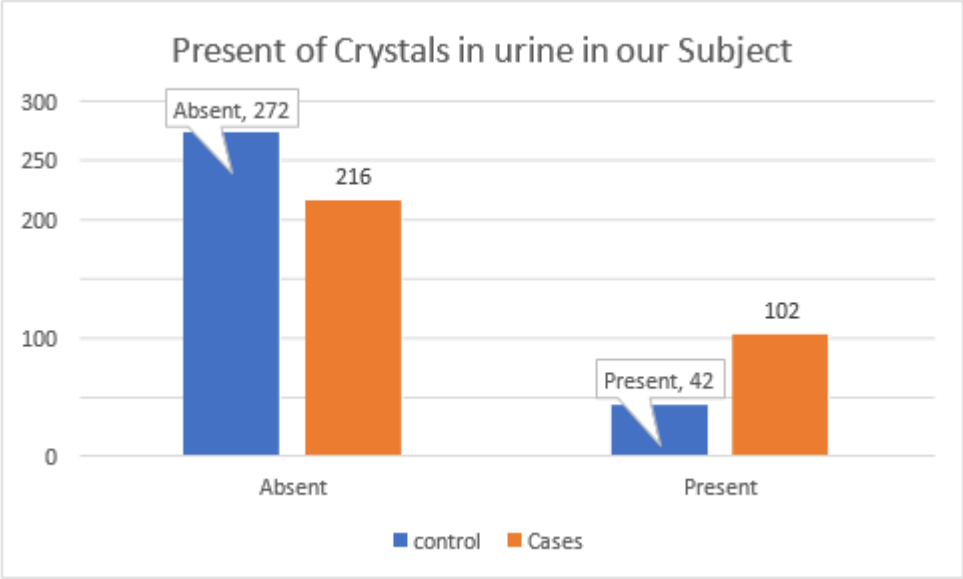


Line chart 2 showing the present of epithelial cells in our both control and cases group.

In our present study we observed 272 subjects showing absent of 272 subjects showing absent of urine and 42 subjects showing present of crystals where as in cases group it was 216 having absent and 102 having present of crystals.

Table number 19 showing the present of crystals having control and cases

S. No	Crystals	control	Cases	Chi-Square	p-value	Degrees of Freedom
1	Absent	272	216	1.64	0.20	1
2	Present	42	102			

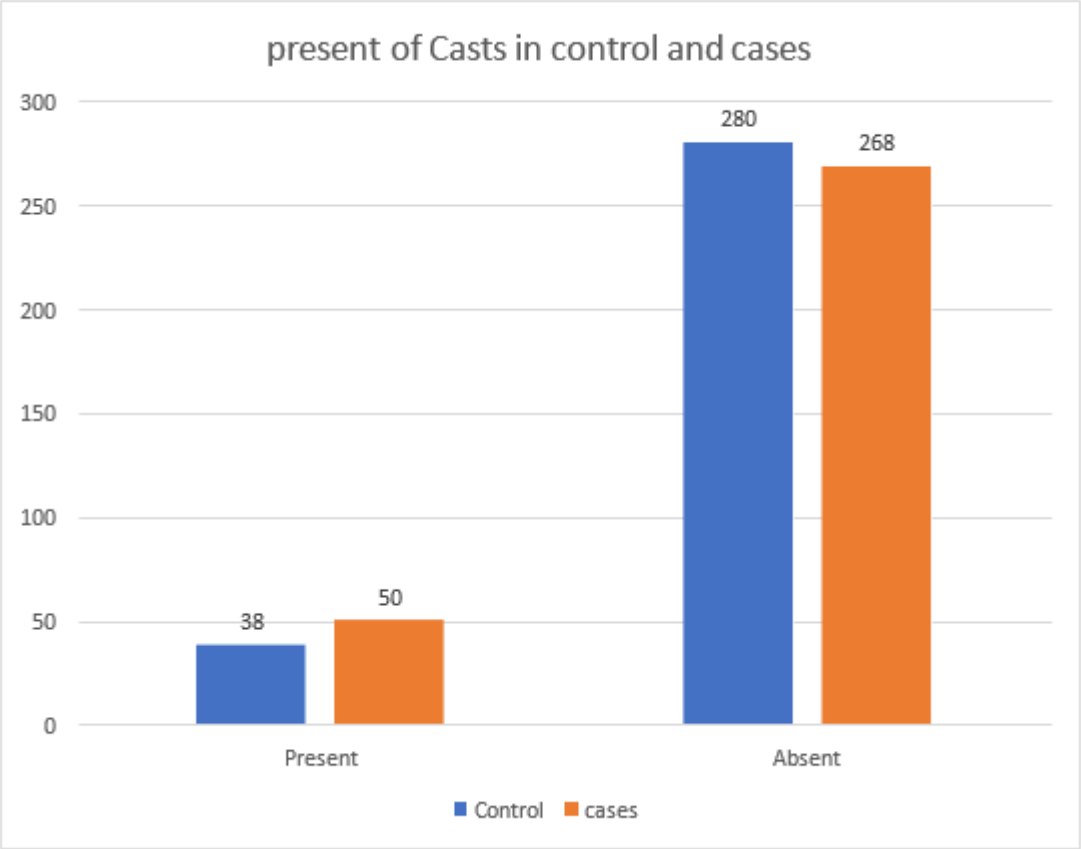


Bar graphs number 13 showing Presence of crystal in urine of control and cases group.

In our present study we observed that there was the absent of 280 and 38 present in control group where as in cases group it was 50 subject having present cast and 268 having absent in cases group as given in table number

Table number 20 Showing the presence of Casts in both control and cases group.

S. No	Casts	Control	cases	Chi-Square	p- value	Degrees of Freedom
1	Present	38	50	0.49	0.48	1
2	Absent	280	268			

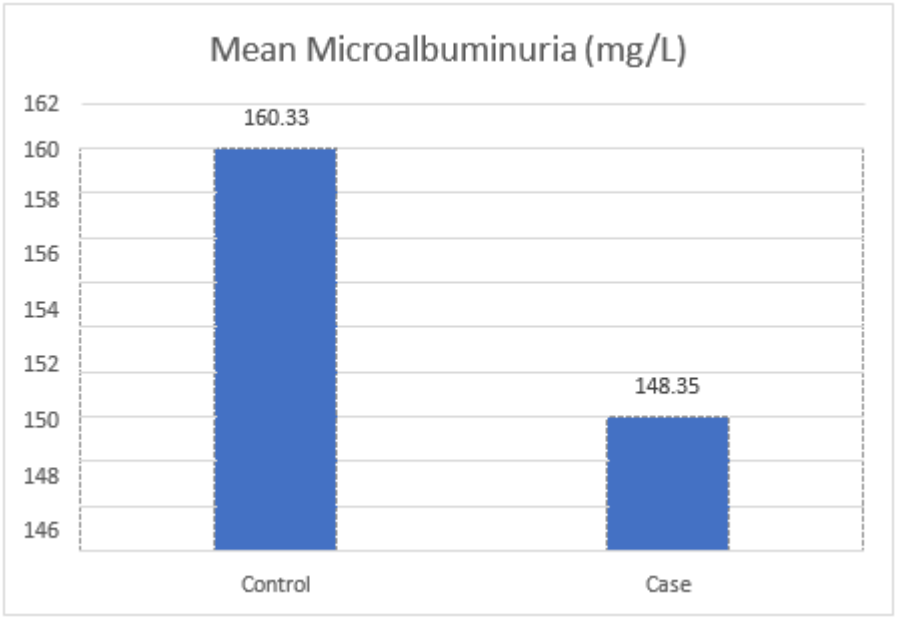


Bar graphs number 14 showing Presence of casts in urine of control and cases group.

