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Influence of Contrast Media on Renal Function in Patients Undergoing CT Coronary Angiogram

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﴿ فَتَعَالَى اللَّهُ الْمَلِكُ الْحَقُّ ۗ وَلَا تَعْجَلْ بِالْقُرْآنِ مِن قَبْلِ أَن يُقْضَىٰ إِلَيْكَ وَحْيُهُ ۖ وَقُل رَّبِّ زِدْنِي عِلْمًا ﴾

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Certification

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In

Biomedical Sciences

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In view of the available recommendation, I forward this project for debate by the examining committee

…….

DEDICATION

We dedicate this research to our families, whose unwavering support, love, and encouragement have been our guiding light throughout this journey. To our esteemed professors and mentors, your invaluable guidance and dedication to our academic growth have been instrumental in the completion of this work. Lastly, we extend our deepest gratitude to all patients and healthcare professionals who contributed to this study, without whom this research would not have been possible.

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ABSTRACT

Contrast media (CM) are essential for CT coronary angiogram (CTCA), but may affect renal function raising concerns about contrast-induced nephropathy. This study examines changes in serum creatinine, blood urea nitrogen, and glomerular filtration rate 48 hours after contrast exposure. A total of 40 patients (16 females, 24 males) undergoing CTCA were evaluated, and renal function was assessed using Cobas C111 analyzer, with eGFR calculated by the Jaffe method. The post contrast changes were analyzed to determine renal impairment risk. The findings contribute to a better understanding of CM effects on renal function, supporting clinical decision-making for high-risk patients. By stratifying patients based on their underlying comorbidities, the research may help guide safer clinical practices, including the use of preventive strategies for at-risk populations.

KEY WORDS: Contrast Media, Contrast-Induced Nephropathy, Renal Function, Serum Creatinine, Glomerular Filtration Rate, CT Coronary Angiogram

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List of Abbreviations

CTCA ……...	Computed tomography coronary angiogram
CI-AKI …….	Contrast-induced acute kidney injury
CIN ……...	Contrast-induced nephropathy
CKD …….	Chronic kidney disease
CT ……	Computed tomography
eGFR ……	Estimated glomerular filtration rate
ICM ……..	Iodinated contrast media
BBB ……..	Blood brain barrier
RRT ……..	Renal replacement therapy
PC-AKI ……	Post-contrast acute kidney injury
AKI ……...	Acute kidney injury
NSAIDs ……	Non-steroidal anti-inflammatory drugs
CECT ……	Contrast-enhanced computed tomography
CTP ……..	Computed tomography perfusion
CTA ……..	Computed tomography angiogram
LVO ……..	Large vessel occlusion
ADRs ……	Adverse drug reactions
CA-AKI ……	Contrast-associated acute kidney injury
CM ……	Contrast media

ESUR ……	European society of urogenital radiology
ROS ……..	Reactive oxygen species
MRI ……..	Magnetic resonance imaging
NO…….	Nitric oxide
SCr……	Serum Creatinine
CMIN ……...	Contrast media induced nephropathy
ARF ……..	Acute renal failure
HOCM ……	High-osmolar Contrast Media
LOCM ……..	Low-osmolar Contrast Media
EDs ……...	Emergency departments
CV…...……...	Contrast media volume
ICM ……..	Intravascular contrast media
DM ……...	Diabetes mellitus
NAC …….	N-acetylcysteine
GFB …….....	Glomerular filtration barrier
GFR ...……...	Glomerular filtration rate
SRNS ……	Steroid resistant nephrotic syndrome
CHF …….	Chronic heart failure
CC ...……..	Cubic centimeter

CHAPTER ONE

1.0 INTRODUCTION

1.1 Contrast Media

The definition of contrast-induced acute kidney injury (CI-AKI) is an elevation in plasma (P)-creatinine assessed a few days following computed tomography (CT) (1). Around the middle of the 20th century, patients undergoing contrast-added X-ray imaging of their urinary tracts were reported to have experienced the first cases of CI-AKI. As a prime example, several studies have demonstrated that individuals with chronic kidney disease (CKD) are reluctant to have percutaneous revascularization or angiography performed because they fear their renal function would deteriorate (2). Iodine contrast media (ICM) recipients are frequently thought to be at higher risk of developing acute kidney injury. One typical consequence for patients receiving treatment in intensive care units is acute kidney damage. Over 50% of patients in critical care experience kidney injury, which is linked to longer hospital stays, the requirement for renal replacement therapy (RRT), the advancement of chronic kidney disease (CKD), and higher mortality rates. Any AKI occuring 48–72 hours after ICM exposure is referred to as post-contrast acute kidney injury (PC-AKI). The incidence of PC-AKI varies from 7 to 22%, and its effect on critically ill patients is unknown (3).

1.2 Effect of Contrast Media

In earlier times, the injection of contrast media has been linked to serious adverse effects, such as renal failure, dialysis, and death, and has been thought to be the third most prevalent cause of iatrogenic (4). Shortly after injection, contrast media can cause hypersensitivity reactions such erythema and urticaria, which can lead to severe shock. Contrast-induced nephropathy (CIN) frequently has temporary consequences on renal function. Serum creatinine levels, for instance, reach their peak 2-4 days after contrast delivery and return to

baseline levels after 2 weeks. Frequent contrast studies and imaging-based treatments for chronic diseases raise concerns about the use of contrast agents, which are taxing on the kidneys, and other risk factors, like diabetes and the use of non-steroidal anti-inflammatory drugs (NSAIDs), which could accelerate the progression of long-term chronic kidney diseases (CKD) (5). The correlation between the development of chronic kidney disease (CKD) and (CI-AKI) is well established. According to reports, after percutaneous coronary intervention, (CI-AKI) can occur in as many as 55% of patients who had contrast exposure during coronary angiogram, and among high-risk patients, this can lead to a 12.6% requirement for dialysis. Recently, there has been a lot of focus on contrast medium exposure, particularly on high doses, because of possible links to kidney damage (6). The osmotic pressure and viscosity of contrast agents correlate with iodine content, and the quantity used may independently contribute to contrast-induced nephropathy. For medical safety, it is crucial to choose the best contrast agents, iodine concentrations, and least amount of contrast agents (7). Iodinated contrast media ICM is frequently used in computed tomography (CT) to produce contrast-enhanced CT (CECT) scans, such as CT perfusion (CTP) and CT angiograms (CTA). Numerous significant diseases, including as aortic dissection, pulmonary embolism, and large-vessel occlusion (LVO) stroke, can be diagnosed with these scans. Acute myocardial ischemia, stroke, and potentially fatal hemorrhages are among the percutaneous interventions that are guided by ICM. Catheter angiography is another usage for ICM (8). Certain parts of the hypothalamus and the area postrema are examples of brain regions that are not shielded by the blood brain barrier (BBB) and may be more vulnerable to the effects of contrast agents when exposed to blood concentrations greater than normal (9).

1.3 Aim of the Study

This study was planned to evaluate the renal functions decline with iodine -contrast media in the early period in patients scheduled for undergo CT coronary angiogram (CTCA) and determine the risk factors that induce the nephrotoxic effect and evaluate the safety of dose of iohexol use in the diagnostic test within various group of patients.