In our present study we observed that the mean microalbuminuria was 160.33 mg/l present in control and in cases group it was 148.35 mg/L as static description given in table number 21 given below.

Table number 21 showing the presence of microalbuminuria in urine sample both in control and cases group.

	Mean	Standard deviation	Standard Error	T value	P Value
Control	160.33	78.31	4.39	26.45	0
Case	148.35	86.75	4.86	1.83	0.07

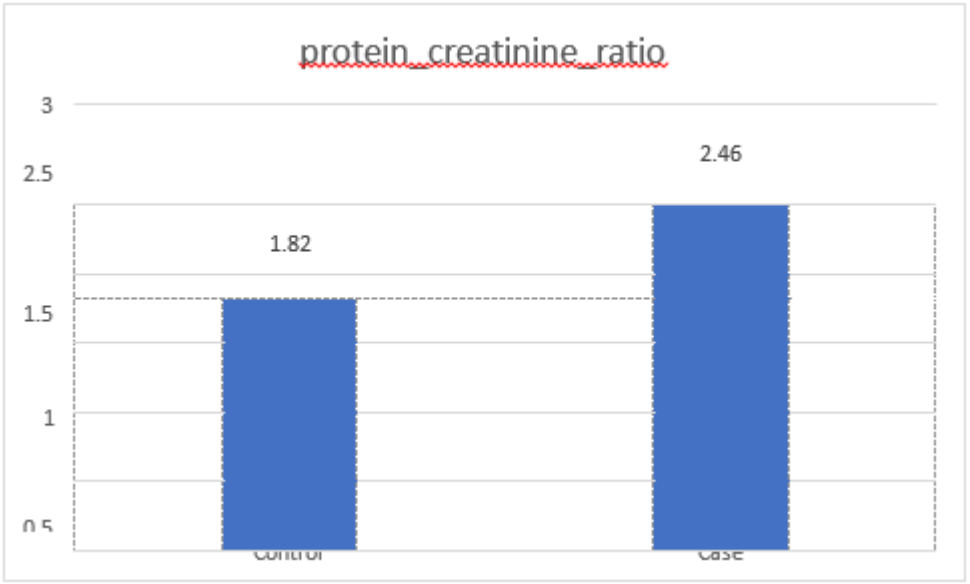


Bar graphs number 15 showing the presence of Microalbuminuria in control and cases group.

In our present study we observed that the mean protein creatine ratio was 1.82 in control group and 2.46 in cases group as static description given in table number 22 given below.

Table number 22 showing the Protein creatine ratio in control and cases group.

S. No	protein_creatinine_ratio	Mean	Standard deviation	Standard Error	T value	P Value
1	Control	1.82	0.99	0.06	6.72	0
2	Case	2.46	1.39	0.08		

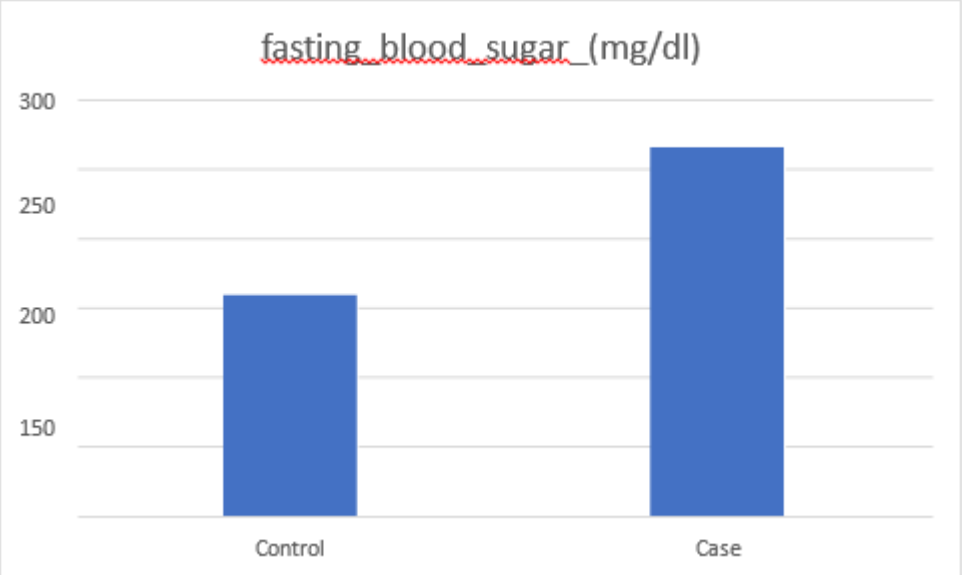


Bar graphs number 16 showing the presence of Protein creatine ratio in control and cases group.

In our present study we observed that the mean fasting blood sugar in control group was 158.6 mg/dl in cases group 264.6 g/dl as static description given in table number 23 given below.

Table number 23 showing the mean fasting blood sugar in both control and cases group.

S. No	fasting_blood_sugar_(mg/dl)	Mean	Standard deviation	Standard Error	T value	P Value
1	Control	158.6	52.38	2.94	19.5	0
2	Case	264.6	81.58	4.57		

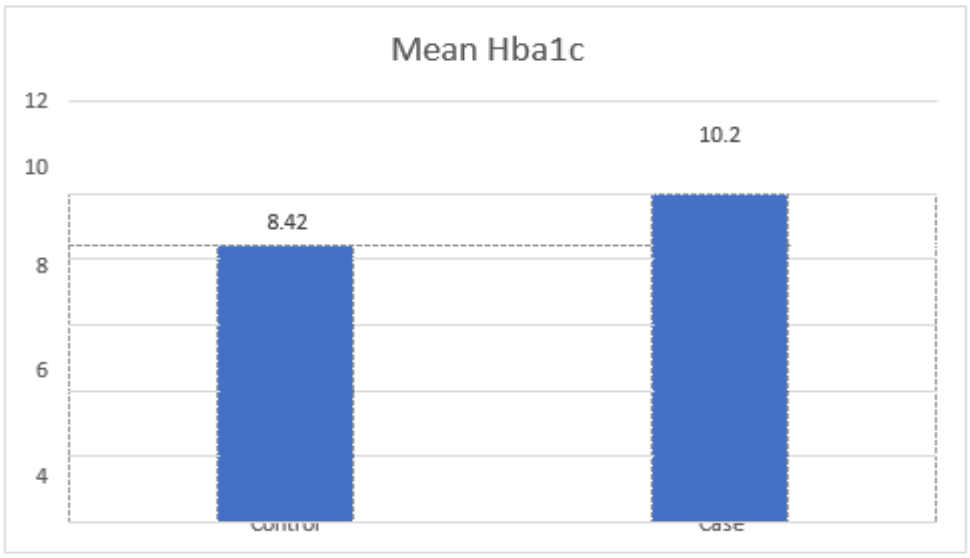


Bar graphs number 17 showing the presence of fasting_blood_sugar_(mg/dl) in control and cases group.

In our present study we observed that the mean Hba1c in control group was
8.42 in cases group 10.2 mg/dl as static description given in table number 24 given below.

Table number 24 showing the mean Hba1c in control and cases group.

S. No	Hba1c	Mean	Standard deviation	Standard Error	T value	P Value
1	Control	8.42	2.07	0.12	10.96	0
2	Case	10.2	2.01	0.11		

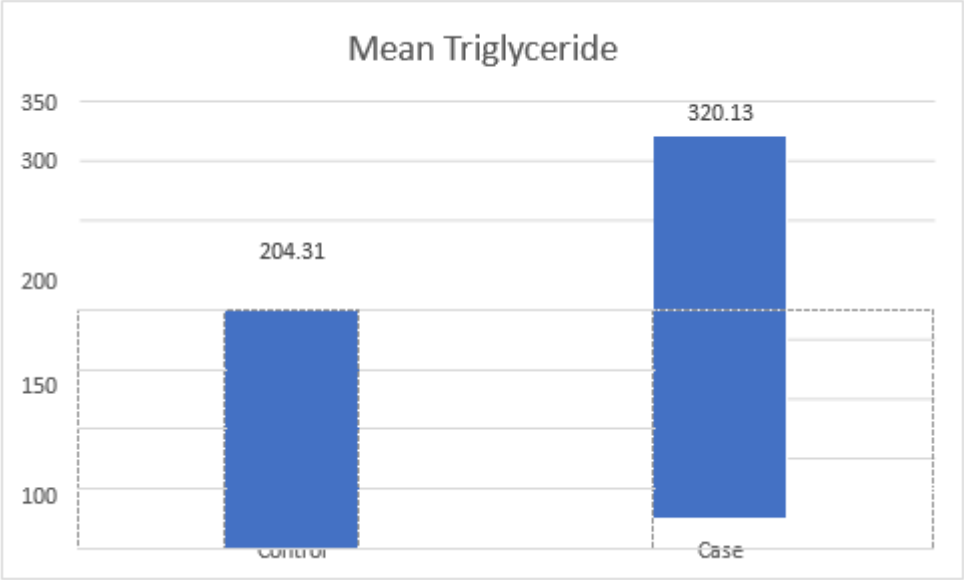


Bar graphs number 18 showing the presence of Hba1c in control and cases group.

In our present study we observed that the mean Triglyceride in control group was 204.31 in cases group 320.13 as static description given in table number 25 given below.

Table number 25 showing the mean Triglyceride in control and cases group.

S. No	Triglyceride	Mean	Standard deviation	Standard Error	T value	P Value
1	Control	204.31	54.49	3.06	17.5	0
2	Case	320.13	104.7	5.87		

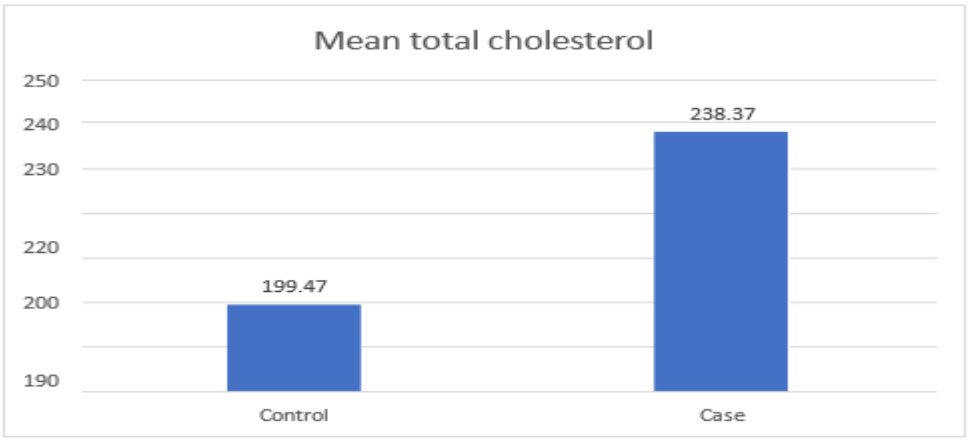


Bar graphs number 19 showing the presence of Mean Triglyceride in control and cases group.

In our present study we observed that the mean total cholesterol in control group was 199.47 in cases group 238.37 as static description given in table number 26 given below.

Table number 26 showing the mean total cholesterol in control and cases group.

S. No	total cholesterol	Mean	Standard deviation	Standard Error	T value	P Value
1	Control	199.47	29.1	1.63	15.26	0
2	Case	238.37	34.91	1.96		

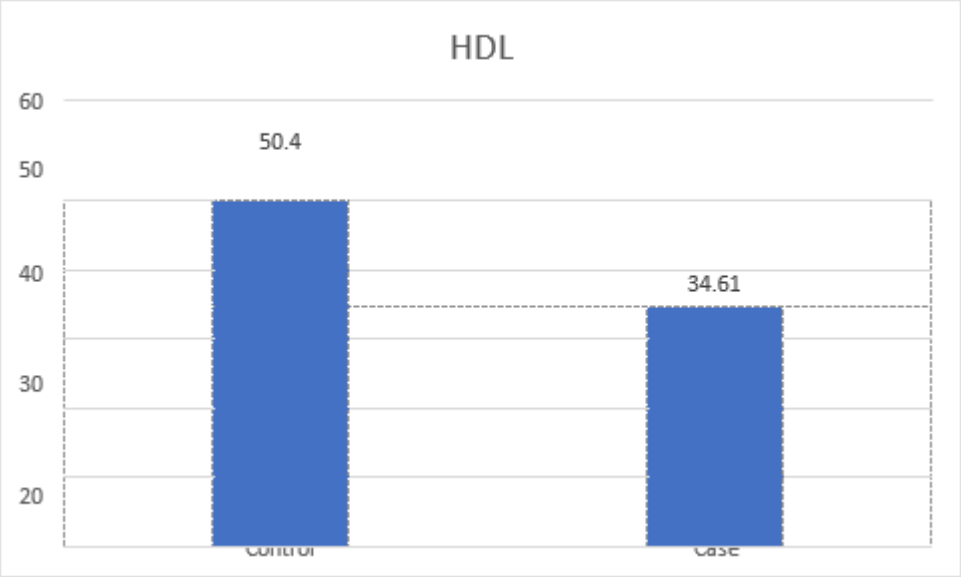


Bar graphs number 20 showing the presence of Mean total Cholesterol in control and cases group.

In our present study we observed that the mean HDL in control group was 50.4 in cases group 34.61 as static description given in table number 27 given below.

Table number 27 showing the mean HDL in control and cases group.

S. No	HDL	Mean	Standard deviation	Standard Error	T value	P Value
1	Control	50.4	12.11	0.68	-18.75	0
2	Case	34.61	8.89	0.5		

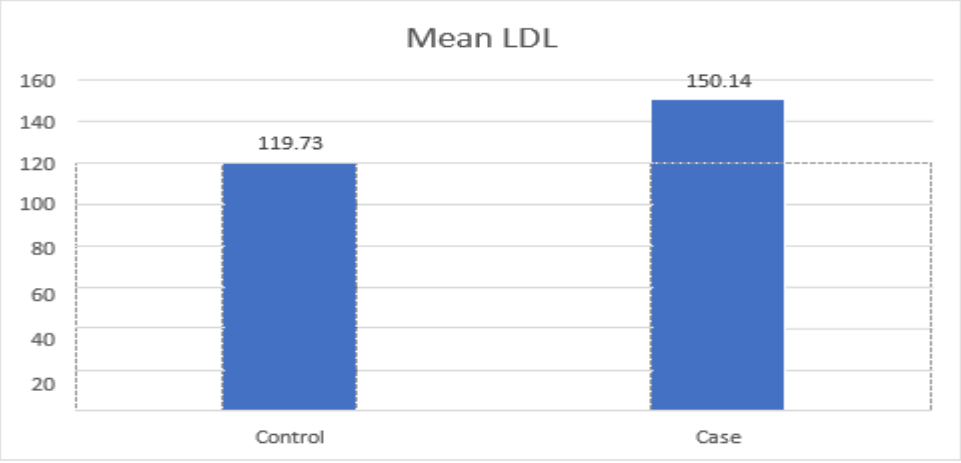


Bar graphs number 21 showing the presence of Mean HDL in control and cases group.