CHAPTER TWO

2.0 LITERATURE REVIEW

2.1 Definition and Development of Contrast Material Nephropathy

The use of contrast media can increase the precision of illness diagnosis by aiding in the differentiation of various bodily tissues. There are several contrast media for different medical imaging modalities, including X-ray-based imaging, magnetic resonance imaging, and sonography. On the other hand, adverse drug reactions (ADRs) are linked to the delivery of contrast media. Compared to sonography and magnetic resonance imaging, contrast media employed in X-ray-based imaging have a higher frequency of adverse drug reactions (ADRs). There are two types of ICM: water-soluble and oil soluble. The only applications for oil-soluble ethiodized oil (lipiodol) are trans-arterial chemotherapy embolization, lymphangiography, and hysterosalpingography. Water-soluble iodinated contrast agents, however, are also crucial for disease diagnosis and treatment response assessment, particularly in CT imaging of tumors and arteries (10). Knowing who has CA-AKI and who is at risk of getting it is essential to giving each patient the best care possible. Risk variables connected to patients and contrast have an impact on the incidence of CA-AKI (Contrast-Associated Acute Kidney Injury).

The incidence of CA-AKI may be influenced by both the quantitative and qualitative features of CM, one of the contrast-related variables. An increased incidence of CA-AKI has been linked to larger quantities of CM. However, the osmolality and viscosity of CM also affect the risk of CA-AKI; elevated osmolality and viscosity are linked to nephrotoxic potential (11). The use of contrast media in contemporary medical imaging frequently requires doctors and diagnostic radiologists to weigh the possible hazards of administering contrast media against its potential benefits for diagnosis. AKI, also known as contrast-induced nephropathy, develops 48 hours after intravenous contrast media delivery and after other nephrotoxic causes have been ruled out (12). A number of significant articles in recent years have advanced our understanding of CM's renal toxicity and tolerance. To address this,

the European Society of Urogenital Radiology (ESUR) has developed recommendations for the treatment of CM and the avoidance of its adverse effects (13). There are three pathophysiological processes that have been proposed: intrarenal vasoconstriction, direct tubular toxicity, and increased reactive oxygen species (ROS) generation. Direct effects include mitochondrial malfunction, cellular apoptosis or necrosis, and interstitial inflammation brought on by CM with high osmolality directly causing cytotoxicity in nephrons, including renal tubular epithelial cells and endothelial cells. Indirect effects of CM include changes in renal hemodynamics, intrarenal vasoconstriction, and medullary hypoxia. When it comes to ROS creation, CM can either increase oxidative stress and impair renal function by reducing antioxidant enzyme activity or by producing excessive ROS production (14).

2.2 Contrast Media Nephropathy Definition

To help with diagnosis, contrast media (CM) is administered to patients prior to some radiological imaging tests in order to highlight specific features in the image. These include magnetic resonance imaging (MRI) and computed tomography (CT). The administration of CM can be done intravenously, intra-arterially, or orally, depending on the condition being assessed. " While there are other types of CM, gadolinium and iodine-based compounds are the most commonly employed. According to reports, the third most frequent cause of acute renal failure acquired in hospitals is CIN. When CM is administered, renal oxygenation in medullary structures is decreased without a decrease in tubular reabsorption. This is brought on by neurohumoral vasoconstrictive impulses that are initiated by endothelial cells exposed to CM releasing endothelin and inhibiting nitric oxide (NO) (15).

Special CM, which are typically given intravenously to patients, draw attention to pathological alterations in organs and tissues in pictures and provide experts with information to help them formulate strategies for implementing a certain treatment. The primary side effect of CM injection is acute renal failure, commonly known as "contrast-induced acute kidney injury" (CI-AKI) or "contrast-induced nephropathy" (CIN), which is still one of the most dangerous and occasionally lethal adverse effects. Additionally, this

leads to a rise in the negative reactions that are linked to it. Renal hemodynamics, oxidative stress, apoptosis, immunological/inflammatory response, and epigenetic regulation are all directly impacted by the cytotoxic actions of all CM on renal tubular epithelial and vascular endothelial cells (16).

According to earlier studies, patients having coronary angiography had a CIN risk of more than 3%, which may have been brought on by intra-arterial CIN usage and the patients' comorbidities (17). Ironic iodinated contrast media (ICM), the initial generation of ICM, consists of a single benzene ring monomer with three iodine atoms and a carboxylic acid (–COOH) group on the side chain. Non-ionic ICM, the second generation of ICM, likewise includes a single benzene ring monomer with a variety of side chains that have polar alcohol (-OH) groups but no -COOH groups. Its non-ionic properties cause the osmolality to drop to two to three times that of normal serum.

The use of contrast agents has significantly improved medical imaging sensitivity; but, due to their inherent toxicity, they can also cause a number of negative side effects, such as contrast-induced nephropathy (CIN). When serum creatinine (SCr) levels rise by more than 44.2 μmol/l (0.5 mg/dl), or 25%, relative to the baseline SCr level, 48 hours following iodine-based contrast exposure, it is referred to as CIN, a pathological condition (18).

2.3 Pathophysiology of Contrast Media Nephropathy

Before some radiological imaging tests, patients are given contrast medium (CM), which highlights specific characteristics in the image to help with diagnosis. Among these are its applications in magnetic resonance imaging (MRI) and computed tomography (CT). Oral, intravenous, or intra-arterially delivered CM is contingent upon the type of ailment being assessed. For instance, intravenous iodine is utilised for a CT angiography, while oral delivery is more common in a barium swallow examination. Iodine-based and gadolinium, a heavy metal CM utilised in MRI, are the most commonly used agents, while there are other types of CM as well. Contrast-induced nephropathy (CIN), a significant new-onset consequence or aggravation of renal impairment following administration of CM, is a problem linked to CM use. CIN is generally understood to be a reduction in renal function

that happens within a short period of time following the administration of iodinated contrast media (15). It is linked to longer hospital stays, higher rates of morbidity and mortality, and is the third most frequent cause of hospital-acquired acute kidney damage (AKI) (19). Nosologically, CMIN usually manifests as an ARF 24–72 hours following the ICM injection (19). Regrettably, a large number of patients in this typically elderly group suffer from comorbid conditions such diabetes, anemia, cardiovascular disease, and chronic kidney disease, which lower renal vasodilatory capacity (also known as functional renal reserve) (20). Although the fundamental processes of contrast-induced nephropathy, a well-known cause of acute renal failure, are still unclear (21).

The pathophysiology of CI_AKI involves a number of mechanisms, including altered microcirculation, direct toxic effects of radiocontrast media on renal cells linked to renal endothelial dysfunction, and hemodynamic abnormalities of renal blood flow that result in hypoxia in the renal medulla (22) Specifically, with regard to osmolality, prior research has demonstrated that high-osmolar CM (HOCM) is linked to a higher risk of CI-AKI than low-osmolar CM (LOCM) (23,24) High osmolality CM can directly destroy renal tubular epithelial cells and endothelial cells in nephrons, resulting in interstitial inflammation, cellular apoptosis or necrosis, and mitochondrial dysfunction (14). Through indirect effects, CM can significantly alter hemodynamics, resulting in intrarenal vasoconstriction and medullary hypoxia (25). According to reports, CM increases the generation of oxygen free radicals via boosting lipid peroxidation and decreasing the biological activity of renal tubular epithelial cells. Additionally, it decreases the activity of superoxide dismutase and antioxidant enzymes, which raises the generation of reactive oxygen species (ROS) (26).