In our present study we observed that the mean LDL in control group was
119.73 in cases group 150.14 as static description given in table number 28 given below.

Table number 28 showing the mean LDL in control and cases group.

S. No	LDL	Mean	Standard deviation	Standard Error	T value	P Value
1	Control	119.73	22.48	1.26	14.76	0
2	Case	150.14	29.05	1.63		

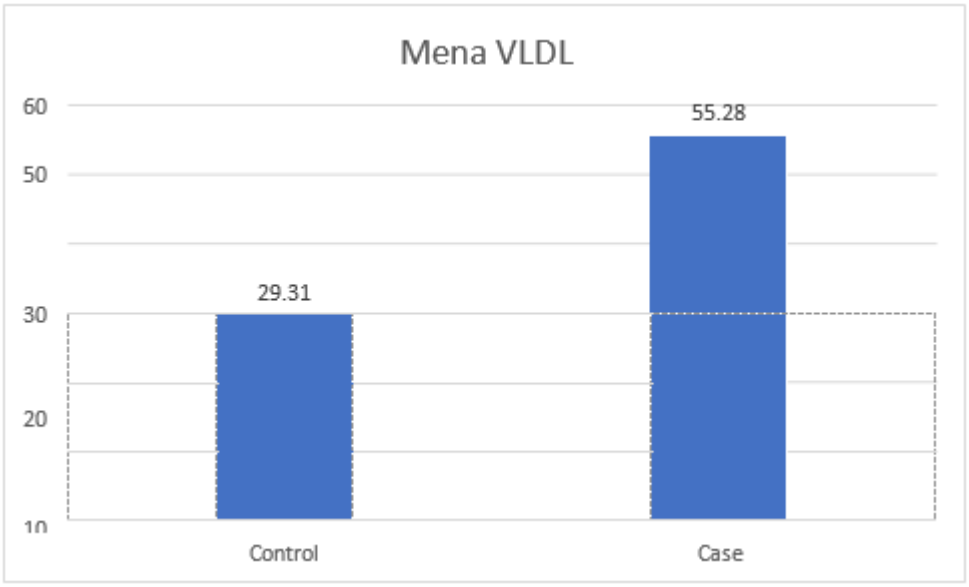


Bar graphs number 22 showing the presence of Mean LDL in control and cases group

In our present study we observed that the mean VLDL in control group was 29.31 in cases group 55.28 as static description given in table number 29 given below.

Table number 29 showing the mean VLDL in control and cases group.

S. No	VLDL	Mena	Standard deviation	Standard Error	T value	P Value
1	Control	29.31	11.87	0.67	25.29	0
2	Case	55.28	13.94	0.78		



Bar graphs number 23 showing the presence of Mean VLDL in control and cases group

This table number 30 showing presents the effect size for different health- related parameters using two statistical measures: Cohen's d and Eta Squared (η^2).

1. Understanding Effect Size Metrics

- Cohen's d: Measures the standardized mean difference between two groups. It helps determine the practical significance of an effect.
  - Small Effect: d = 0.2
  - Medium Effect: d = 0.5
  - Large Effect: d = 0.8 or higher
  - Negative values indicate the direction of the difference.
- Eta Squared (η^2): Measures the proportion of variance in a dependent variable explained by an independent variable. It is commonly used in ANOVA.
  - Small Effect: $\eta^2 = 0.01$
  - Medium Effect: $\eta^2 = 0.06$
  - Large Effect: $\eta^2 = 0.14$ or higher

Interpretation of the Table Parameters with Large Effects

- Urobilinogen (Cohen's d = 2.10, $\eta^2 = 0.52$): Large effect size, meaning there is a strong difference between groups.
- VLDL (Cohen's d = 2.01, $\eta^2 = 0.50$): Large effect size, indicating significant variation.
- Fasting Blood Sugar (Cohen's d = 1.55, $\eta^2 = 0.37$): Shows a strong difference in fasting blood sugar levels.
- HDL (Cohen's d = -1.49, $\eta^2 = 0.36$): Negative effect size indicates a reduction in HDL levels.
- Triglyceride (Cohen's d = 1.39, $\eta^2 = 0.33$): High effect, meaning triglycerides significantly differ between groups.

Parameters with Medium Effects

- LDL (Cohen's d = 1.17, $\eta^2 = 0.26$): Medium to large effect.
- Total Cholesterol (Cohen's d = 1.21, $\eta^2 = 0.27$): Medium effect.
- Epithelial Cells (Cohen's d = 1.08, $\eta^2 = 0.23$): Moderate difference.

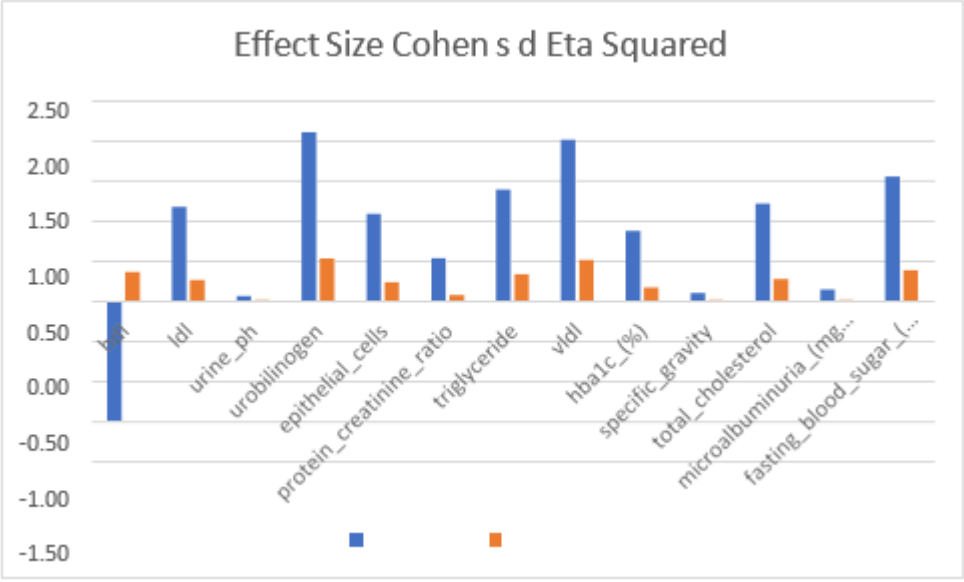
- HbA1c (%) (Cohen's d = 0.87, $\eta^2 = 0.16$): Medium effect, indicating a notable variation in HbA1c levels.

Parameters with Small Effects

- Protein Creatinine Ratio (Cohen's d = 0.53, $\eta^2 = 0.07$): Small to medium effect.
- Microalbuminuria (Cohen's d = 0.15, $\eta^2 = 0.01$): Small effect, meaning little difference between groups.
- Urine pH (Cohen's d = 0.06, $\eta^2 = 0.00$): Almost no effect.
- Specific Gravity (Cohen's d = 0.10, $\eta^2 = 0.00$): No meaningful effect.