However, there is currently little evidence linking the slight elevations in serum creatinine that are typical of contrast-associated AKI to significant negative consequences (27) CIN is diagnosed when serum creatinine (SCr) levels increase by ≥ 0.5 mg/dL or $\geq 25\%$ from baseline within 72 h after contrast radiography using iodinated contrast media (28).

2.4 Epidemiology of Contrast Media Nephropathy

One possible side effect of intravascular iodinated contrast exposure is AKI. Serious side effects, such as progressive renal disease and mortality, are linked to contrast-associated AKI, which usually presents as mild and temporary declines in kidney function that appear a few days after contrast administration (31) A decline in renal function that occurs within a few days after intravascular administration of contrast medium (CM) is known as contrast-induced acute kidney damage (CI-AKI). Clinical professionals usually postpone imaging procedures when they believe that the hazards to the kidneys from CM outweigh the advantages of improved imaging. Scholars have discovered, however, that contrast medium and non-contrast medium variables (including anaemia and haemodynamic instability) are responsible for the decline in renal function following CM, thanks to a thorough understanding of AKI and contrast medium.

Therefore, in the past, the amount of acute kidney damage brought on by CM has been overstated. When referring to AKI following intravascular administration of contrast medium, the term "contrast-associated acute kidney injury (CA-AKI)" has become more popular than "CI-AKI." (30). Iodine contrast medium is now widely employed in clinical diagnostic procedures like CT, which is frequently used for CT angiography and CT perfusion, thanks to the ongoing advancements in imaging technology. Additionally, because they may enhance the physicochemical characteristics of common iodine contrast media, increase the detection rate of lesion locations, and assist lesion localization, qualitative, and differential diagnosis, they are useful for imaging of the body cavities, joints, and spinal cord. (29) One of the traditional causes of acute Kidney infection (AKI) is the administration of iodine-based contrast medium (CM). Therefore, in patients with kidney illness, iodinated CM is often limited or not conducted, which results in decreased imaging procedure precision (13).

In order to increase picture quality and diagnostic precision, intravenous iodinated contrast media is commonly used in emergency departments (EDs). Administration of contrast media has long been thought to be the third most frequent cause of iatrogenic acute kidney injury (AKI) and a risk factor for serious side effects, such as mortality, renal failure, and dialysis. (30) CIN risk factors include chronic kidney disease (CKD), diabetes mellitus,

hemodynamic instability, gender, and age, but are not normally changeable. Recently, the importance of modifiable influencing factors of CIN, particularly the appropriate level of contrast medium volume (CV), has been progressively recognized to minimize the nephrotoxicity. Contrast medium is primarily eliminated by the kidneys based on pharmacokinetics. (31) The diagnosis of CIN is based on serum creatinine (SCr) levels, which rise within 24-48 hours and peak three to five days following CM exposure. Renal injury can occur immediately after consuming CM, and SCr increases when more than half of the nephrons are injured. The rise of the SCr level also depends on several characteristics such as age, gender, weight, muscle mass, medicine usage, and hydration status (39). In their first survey-based guideline, the contrast media safety committee (CMSC) of the ESUR defined Contrast-Induced Nephropathy (CIN) as "a condition in which an impairment in renal function (an increase in SCr by more than 25% or 44 umol/L, or 0.5 mg/dl) occurs within 3 days following the intravascular administration of a contrast medium in the absence of an alternative aetiology" (32).

In particular, renal tubular epithelial cells (vacuolization of tubular cells and osmotic nephrosis and endothelial cells are directly harmed by the iodine present in ICM. This cytotoxicity's precise pathophysiological mechanism is still unknown (33).

2.5 Risk Factor for the Development of Contrast Media Nephropathy

The awareness of contrast-associated acute kidney damage (CA-AKI) has increased as a result of the growing clinical usage of medical imaging for diagnosis and therapy. CA-AKI is interchangeable with post-contrast acute kidney injury (PC-AKI), or post-contrast acute kidney damage (34). When contrast media is administered intravasculary for intravascular diagnostic procedures or therapeutic angiographic interventions, an iatrogenic acute kidney injury known as contrast-induced nephropathy (CIN) or contrast-induced acute kidney injury (CI-AKI) occurs.

The prevalence of CIN increased with age in high-risk patients, such as those with multiple myeloma, diabetes mellitus with impaired renal function, congestive heart failure, intra-arterial intervention, higher volume of contrast, volume depletion, hypertension, and

hyperuricemia (14). Reports indicate that older persons are more likely to have CIN. The higher incidence of CIN in the elderly is believed to be caused by a number of factors, such as tubular function, decreased glomerular filtrate, increased contrast because of more difficult arterial access, mult-ivessel diseases, and comorbidity. Previous studies have shown that CIN is common in persons over 70(35). Numerous forms of diabetes mellitus (DM) and its acute or long-term sequelae have emerged as prevalent illnesses endangering our lives and well-being; these conditions can be thought of as the root cause of secondary disorders. DM is a possible predisposing factor for AKI even if several recent reviews and research do not highlight it as a direct risk factor of CI-AKI. Regarding diabetes According to data, the incidence of CI-AKI ranges from 5.7% to 29.4% in individuals with diabetes and roughly 13% in those without the disease (36).

Patients with diabetes have emerged as a significant high-risk population for CIN due to the correlation between diabetes and coronary heart disease (37). More recent research does not support the long-held belief that multiple myeloma is a risk factor for CA-AKI. More recent research does not support the long-held belief that multiple myeloma is a risk factor for CA-AKI (38). Less than 3% of patients present before the age of 40, and the median age for Multiple myeloma diagnosis is 65 (MM) which is typically a disease that affects the elderly (39). In patients with normal renal function, the contrast media has been observed to be excreted by the urine tract 50% of the time within two hours of intravascular delivery. Patients with impaired renal function will have longer excretion times. The contrast media penetrate the aquatic environment and eventually the drinking water supply system as a result of excretion through the urine (40).

2.6 Prevention of Contrast Media Nephropathy

IV crystalloid hydration using normal saline, sodium bicarbonate, or N-acetylcysteine (NAC) are among the modalities that have been investigated for the prevention of CIN (41). One of the primary preventative measures for CIN has been identified as intravenous fluid hydration with normal saline, a low-risk and inexpensive fluid treatment. Hydration enhances diuresis, dilutes the total intravascular contrast burden, and increases intravascular

volume (15). Patients who received hydration with either 0.45% saline or 0.45% saline + loop diuretics in research had a considerably higher incidence of CIN than those who received saline alone (32). Also, Vitamin C, commonly referred to as ascorbic acid, shown some potential in avoiding contrast-induced nephropathy.

According to studies, ascorbic acid's antioxidant qualities may aid in shielding the kidneys from the oxidative stress brought on by contrast media (42). Acetylcysteine may have an indirect antioxidant effect by promoting glutathione production. By stabilizing nitric oxide and influencing renal cortex and medulla microcirculation either directly or indirectly, acetylcysteine can also lessen renal vasoconstriction brought on by contrast medium stimulation (43). A high iodine concentration contrast agent, like the majority of low-osmolar contrast media (LOCM), was thought to produce higher attenuation and greater image quality. However, these contrast agents' high osmolarity dilutes the contrast material and causes water to be absorbed from the extravascular space, reducing the total vascular attenuation (44).

2.7 Evaluation of Glomerular Functions

Over 10% of people suffer from abnormal kidney function, which raises the risk of morbidity and death. Immunosuppressive drugs, antihypertensives, and diuretics are currently used to treat kidney disorders, with varying degrees of efficacy. The glomerular filtration barrier (GFB) breaking down is a hallmark of most renal diseases. Proteinuria, a protein leak from plasma into the urine, is a sign of the glomerular filtration barrier (GFB) breaking down. It is linked to an overall decrease in normal kidney function and is a significant risk factor for the disease's development to organ failure1. Clinically, individuals may exhibit isolated proteinuria, proteinuria with hypertension, or nephrotic syndrome, a high-grade form of proteinuria with puffiness. Blood tests can also reveal abnormal kidney function.

Clinical evaluation is used to treat childhood nephrotic syndrome, which is classified as either steroid-resistant nephrotic syndrome (SRNS) or sensitive to steroid medication (45). Patients with CKD Stages 4 and 5 frequently have multimorbidity, which can make

evaluating them for future kidney replacement therapy more difficult, especially in older patients. In patients with chronic heart failure (CHF), for instance, the ejection fraction is significantly diminished, and it has been discovered that eGFR cannot be accurately predicted using creatinine-based approaches alone (46). Despite being educated to assess the anatomy and physiology of the heart; cardiologists pay less attention to the assessment of the kidney.

However, changes in kidney function brought on by the disease, medication, and hemodynamics are what define heart failure syndrome. Renal damage may be indicated by some of these changes, while others may just reflect a normal renal response. Inappropriate stopping of disease-modifying heart failure treatments, early stopping of decongestive treatments, or persistent renal damage could arise from misinterpreting these changes. An overview of kidney function assessment and interpretation across the heart failure trajectory is given in this position paper (47). Improvements in the average life expectancy of people around the world over the past few decades have been linked to a notable rise in the percentage of the old population, concurrent with an increase in the prevalence of non-communicable diseases like diabetes and hypertension. Given that the kidney is frequently the target organ of numerous illnesses, it is very important to assess renal function in this population.

Additionally, it is well known that the kidneys experience aging-related changes that are manifested by a decrease in glomerular filtration rate (GFR), which reveals a loss of kidney function brought on either by the length of exposure to diseases that can lead to kidney damage or by a natural senescence process linked to healthy aging. It is especially crucial to accurately test renal function in older people to figure out the extent of kidney function loss and facilitate personalized therapeutic interventions (48).

2.8 Measurement of Serum Creatinine and Glomerular Filtration

A nitrogenous amine called creatine is essential for bioenergetic function in tissues with high metabolic demands. Skeletal muscles store and transform more than 90% of the total creatinine into creatinine (49) And Patients with less muscle mass will have lower serum

creatinine levels as muscle mass impacts serum creatinine concentration (50) also Serum creatinine levels are thought to be an important indicator of renal bio-function, the typical range for creatinine in the blood is 0.84-1.21mg/dL Creatinine is a harmless chemical with no substantial role in bio metabolism, and its concentration is controlled by renal excretion .The amount of creatinine in serum and urine is proportional to muscle mass and renal elimination, and it is rather steady in serum(51).

GFR serves as a comprehensive evaluation of the kidney's many vitally important activities. Chronic kidney disease (CKD) is diagnosed, staged, and managed using GFR. It is also used to calculate drug dosages and the prognosis for events and death due to CKD. GFR, which can be measured by serum or clearance levels, is the rate at which the glomerulus filters plasma to create an ultrafiltrate (52). Also, the accurate assessment of the glomerular filtration rate (GFR) is critical for the early detection of renal disease. In clinical practice, GFR is often estimated (eGFR) using blood concentrations of endogenous filtration indicators (53). Glomerular filtration rate is the best global indicator of kidney function in both health and disease.

It represents the kidney's excretory capacity, correlates directly with kidney functioning mass, and can be used to classify CKD into stages based on progression risk and drug dosing (55). The glomerular filtration rate (GFR) was first used to assess glomerular function by estimating the amount of fluid filtered through the renal glomeruli per unit of time. It is not a measure of single-nephron glomerular function, but rather a metric reflecting the sum of the filtration of all glomerular capillaries in the human kidney (56).

CHAPTER THREE

3.0 MATERIALS AND METHODS

3.1 Study Design

This study was designed to evaluate the efficacy of contrast media on renal functions in patients undergoing CT coronary angiogram. For this purpose, a total of 40 patients scheduled for elective CT coronary angiogram at the Cardiac Center were included in the study. The protocol of the study was approved by the general directorate of health/Erbil. The inclusion and exclusion criteria are listed in **Table 3.1**.

Table 3. 1 The criteria for excluding and including patients.

Criteria for inclusion	Criteria for exclusion
1. Giving consent to work 2. Elective coronary angiogram planned 3. Patients aged 20-90	1. Chronic renal failure 2. Serum creatinine >2mg/dl 3. Chronic liver disease and failure 4. Acute coronary syndrome 5. Stage 3-4 decompensated 6. Contrast exposure 3 months before procedure 7. Active infection 8. Systemic inflammatory disease 9. Malignancies 10. Presence of a single kidney 11. Presence of hypothyroidism or hyperthyroidism 12. Antibiotic use 13. Steroid use

3.2 Materials

The materials and machines which were used in this research are listed in **Table 3.2.**

Table 3. 2 The machines and materials used in the study.

Devices and Materials	Manufacturer	Country
Contrast Agent (Iohexol (omnipaque 350 mg iodine/mL)	Ge Health Care	United States
Cobas C111 (Photometric Analyzer)	Roche Diagnostics	Germany
Philips ICT 256-slice CT Scanner	Radiology oncology systems	United States (California)
Automated Injector for CM	Soma Technology	United States
Centrifuge (80-1 electronic centrifuge)	Gemmy industrial corp	Taiwan
Hitachi cup	Ningbo Yingmed medical	China
Gel Tube	Razimed	Iraq
Syringe	Genject	Turkey
Alcohol	Al-Joud	Iraq
Cotton	Laura	Iran

3.3 Sample Collection

A total of 40 patients undergoing CT coronary angiogram were included in this study. Out of the 40 patients 24 were male and 16 were female. A blood sample was obtained in a gel tube prior to contrast administration to measure GFR and serum creatinine. A second blood sample was collected 48 hours after contrast administration to evaluate any changes in renal function parameters. The tests that were done after contrast exposure were (serum creatinine, GFR, and BUN). Standardized protocols were followed for sample collection, handling, and analysis to ensure data accuracy and reliability.