Table number 30 Showing Effect Size

	Cohen's d	Eta Squared
Hdl	-1.49	0.36
Ldl	1.17	0.26
Urine_ph	0.06	0.00
Urobilinogen	2.10	0.52
Epithelial_cells	1.08	0.23
Protein_creatinine_ratio	0.53	0.07
Triglyceride	1.39	0.33
Vldl	2.01	0.50
Hba1c_(%)	0.87	0.16
Specific_gravity	0.10	0.00
Total_cholesterol	1.21	0.27
Microalbuminuria_(mg/l)	0.15	0.01
Fasting_blood_sugar_(mg/dl)	1.55	0.37

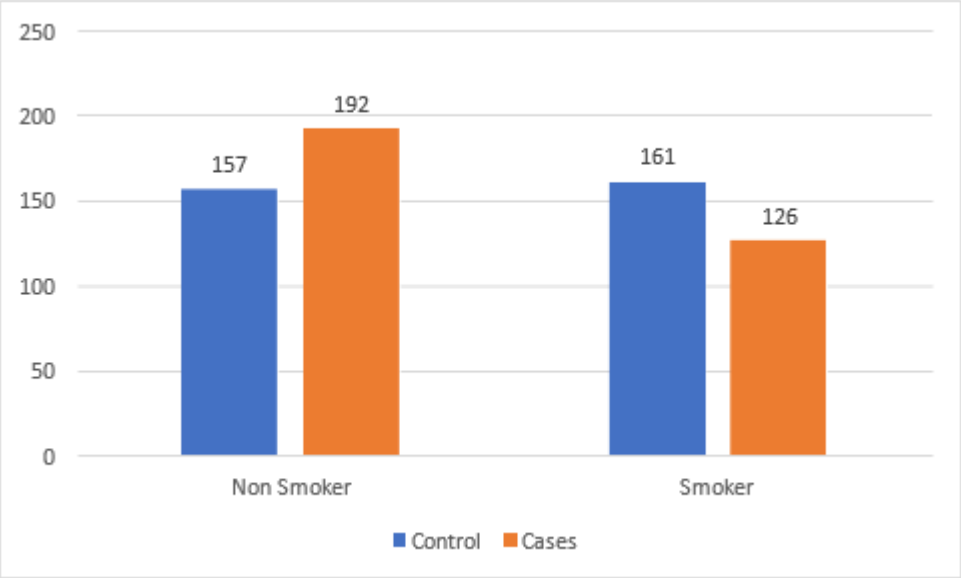


Bar graphs **number** 24 showing the presence of Effect Size Cohen s d Eta Squared

In our present study we divided subjects of control and cases in smoker and non smoker group in which we obtained non smoker 157 and 161 smoker in control group where as in cases group we obtained 192 non smoker and 126 smoker. As show in table number 31

Table number 31 showing the smoker and non smoker in our subjects.

S. No	Parameter	Control	Cases
1	Non Smoker	157	192
2	Smoker	161	126

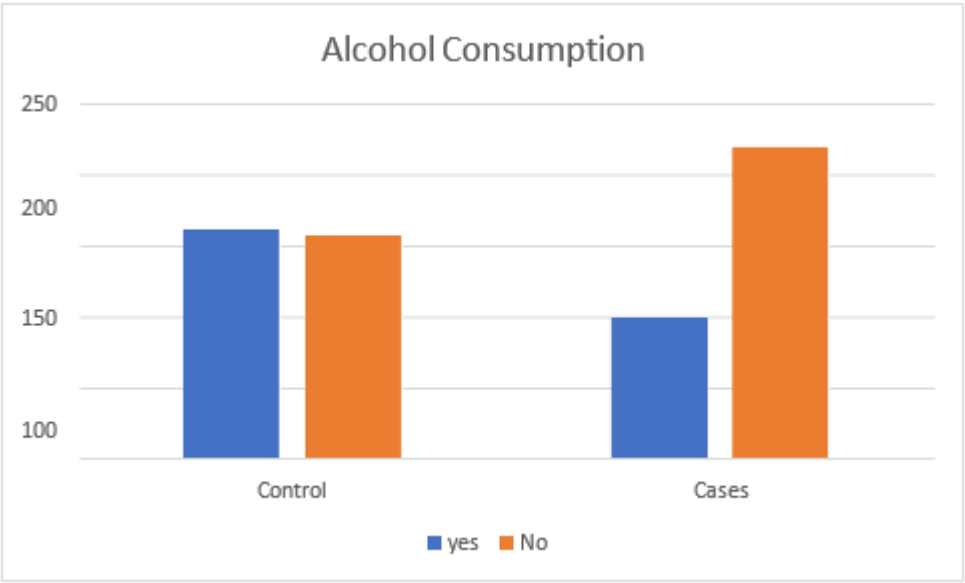


Bar graphs number 25 showing the smoker and nonsmoker in our subjects.

In our present study we divided subjects of control and cases in sAlcohol Consumption group in which we obtained yes 161 and 157 no in control group where as in cases group we obtained 192 yes and 219 no. As show in table number 32

Table number 32 showing the Alcohol Consumption in our subjects.

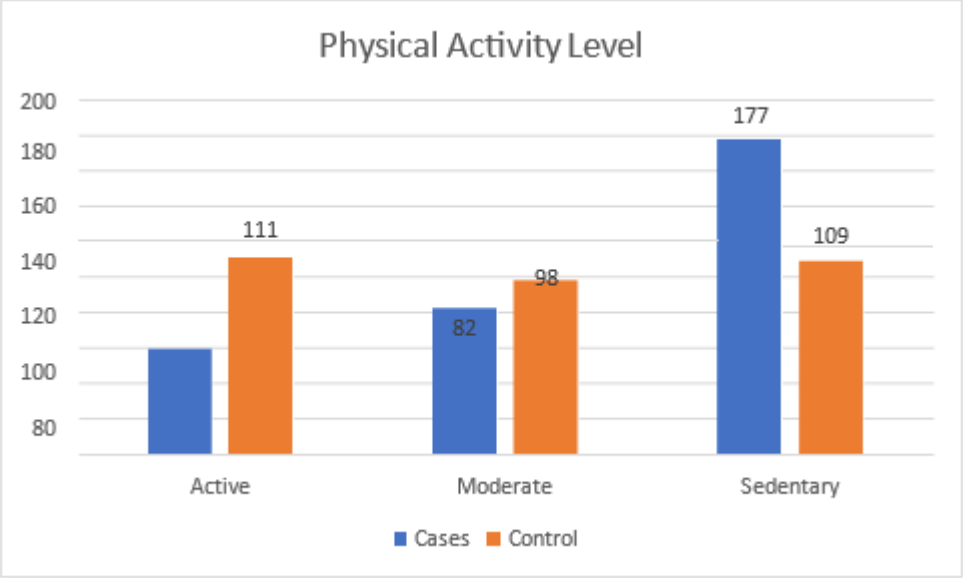
S. No	Alcohol Consumption	yes	No
1	Control	161	157
2	Cases	99	219



Bar graphs number 26 showing Alcohol Consumption in our subjects.

Table number 33 showing the Physical Activity level in our subjects.

S. No	Physical Activity Level	Active	Moderate	Sedentary
1	Cases	59	82	177
2	Control	111	98	109



Bar graphs number 27 showing Physical Activity Level in our subjects.