3.4 Methods

3.4.1 Contrast Media

Iohexol, a non-ionic, low-osmosis, iodinated contrast agent, was used in all patients undergoing coronary angiograms. The angiogram technician recorded the amount of contrast material in cubic centimeters (cc).

Before a CT scan, the doctor evaluates the patient's symptoms and family history to determine if cardiovascular disease is present. Initial tests include serum creatinine, treadmill stress tests, and electrocardiograms (ECG). Based on the results, patients with mild cases undergo a CT scan, while severe cases are referred for catheterization.

Once the patient is ready, they lie on a narrow table that slides into the scanner tunnel. Four ECG leads are placed (two on the shoulders, two on the ankles), and the contrast media line is connected to a cannula. The contrast was administered using an injector, which delivered both contrast media and normal saline. The contrast agent (omnipaque 350 Mg Iodine/ MI intravenous solution) was administered before the CT scan to enhance image quality.

3.4.2 Evaluation of Renal Function Before and After the Procedure

Before the procedure, blood samples were tested to measure baseline renal markers, including serum creatinine and GFR. After the patient was exposed to contrast media, they were asked to return for follow-up testing 48 hours after the procedure. At this follow-up, the same renal markers were reassessed, along with blood urea nitrogen (BUN), to detect any changes in kidney function. Serum creatinine and BUN were measured using the Cobas 111 (Roche company), which is an automated clinical chemistry analyzer that operates as a photometer to measure light absorption. GFR was calculated using GFR calculation analyzer based on the Jaffe method, which estimates kidney function based on the patient's age and pre-procedure serum creatinine levels.

3.6 Statistical Analysis

Statistical analysis was performed using GraphPad (version 8) and R programming (R 4.3.1). Descriptive statistics were used to summarize the patient's demographic and baseline renal function markers. Paired t-tests or Wilcoxon signed-rank tests were applied to compare pre- and post-contrast renal function values, depending on data normality. Correlation analysis was conducted to assess relationships between contrast volume and renal function changes. A p-value of (<0.05) was considered statistically significant. Graphical representations and data visualizations were generated using GraphPad to illustrate findings.

Additionally, a Chi-square test was employed to evaluate the distribution of patients across the diagnostic classification groups (mild, moderate, and severe) based on post-procedure renal function changes.

CHAPTER FOUR

4.0 RESULTS

4.1 Study Population Characteristics

The research population consisted of 40 individuals, including 16 females (40%) and 24 males (60%), as shown in **Figure 4.1**. The participants' ages varied from 20 to 90 years, as observed in **Figure 4.2**. Addressing comorbidities, 11 patients were diagnosed with hypertension, 1 patient had diabetes, 2 patient exhibited hypercholesterolemia, 1 patient had asthma, and 1 patient had cardiovascular disease while 8 patients showed more than on comorbidity. The remaining subjects reported no known comorbidities, as shown in **Figure 4.3**.

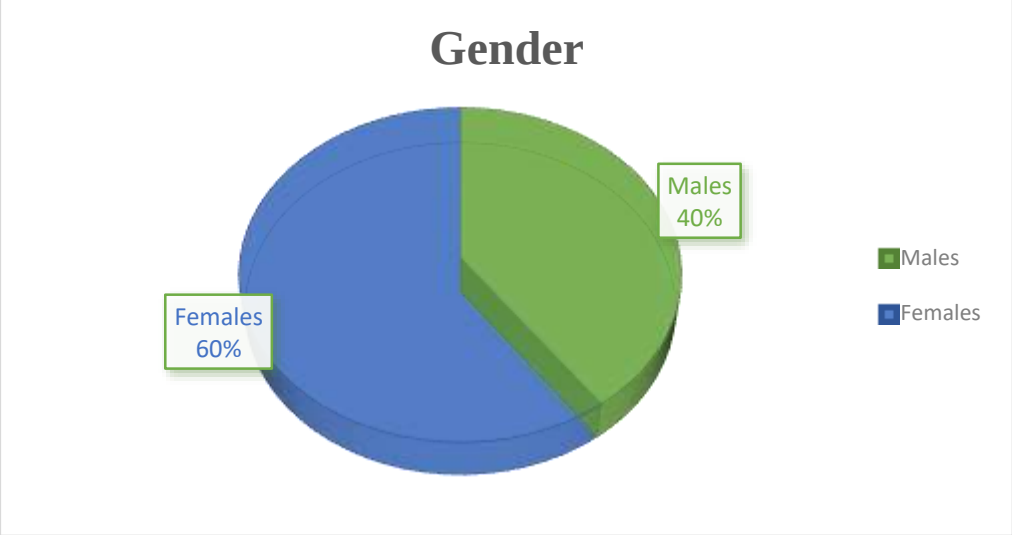

Figure 4.1 Proportion of research participants by gender. The graphic indicates the distribution of male and female participants in the research, while male constituting 60 % and female 40% of the overall population.

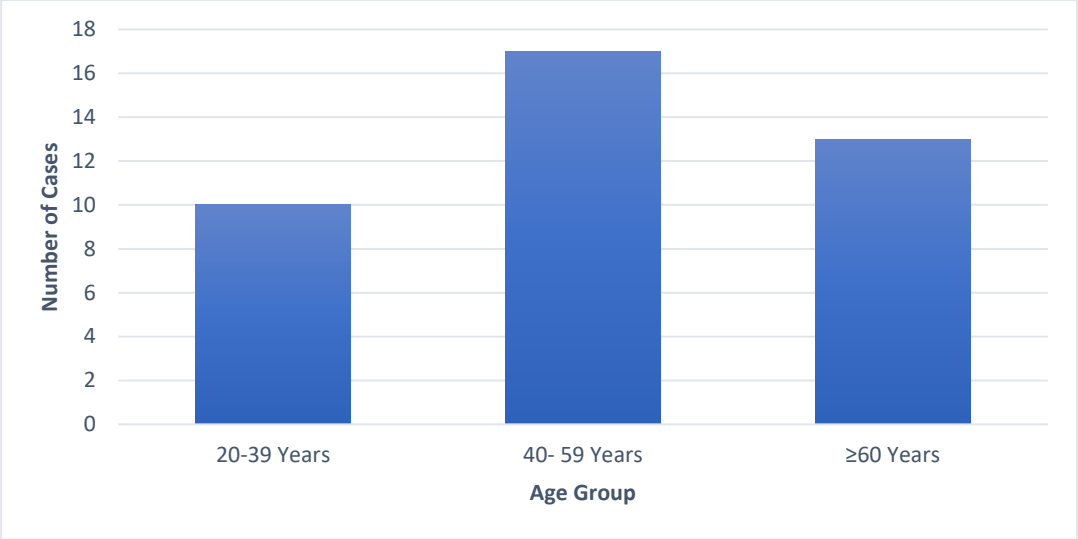

Figure 4.2 Demographic distribution of the study population bye age. This figure categorizes participants into three age groups, indicating that the 40-59-year age group was the most prevalent, with 17 cases. This was followed by the ≥60-year age group, which included 13 cases, and the 20-39 year, which had 10 cases.

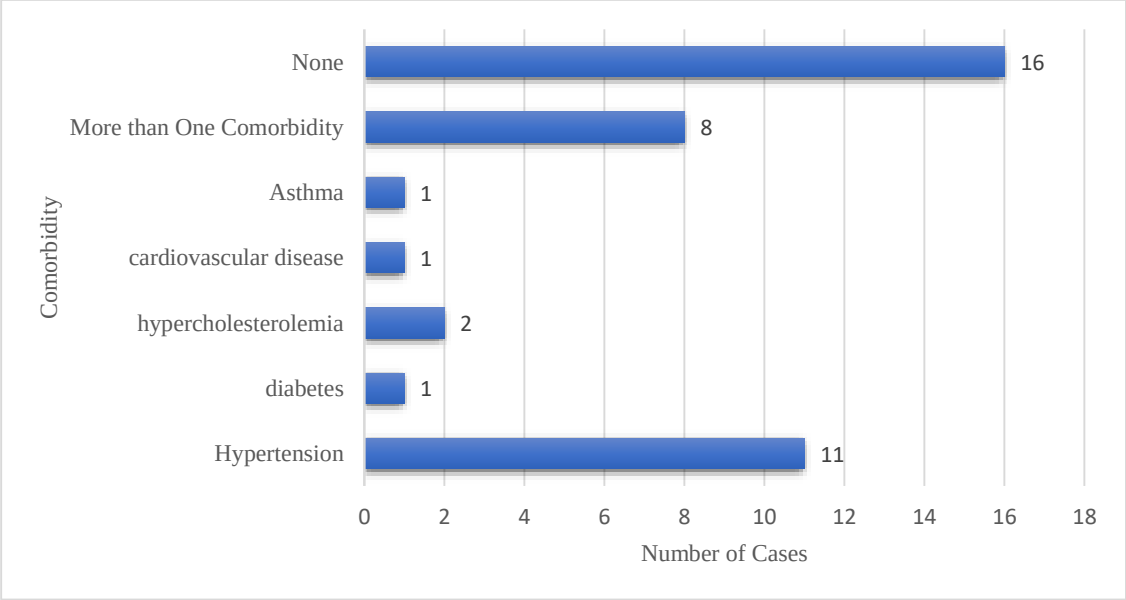

Figure 4.3 Prevalence of comorbidities within the research group. This figure shows the prevalence of many comorbid diseases, with hypertension being the most prevalent, identified in 11 cases. Furthermore 1 patient had asthma, and 1 patient suffered from cardiovascular disease.

Notably 8 patients presented with multiple comorbidities, while the remaining participants reported no known underlying health conditions.

4.2 GFR Box Plot

The comparison of glomerular filtration rate (Before), and (After) the administration of contrast media is shown in **Figure 4.4.** The results were analyzed using the Wilcoxon test. The result indicates no significant difference between the two measures, suggesting that contrast media did not substantially affect GFR in this investigation.

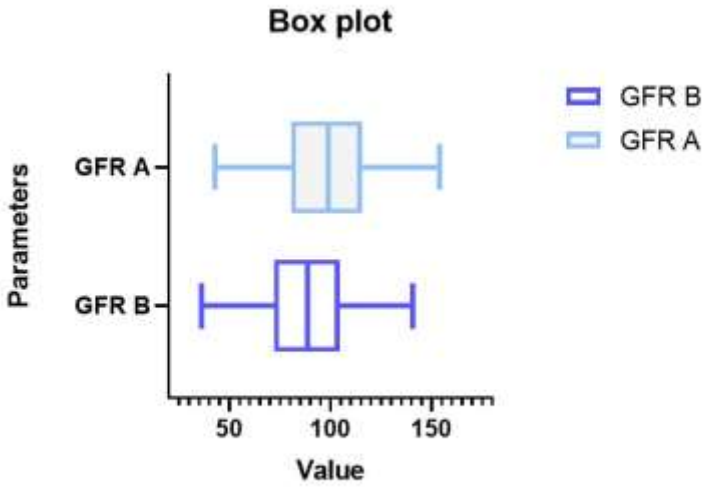

Figure 4. 4 Shows a box plot for comparing the difference between the results of (GFR) before and after exposure to contrast media.

4.3 Serum Creatinine Bar Plot

The comparison of serum creatinine (Before) and (After) the administration of contrast media is shown in **Figure 4.5.** The results were analyzed using a paired t-test. The study reveals no significant difference between two measurements, indicating that contrast exposure did not significantly impact blood creatinine levels. This data indicates that serum creatinine levels were relatively stable after contrast injection.

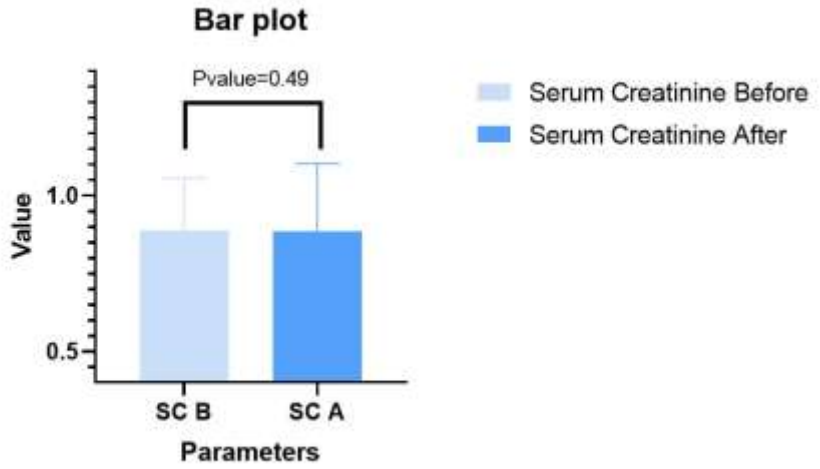

Figure 4.5 Shows a comparison of the level of serum creatinine before and after contrast exposure. The results show that there is no significant difference between serum creatinine levels before and after contrast exposure.

4.4 Relationship Between Contrast Dose and Renal Function Markers

Scatter plots were generated to assess the relationships between contrast dose and BUN, GFR, and serum creatinine.

4.4.1 Contrast Dose and BUN

The analysis of BUN in relation to contrast dose is shown in **Figure 4.6**. The findings indicate a positive correlation between BUN levels and contrast dosage, indicating a potential link. The data had a normal distribution, as confirmed by statistical analysis. However, despite the identified trend, the association failed to achieve statistical significance, suggesting that the impact of contrast dosage on BUN levels may lack sufficient strength or consistency to support definitive conclusions.