Control Group Correlation Matrix

1. **Urine pH vs. Total Cholesterol (-0.12)**
   - A weak negative correlation, indicating that as urine pH increases, total cholesterol tends to decrease slightly.
2. **Specific Gravity vs. HBA1c (0.06)**
   - A weak positive correlation, suggesting that specific gravity may slightly increase with higher HbA1c levels.
3. **Microalbuminuria vs. Urine pH (-0.10)**
   - A weak negative correlation, meaning higher urine acidity might be associated with increased microalbuminuria levels.
4. **RBC Cells vs. HDL (-0.11)**
   - A slight negative correlation, suggesting that more RBC cells in urine might be linked with lower HDL levels.

Table number 34 showing the Control Group Correlation Matrix

	Urine pH	Specific Gravity	Urobilinogen	RBCs (cells/HPF)	WBCs (cells/HPF)	Epithelial Cells	Microalbuminuria (mg/L)	Protein Creatinine Ratio	Fasting Blood Sugar (mg/dL)	HBA1c (%)	Triglyceride	Total Cholesterol	HDL	LDL	VLDL
Urine pH	1	-0.067029116	0.000695049	-0.116274214	0.046037303	-0.121517923	0.041554957	0.090491653	0.017930613	-0.073721309	0.008995819	0.013690 41	-0.043593 54	-0.015166125	0.049752298
Specific Gravity	-0.067029116	1	0.019076217	0.119781034	0.002398441	-0.037210132	-0.048830598	-0.035732425	-0.031578509	-0.024574706	-0.020253616	0.006861369	-0.020157 52	0.138612309	0.051534411
Urobilinogen	0.000695049	0.019076217	1	-0.036349617	-0.066253862	-0.082847112	0.017317994	0.028238593	0.058861841	0.065288482	-0.005089 05	0.033517364	0.002268743	0.015672649	0.000755108
RBCs (cells/HPF)	-0.116274214	0.119781034	-0.036349617	1	-0.054342124	0.043178877	0.075203829	0.032062195	-0.002501256	0.065614954	-0.029174726	0.065309743	0.050794026	-0.043621238	-0.047387485
WBCs (cells/HPF)	0.046037303	0.002398441	-0.066253862	-0.054342124	1	-0.022845152	0.023580976	-0.012519317	0.082401709	-0.008816532	-0.031086769	0.017136556	-0.030210772	0.032002696	0.039970471
Epithelial Cells	-0.121517923	-0.037210132	-0.082847112	0.043178877	-0.022845152	1	-0.029627	-0.045774575	-0.036158273	0.054161662	-0.091643239	-0.132565737	0.014595097	-0.147799673	0.002227 82
Microalbuminuria (mg/L)	0.041554957	-0.048830598	0.017317994	0.075203829	0.023580976	-0.029627	1	0.039748342	-0.069712 26	0.070128772	-0.051734884	0.044358303	0.007767077	-0.081137494	0.039220 97
Protein Creatinine Ratio	0.090491653	-0.035732425	0.028238593	0.032062195	-0.012519317	-0.045774575	0.039748342	1	-0.074803074	-0.044442098	-0.108279484	-0.042461758	-0.008700639	0.059075158	-0.067564231
Fasting Blood Sugar (mg/dL)	0.017930613	-0.031578509	0.058861841	-0.002501256	0.082401709	-0.036158273	-0.06971226	-0.074803074	1	-0.052398 38	-0.046608212	-0.015232894	0.069157908	-0.017431889	0.024970436
HBA1c (%)	-0.073721309	-0.024574706	0.065288482	0.065614954	-0.008816532	0.054161662	0.070128772	-0.044442098	-0.052398 38	1	-0.047187674	0.012626609	-0.017149708	0.091954311	0.007997016
Triglyceride	0.008995819	-0.020253616	-0.005089 05	-0.029174726	-0.031086769	-0.091643239	-0.051734884	-0.108279484	-0.046608212	-0.047187674	1	0.036276512	0.045752947	0.003063058	-0.053493258

Cases Group Correlation Matrix

1. Fasting **Blood** Sugar vs. HbA1c (Strong Positive Correlation)

o As expected, fasting blood sugar is strongly correlated with HbA1c, indicating a close link between chronic and acute glucose levels.

2. Triglycerides vs. LDL & VLDL (High Positive Correlation)

o Higher triglycerides are strongly linked with LDL and VLDL, a key cardiovascular risk factor.

3. Microalbuminuria vs. Protein Creatinine Ratio

o A moderate correlation indicates possible kidney impairment in cases.

Table number 35 showing the Cases l Group Correlation Matrix

	Urine pH	Specific Gravity	Urobilinogen	RBC cells/HPF	WBC cells/HPF	Epithelial cells	Microalbuminuria (mg/L)	Protein Creatinine Ratio	Fasting Blood Sugar (mg/dL)	HBA1c (%)	Triglyceride	Total Cholesterol	HDL	LDL	VLDL
Urine pH	1	-0.108257368	-0.063073092	0.007571678	-0.063665819	-0.072306973	-0.102939591	-0.033988733	0.042087432	-0.118731419	-0.053989886	-0.123277252	-0.04021072	-0.040157	-0.022902533
Specific Gravity	-0.108257368	1	-0.097671622	-0.082574737	-0.015172416	-0.066435301	0.050113126	-0.036423585	-0.038433682	0.059148434	-0.0482708	0.063559654	0.040039392	-0.003719003	-0.020999963
Urobilinogen	-0.063073092	-0.097671622	1	0.0788544	-0.02267491	0.075612333	-0.050842626	-0.097787626	0.001069228	-0.023740609	0.019164882	-0.098170064	-0.090057036	0.010708761	-0.035108602
RBC cells/HPF	0.007571678	-0.082574737	0.0788544	1	0.046722939	-0.022821601	-0.028510044	-0.051362765	0.023206009	0.017830681	0.006009854	0.038614933	-0.11612068	-0.055240402	-0.0631456
WBC cells/HPF	-0.063665819	-0.015172416	-0.02267491	0.046722939	1	0.076048903	0.064529103	0.027668304	-0.00339511	0.058310912	-0.02276041	0.075677254	-0.051413713	0.054919914	0.072198467
Epithelial cells	-0.072306973	-0.066435301	0.075612333	-0.022821601	0.076048903	1	-0.103235622	0.060232344	0.021637006	0.023271555	-0.102134342	0.025492213	0.012216663	0.001986134	8.74E-05
Microalbuminuria (mg/L)	-0.102939591	0.050113126	-0.050842626	-0.028510044	0.064529103	-0.103235622	1	0.15905067	-0.040283 29	-0.025632313	-0.044831054	-0.008710454	0.089501061	-0.09730457	-0.024948324
Protein Creatinine Ratio	-0.033988733	-0.036423585	-0.097787626	-0.051362765	0.027668304	0.060232344	0.15905067	1	-0.033055531	0.138819809	-0.031302268	-0.021593652	-0.055786329	-0.084499776	0.039982289
Fasting Blood Sugar (mg/dL)	0.042087432	-0.038433682	0.001069228	0.023206009	-0.00339511	0.021637006	-0.04028329	-0.033055531	1	0.008606461	0.107710252	-0.027425317	0.055861092	-0.069037153	-0.075664567
HBA1c (%)	-0.118731419	0.059148434	-0.023740609	0.017830681	0.058310912	0.023271555	-0.025632313	0.138819809	0.008606461	1	0.058229674	-0.105255033	-0.000473274	-0.014466677	-0.008777238
Triglyceride	-0.053989886	-0.0482708	0.019164882	0.006009854	-0.02276041	-0.102134342	-0.044831054	-0.031302268	0.107710252	0.058229674	1	-0.027510705	-0.015841496	0.005184497	0.029190837

Comparison:

- The case group shows stronger correlations between fasting blood sugar, HbA1c, and lipid profiles compared to the control group.
- The control group exhibits generally weaker correlations, indicating a more balanced metabolic state.
- Kidney function markers (microalbuminuria, protein-creatinine ratio) show more variation in the cases group, suggesting renal involvement.