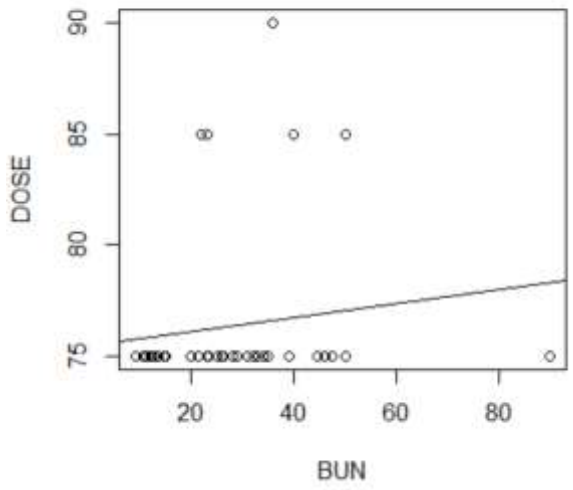

Figure 4.6 Shows a positive association was observed between BUN and contrast dose. Its showing that higher BUN values may be associated with increased contrast doses. As the dose increases, the BUN value also increases.

4.4.2 Contrast Dose and GFR

The analysis between dose of contrast media and GFR are shown in **Figure 4.7**. The results indicate a negative correlation, suggesting that an increase in contrast dosage is associated with a reduction in GFR. Moreover, the data demonstrated a normal distribution, as confirmed by statistical analysis. In contrast to the prior analysis, this correlation was statistically significant, indicating a meaningful effect on contrast media dose on renal function as assessed by GFR.

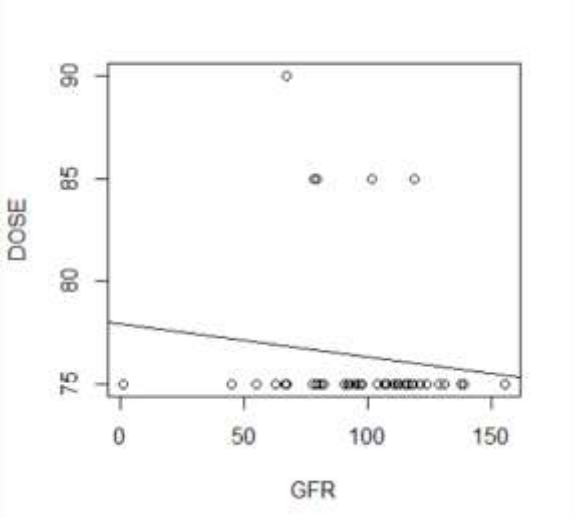

Figure 4.7 Shows the result of the relationship between glomerular filtration rate (GFR) and dose. The plot and line show the negative associations between dose and GFR; when the values of the GFR increase, the value of the dose will be decreased.

4.4.3 Contrast Dose and Serum Creatinine

The analysis between dose of contrast media and serum creatinine are shown in **Figure 4.8**. The data demonstrates a negative correlation. Indicating that increased contrast dosages are associated with decreased serum creatinine levels. The data had a normal distribution, as verified by statistical analysis. However, despite this observed tendency, the relationship lacked statistical significance, suggesting that the impact of contrast media dosage on serum creatinine levels be minimal or inconsistent.

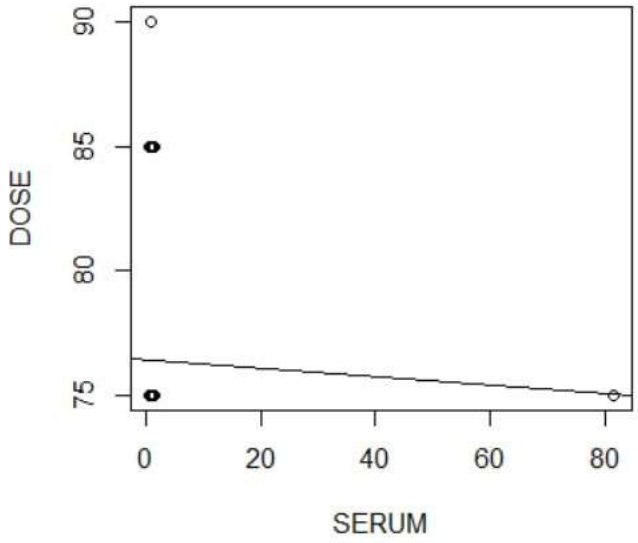

Figure 4.8 Shows that there is a negative correlation between the dose of contrast media and the serum creatinine level; when the dose decreases the serum creatinine level increases.

CHAPTER FIVE

5.0 DISCUSSION

Focus in this study was on the correlation between contrast dose and renal function markers: blood urea nitrogen (BUN), glomerular filtration rate (GFR), and serum creatinine. In Chapter 4, scatter plots and statistical analyses were performed to assess the correlation between these factors. Results indicated that while BUN and serum creatinine had a weak or non-significant association with contrast dose, GFR had a statistically significant negative correlation with contrast dose. When comparing the GFR and serum creatinine before and after contrast administration, no significant difference was found, indicating minimal renal function impairment for this cohort after contrast administration. The results would be beneficial for clinicians in the application of these markers to monitor patients undergoing contrast-enhanced imaging.

BUN and contrast volumes show weak, inconclusive associations. In the analysis of BUN levels and contrast dose, the positive correlation indicates higher doses of contrast media may increase BUN levels; however, it was not statistically significant. This suggests that any such relationship may not be strong or consistent enough to draw any conclusions. Historically, BUN has been a marker of kidney function; however, it is increasingly becoming clear that BUN can be influenced by many non-renal factors: dehydration, dietary protein intake, and liver function (57). For example, BUN is something that responds greatly to any change in the state of hydration (58), and thus should not be the only sole measure to be relied upon when assessing CIN due to contrast media exposure. Moreover, studies have contested the usefulness of BUN as a predictor of CIN, especially since BUN may remain normal despite notable derangement in renal function (59). For instance, it has been reproached that isolated use of BUN for acute kidney injury diagnosis characteristically fails to detect early renal damage (60) The findings from this study indicated a trend that appeared to suggest a possible relationship between BUN and contrast dose, but the lack of statistical significance served to highlight the limitations of that relationship wherein BUN is concerned. More reliable markers like GFR, therefore, should be the next focus to evaluate renal function after contrast exposure. GFR and Contrast Dose: A Clinically Relevant and Strong Correlation GFR indicates statistically significant negative correlation with contrast dose; that is,

with increase in the dose of contrast media, GFR showed a tendency of decline. This is in agreement with the well-known detrimental effects of contrast media on renal functions especially in subjects having renal compromise (58). The GFR is accepted to be the best measure of kidney function since it directly measures the kidney's ability to filter waste products from the blood. Unlike BUN and serum creatinine, which are influenced by extra-renal factors, the GFR indicates with confidence and specificity the health of the kidney function (59).

The observed statistically significant negative correlation between contrast dose and GFR finds concordance with several studies that document a decline in GFR post-contrast media administration. (57) contend that contrast-induced nephropathy is presaged by declines in GFR, which now becomes a reliable and clinically relevant index of renal function in the patient receiving contrast. In addition, it has the advantage of being less influenced by external factors such as hydration status or dietary intake, which enhances its utility as a reliable aid for clinicians when assessing renal function in the situation of contrast-enhanced imaging (58). GFR finds extra merit in revealing slight and minute alterations in the kidney since GFR could decrease even before serum creatinine or BUN rise perceptibly (60) Thus, GFR becomes relevant for the early detection of CIN and the basis of the findings in this study translated into clinical practice. With that, the statistically significant relationship found between contrast dose and GFR in this study substantiates the claim that GFR ought to be the principal marker for assessing renal function in patients undergoing contrast-enhanced imaging examinations.

Serum creatinine and Contrast dose: Irreconcilable and inaccurate markers Another point of interest is the relationship between contrast dose and serum creatinine levels. The data presented a negative correlation between contrast dose and serum creatinine levels, but the relationship was not statistically significant; in fact, these findings suggest that serum creatinine would not be a reliable marker for correlating the effect of contrast media on renal function. Serum creatinine has always been with the evaluation of kidney function; however, it has been well established that it has several drawbacks in recognizing early renal impairment. One of the main problems with serum creatinine is that it does not increase substantially until the kidney is already affected and damage is well-established, usually 24–48 hours' post-injury (57).

Factors such as muscle mass, age, and gender can also contribute variability in serum creatinine levels and detract from its sensitivity to subtle change in renal function (61) This renders serum creatinine an insensitive marker for detecting contrast-induced nephropathy, especially in cases of

normal baseline renal function or low muscle mass (59). In the present study, the lack of a statistically significant relationship between contrast dose and serum creatinine further supports the growing evidence that serum creatinine alone may not fulfill the criteria for diagnosing CIN or assessing renal function in patients undergoing contrast imaging. No Significant Difference in GFR Pre and Post-Contrast

Another important factor of this study was to evaluate GFR measurements before and after contrast administration. Results yield no significant difference between both measurements and indicate that contrast exposure did not cause immediate or major effect on the renal function of the subjects. This is important because although contrasts can affect renal function, differences may not be established in all patients. The variation with regard to changes in renal function due to contrast can include baseline renal function and contrast volume and risk factors in the patient (58). Moreover, several studies have indicated that the risk for nephropathy induced by contrast is generally exaggerated, especially in patients with normal baseline renal function (60) Thus, this study proves that there was no significant difference between GFR measurements before and after administration of contrast, consistent with the above studies suggesting that such exposure was not associated with nephrotoxicity in most patients. As a conclusion to this present study, it seems that the relationship between contrast media dose and renal function markers is characterized by variability. While GFR was shown to correlate significantly to contrast dose negatively, BUN and serum creatinine had lesser correlations and were not significant. This makes GFR a more dependable and precise method of assessing renal function in patients receiving contrast-enhanced imaging studies. Because BUN and serum creatinine are unreliable in the early detection of kidney damage, GFR should be focused on in clinical practice and intervention for the early detection of contrast-induced nephropathy (62).

CONCLUSIONS

This study evaluated the impact of contrast media on renal function in patients undergoing CT coronary angiogram. The findings suggest that contrast media may have statistically significant negative correlation with GFR, but show no significant effect on serum creatinine or BUN levels. These results highlight the importance of GFR as a more reliable marker for detecting contrast-induced nephropathy compared to serum creatinine or BUN. Although contrast media exposure did not result in significant renal impairment in the studied cohort, further research with larger sample sizes and longer follow-up periods is recommended. Future studies should also explore alternative imaging methods or contrast agents to mitigate renal risks, particularly in high-risk populations.

RECOMMENDATIONS

Based on our study on the influence of contrast media on renal function in patients undergoing CT angiogram, we recommend the following point:

- Larger, longer-term studies with an expanded sample size to improve statistical power and generalizability.
- Data should be collected from multiple centers across Kurdistan region to ensure a more representative sample, including patients from various cities, provinces, hospitals, and healthcare facilities.
- Follow-up assessments at 48 hours and 168 hours (7days) post-contrast exposure are crucial to detect delayed nephrotoxicity.
- Further research should evaluate the effectiveness of pre and post procedural hydration strategies in preventing contrast-induced nephropathy, especially in high-risk patients such as those with diabetes, hypertension, or pre-existing renal impairment.
- Exploring novel biomarkers like Cystatin C may provide earlier and more sensitive detection of contrast- induced nephropathy, enabling targeted preventive measures.
- Further studies should also investigate low-contrast or contrast-free imaging technique, such as MRI, ultrasound, or low-dose contrast protocols, to offer safer diagnostic options for high-risk patients.

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الخلاصة:

تعد مواد التباين ضرورية لتصوير الشرايين التاجية بالتصويرالمقطعي المحسوب ولكنها قد تؤثر على وظائف الكلى مما يثير المخاوف بشأن اعتلال الكلى الناجم عن التباين. تفحص هذه الدراسة التغيرات في الكيراتينين في مصل الدم، ونتروجين اليوريا في الدم، ومعدل الترشيح الكبيبي بعد 48 ساعة من التعرض للتباين. تم تقييم ما مجموعه 40 مريضا (16 انثى -24 ذكرا)خضعوا لتصوير الشرايين التاجيية بالتصوير المقطعي المحسوب مع حساب معدل الترشيح الكبيبي المقدر بطريقة (جاف) وتم تقييم وظائف الكلى باستخدام جهاز تحليل(كوباس س-111) تم تحليل التغيرات التي حدثت بعد التباين لتحديد مخاطر التأثير عل وظائف الكلى. تساهم النتائج في فهم افضل لتأثيرات مواد التباين على وظائف الكلى ودعم اتخاذ القرارات السريرية للمرضى المعرضيين للخطر. من خلال تصنيف المرضى بناء على الامراض المزمنة الاساسية لديهم. قد يساعد البحث في توجيه الممارسات السريرية الاكثر امنا، بما في ذلك استخدام استراتجيات وقائية للمرضى المعرضين للخطر.

پوختە:

کۆنتراست میدیا گرینگە بۆ ئەنجیۆگرامی سی تی ((CTCA)، بەڵام لەوانەیە کاریگەری لەسەر کارکردنی گورچیلە هەبێت. ئەم تۆژینەوەیە گۆڕانکاریەکانی کریاتینینی سیرۆم، نایترۆجینی یوریا لە خوێن و ڕێژەی پاڵاوتنی گلۆمێرولار دوای ٤٨ کاتژمێر دوای بەرکەوتنی کۆنتراست دەکۆڵێتەوە. کۆی نەخۆش (١٦ مێ،٢٤ نێر) هەڵسەنگاندنیان بو کرا بە بەکارهێنانی ئامێری (کۆباس س١١١) لەگەڵ هەژمارکراوی شێوازی (جاف). گۆڕانکاریەکانی دوای کۆنتراست شیکرانەوە بۆ دیاریکردنی مەترسی تێکچوی گورچیلە. دۆزینەوەکان بەشدارن لە تێگەیشتنێکی باشتر لە کاریگەرییەکانی کۆنتراست میدیا لەسەر کارکردنی گورچیلە، پشتگیری لە بڕیاردانی کلینیکی بۆ نەخۆشانی مەترسیدار دەکات. تۆژینەوەکە ڕەنگە یارمەتیدەر بێت لە ڕێنماییکردنی پراکتیزە کلینیکییە سەلامەتەکان، لەوانەش بەکارهێنانی ستراتیژییەکانی خۆپارێزی بۆ دانیشتوانی کە لە مەترسیدان.