7. DISCUSSION

This chapter presents a comprehensive interpretation of the findings obtained from the comparative study conducted among diabetic and non-diabetic individuals. The results have been critically analyzed in the context of existing literature to establish their scientific validity and clinical significance. Such comparisons not only support the trends observed in the present study but also help explain similarities and variations, thereby providing a broader understanding of metabolic, renal, and urinary alterations associated with diabetes mellitus.

Demographic and Anthropometric Characteristics

The present study included a total of 636 participants, equally divided into diabetic (case) and non-diabetic (control) groups. A nearly equal sex distribution was maintained in both groups. It was observed that a greater proportion of diabetic individuals belonged to the 40–50 years age group, whereas controls were more represented in the 50–60 years range. This finding is consistent with Bhambhani et al.[24], who reported that middle-aged individuals constitute a significant proportion of T2DM patients, indicating age as an important risk factor.

The mean body weight of diabetic participants (89.34 kg) was significantly higher than that of controls (74.17 kg), although the difference in BMI was not statistically significant. This suggests that body weight may be a stronger indicator of metabolic risk than BMI alone. Warjuka et al.[15] demonstrated a positive association between BMI and microalbuminuria,

while Bonora et al.[33] emphasized the role of BMI in predicting metabolic and renal complications. These findings highlight the importance of weight management in diabetes prevention.

Blood Pressure Comparison

Both systolic and diastolic blood pressures were higher in diabetic individuals (140/89 mmHg) compared to controls (129/85 mmHg). This observation is in agreement with Sudhakar et al.[27], who reported significantly elevated blood pressure among diabetic patients. Hypertension is a well-recognized comorbidity of diabetes and contributes to the progression of diabetic nephropathy. Bakris et al.[13] further emphasized that strict blood pressure control is essential to reduce renal and cardiovascular complications. The present findings reinforce the interrelationship between diabetes and hypertension.

Urine Analysis Parameters

Urinary pH and Specific Gravity

No statistically significant difference was observed in urinary pH and specific gravity between the two groups. This indicates that these parameters are not sensitive markers for early diabetic changes. Similar findings were reported by Sumida et al.[21], who suggested that specific markers such as protein-creatinine ratio (PCR) and albumin-creatinine ratio (ACR) are more reliable indicators of renal dysfunction.

Glucose, Ketone, and Bilirubin

A marked difference in urinary glucose excretion was observed between the two groups. While the majority of control participants were negative for glucosuria, diabetic individuals showed varying degrees of glucose positivity (1+ to 4+), reflecting uncontrolled hyperglycemia. The presence of ketones and bilirubin further indicates metabolic imbalance. Alam et al.[22] and Shabani et al.[31] reported similar findings, highlighting glucosuria and ketonuria as indicators of poor glycemic control.

Urobilinogen, Nitrite, and Blood

Urinary urobilinogen levels were significantly higher in diabetics, suggesting altered metabolic and hepatobiliary function. Increased nitrite positivity and hematuria indicate a higher susceptibility to urinary tract infections and microvascular damage. Warjuka et al.[15] and related studies support these findings, indicating compromised renal integrity in diabetic individuals.

Renal Function Biomarkers

Microalbuminuria

In the present study, microalbuminuria levels did not show a statistically significant difference between groups. Although unexpected, this may be due to early-stage disease or subclinical renal involvement in controls. Bakris et al.[13] reported that microalbuminuria can predict renal risk even in non-diabetic individuals, supporting its role as a general marker of renal dysfunction.

Protein-Creatinine Ratio (PCR)

The protein-creatinine ratio was significantly higher in diabetic subjects, indicating early renal involvement. This finding is consistent with Kara et al.[14], Kaminska et al.[20], and Karthikeyan et al.[19], who demonstrated that PCR is a reliable and practical marker for detecting proteinuria. The present study supports PCR as a superior early diagnostic tool compared to microalbuminuria alone.

Glycemic Control Indicators

Fasting blood sugar and HbA1c levels were significantly elevated in diabetic individuals, reflecting poor glycemic control. These findings are consistent with Baranwal et al.[25] and Cai et al.[29], who reported a strong association between elevated HbA1c and metabolic complications. HbA1c serves as a key indicator for long-term glycemic status and risk of diabetic complications.

Lipid Profile Alterations

Triglycerides and VLDL

Triglyceride and VLDL levels were significantly elevated in diabetics, indicating characteristic diabetic dyslipidemia. Athyros et al.[26] reported similar findings, with a high prevalence of mixed dyslipidemia in T2DM. These abnormalities increase cardiovascular risk and require clinical attention.

Total Cholesterol, LDL, and HDL

Diabetic individuals exhibited increased total cholesterol and LDL levels along with reduced HDL levels, forming an atherogenic lipid profile. These findings align with Bhambhani et al.[24] and Gordon et al.[28]. Such lipid abnormalities significantly contribute to cardiovascular morbidity. Studies by Panahi et al.[30] and Shabani et al.[31] suggest that adjunct therapies may help improve lipid parameters.

Urinary Sediments and Cellular Components
Higher prevalence of urinary RBCs, WBCs, epithelial cells, casts, and crystals was observed in diabetic individuals, indicating renal inflammation and structural damage. Chauhan et al.[23] reported similar findings, emphasizing the diagnostic value of urinary sediment analysis in detecting renal involvement.

Lifestyle and Behavioral Factors
A greater proportion of diabetic participants were sedentary, supporting the role of physical inactivity as a major modifiable risk factor. Ko et al.[32] demonstrated that combined pharmacological and lifestyle interventions improve metabolic outcomes. Behavioral changes such as reduced smoking and alcohol intake in diabetics may reflect post-diagnosis lifestyle modifications.

Correlation Analysis and Effect Size Interpretation
Stronger correlations among glycemic, lipid, and renal parameters were observed in diabetic individuals, indicating multisystem involvement. Effect size analysis identified triglycerides, VLDL, HDL, and urobilinogen as strong discriminators. Alicic et al.[34] emphasized the importance of improved biomarkers in diabetic kidney disease, while McGill et al.[17] supported the role of albuminuria in prognostic evaluation.

8. CONCLUSION

The present study demonstrates that individuals with type 2 diabetes mellitus exhibit significant alterations in metabolic, lipid, and urinary parameters when compared to non-diabetic controls. Diabetic subjects showed markedly elevated fasting blood sugar, HbA1c, triglycerides, low-density lipoprotein (LDL), and very low-density lipoprotein (VLDL) levels, along with reduced high-density lipoprotein (HDL) levels. These findings reflect poor glycemic control and a characteristic atherogenic lipid profile, indicating an increased risk of cardiovascular complications.

The presence of glucosuria, ketonuria, and hematuria further highlights the metabolic imbalance and suggests early renal involvement in diabetic individuals. Elevated glycemic indices, particularly HbA1c, indicate prolonged hyperglycemia, which plays a central role in the development of chronic complications such as diabetic neuropathy, nephropathy, and retinopathy. Similarly, dyslipidemia observed in the study contributes significantly to the progression of atherosclerosis and cardiovascular morbidity.

Although microalbuminuria did not show a statistically significant difference between the groups, the protein-to-creatinine ratio (PCR) was notably elevated in diabetic patients, suggesting its greater reliability as an early indicator of renal dysfunction. These findings emphasize the importance of incorporating sensitive and practical biomarkers into routine screening for early detection of diabetic nephropathy.

The study highlights that diabetes mellitus is a multisystem disorder with profound metabolic and renal implications. Early diagnosis and timely intervention are crucial to prevent the progression of complications. Comprehensive management strategies should include regular monitoring of blood glucose levels, lipid profiles, and renal function, along with lifestyle modifications and appropriate pharmacological therapy.

Despite providing valuable insights, the study is limited by its cross-sectional design and relatively modest sample size. Therefore, further large-scale, longitudinal studies incorporating diverse populations and advanced biomarkers are recommended to better understand disease progression and improve early diagnostic strategies.

In conclusion, proactive and multidisciplinary management of diabetes, combining early screening, lifestyle intervention, pharmacotherapy, and continuous monitoring, is essential to reduce morbidity and improve the overall quality of life in patients with type 2 diabetes mellitus.

Important finding for clinicians

1. Early Detection of Diabetic Nephropathy: - The study highlights the usefulness of spot urine microalbumin and protein-creatinine ratio (PCR) in identifying early renal involvement in newly diagnosed Type 2 Diabetes Mellitus (T2DM) patients, even before overt clinical symptoms appear.
2. Non-Invasive and Patient-Friendly Approach: - The use of spot urine samples provides a simple, non-invasive, and convenient method for assessing renal function, improving patient compliance compared to cumbersome 24-hour urine collection.
3. Cost-Effective Screening Tool: - Microalbuminuria and PCR estimation are economical and easily accessible investigations, making them suitable for routine screening, especially in resource-limited settings like primary healthcare centers.
4. Clinical Utility in Routine Practice: - The findings support the integration of these urinary biomarkers into routine

clinical protocols for early diagnosis, monitoring, and management of diabetic nephropathy.

5. Correlation with Metabolic Parameters: - The study demonstrates the association of renal markers with glycemic status, lipid profile, BMI, and blood pressure, providing a comprehensive understanding of the metabolic risk factors contributing to renal damage.
6. Reduction in Disease Burden: -Early identification of renal impairment allows timely intervention, which can prevent or delay progression to chronic kidney disease (CKD) and reduce associated morbidity and mortality.
7. Guidance for Therapeutic Decisions: - The study aids clinicians in initiating early therapeutic strategies such as strict glycemic control

Limitations of the Study

1. Cross-Sectional Study Design: - The present study was conducted using a cross-sectional design, which limits the ability to establish a causal relationship between microalbuminuria, protein-creatinine ratio, and progression of diabetic nephropathy. It only provides a snapshot of associations at a single point in time.
2. Single-Center Study: - The study was carried out in a single tertiary care center, which may limit the generalizability of the findings to broader populations, especially rural or different demographic groups.
3. Limited Follow-Up Data: - Since the study focused on newly diagnosed Type 2 Diabetes Mellitus (T2DM) patients without long-term follow-up, it was not possible to assess the progression of renal dysfunction or predict long-term outcomes such as chronic kidney disease (CKD).
4. Reliance on Spot Urine Samples: - Although spot urine microalbumin and protein-creatinine ratio (PCR) are practical and widely accepted, they may be influenced by factors such as hydration status, physical activity, and diurnal variation. This may introduce variability compared to 24-hour urine collection, which is considered a gold standard.
5. Potential Confounding Factors: - Factors such as diet, physical activity, medication use, smoking, alcohol intake, and comorbid conditions (e.g., hypertension, obesity) may influence renal parameters but were not fully controlled or stratified in the analysis.
6. Exclusion of Advanced Diagnostic Markers:- More sensitive biomarkers of early renal dysfunction such as cystatin C, NGAL (Neutrophil Gelatinase-Associated Lipocalin), or imaging-based renal assessment were not included, which could have provided additional diagnostic accuracy.
7. Sample Size Constraints: - Although the sample size was adequate for statistical analysis, a larger multi-centric sample could provide stronger external validity and more robust subgroup analysis.
8. Selection Bias: - Participants were recruited from hospital settings, which may introduce selection bias, as patients attending healthcare facilities might differ from the general diabetic population.
9. Lack of Stratification Based on Duration and Severity: - Since the study included newly diagnosed patients, variations based on duration of disease, severity of hyperglycemia, and treatment status could not be explored in depth.

Future Scope

a. Longitudinal Studies for Disease Progression: - Future research should focus on longitudinal cohort studies to evaluate the progression of microalbuminuria and PCR over time and their predictive value for chronic kidney disease (CKD) and end-stage renal disease (ESRD).
b. Multi-Centric and Larger Population Studies: - Conducting multi-centric studies across different geographical regions and diverse populations was enhance the generalizability and external validity of findings.
2. Comparative Studies with Advanced Biomarkers: - Future studies should compare microalbuminuria and PCR with newer biomarkers such as cystatin C, NGAL, and KIM-1 to improve early detection of diabetic nephropathy.
3. Integration with Imaging Techniques: - Combining biochemical markers with radiological or imaging modalities (e.g., renal Doppler, MRI-based renal perfusion studies) may provide a more comprehensive assessment of early renal changes.
4. Evaluation of Therapeutic Interventions: - Interventional studies assessing the effect of glycemic control, antihypertensive therapy (e.g., ACE inhibitors/ARBs), and lifestyle modifications on microalbuminuria and PCR levels are warranted.
5. Role of Artificial Intelligence and Predictive Models:-Development of AI-based predictive models using biochemical, clinical, and demographic data can help in early risk stratification and personalized management of diabetic nephropathy.
6. Exploration in Pre-Diabetic and High-Risk Groups: - Extending similar studies to pre-diabetic individuals and high-risk populations (obese, hypertensive, family history of diabetes) may help in even earlier detection and prevention strategies.

Recommendations

1. Routine Screening in Newly Diagnosed T2DM Patients: - Spot urine microalbumin and protein-creatinine ratio should be incorporated into routine screening protocols for all newly diagnosed T2DM patients for early detection of renal involvement.
2. Use of PCR as a Practical Alternative: - Protein-creatinine ratio (PCR) can be recommended as a reliable, cost-

effective, and patient-friendly alternative to 24-hour urine protein estimation in routine clinical practice.

3. Early Intervention Strategies: - Patients with elevated microalbuminuria or PCR should be promptly managed with strict glycemic control, blood pressure regulation, and lifestyle modifications to prevent progression of nephropathy.
4. Public Health Awareness and Screening Programs: - Awareness programs should be implemented to educate patients about early kidney damage in diabetes and the importance of regular urine testing.
5. Integration into National Health Programs: - Screening for microalbuminuria and PCR should be integrated into national programs such as NPCDCS for better early detection and management at the primary healthcare level.
6. Regular Monitoring and Follow-Up: - Periodic monitoring of renal parameters should be recommended to track disease progression and evaluate treatment effectiveness.
7. Capacity Building in Primary Healthcare: - Training healthcare workers and improving laboratory facilities at peripheral centers was help in widespread implementation of these simple diagnostic tools.

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