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Kidney Damage Secondary to Iodinated Contrast Products: 46 Cases in Mali

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Abstract **Original Research Article**

***Introduction*:** The number of patients undergoing radiological interventions with injection of iodinated contrast media (PCI) to arrive at a diagnosis is increasing. Prophylaxis of nephropathy with iodinated contrast media (NPCI) is therefore important. AKI (acute renal failure), in the context of NPCI is defined by an increase of 25% or more of 44umol/ in plasma creatinine concentrations compared to the initial values and typically occurs within 3 days after the injection of iodinated product in the absence of other etiologies. The objective of our work was to define the epidemiological, clinical, biological and evolutionary characteristics of nephropathy induced by iodinated contrast media. ***Material and Methods*:** This was a cross-sectional, retrospective study, extending over a period of 5 years in the nephrology department of the CHU du point ''G''. Were included in our study, all patients who had been received in the department for impaired renal function and who had previously undergone a CT scan. The parameters studied were the epidemiological characteristics, clinical and paraclinical data. ***Results*:** Our study involved 46 patients out of a total of 3420 patients admitted to the department for chronic renal failure, i.e. a frequency of 1.34% of cases. The average age of our patients was 53.5 years with extremes ranging from 22 to 85 years. A male predominance was noted with 29 men against 17 women with a sex ratio = 1.70. End-stage chronic renal failure (ERF) represented the majority of our patients in 15 cases, followed by early ERF in 11 cases and moderate ERF in 10 cases. High blood pressure (HBP) was the most common comorbidity in our series, i.e. 24 cases, followed by diabetes in 3 cases. In our series, cerebral CT angiography was the most commonly performed examination in 15 cases, followed by uro-CT in 11 cases and abdominal CT angiography in 10 cases. ***Conclusion*:** Nephropathy induced by iodinated contrast media is thought to be due to vasoconstriction, renal ischemia and direct cellular toxicity of the iodinated contrast media. Renal function should be assessed before any intravenous iodine injection.

Keywords: Renal Failure, Contrast Agent and Mali.

INTRODUCTION

The number of patients undergoing radiological interventions with injection of iodinated contrast products (ICP) to arrive at a diagnosis is increasing [1]. Prophylaxis of nephropathy with iodinated contrast products (ICP) is therefore important. Several studies have studied the interest of extra-renal purification methods to prevent ICP in patients with reduced renal function. Among these, some have demonstrated a utility (hemodiafiltration), while others have proven to be little or not at all effective (hemodialysis) [2]. Acute renal failure (ARF) secondary to the administration of ICP appears to be the third cause of ARF acquired during hospitalization [1]. ARF, in the context of ICP, is defined by an increase of 25% or more of 44umol/ in plasma creatinine concentrations compared to initial values and typically occurs within 3 days after the injection of iodine product in the absence of other etiologies [2]. The clinical syndrome ranges from moderate elevation of creatinine to oligoanuric IRA requiring extra-renal purification. The objective of our work was to define the epidemiological, clinical, biological and evolutionary characteristics of nephropathy induced by iodinated contrast products.

MATERIAL AND METHODS

This was a cross-sectional, retrospective study, spanning a period of 5 years from January 2019 to December 2023 in the nephrology department of the CHU du point "G". Were included in our study, all patients, of any age and sex, who were received in the department for impaired renal function and who had undergone computed tomography (CT) with injection of iodinated contrast product before or after. A survey form was used for data collection. The examinations were done with a GE (General Electric) optima 16-bar scanner. The parameters studied were the epidemiological characteristics, clinical data and paraclinical data (Biological and CT).

RESULTS

Our study included 46 patients out of a total of 3420 patients admitted to the department for chronic renal failure, representing a frequency of 1.34% of cases.

Epidemiological Characteristics

The mean age of our patients was 53.5 years with extremes ranging from 22 to 85 years. A male predominance was noted with 29 men against 17 women **(Figure 1)** with a sex ratio = 1.70.

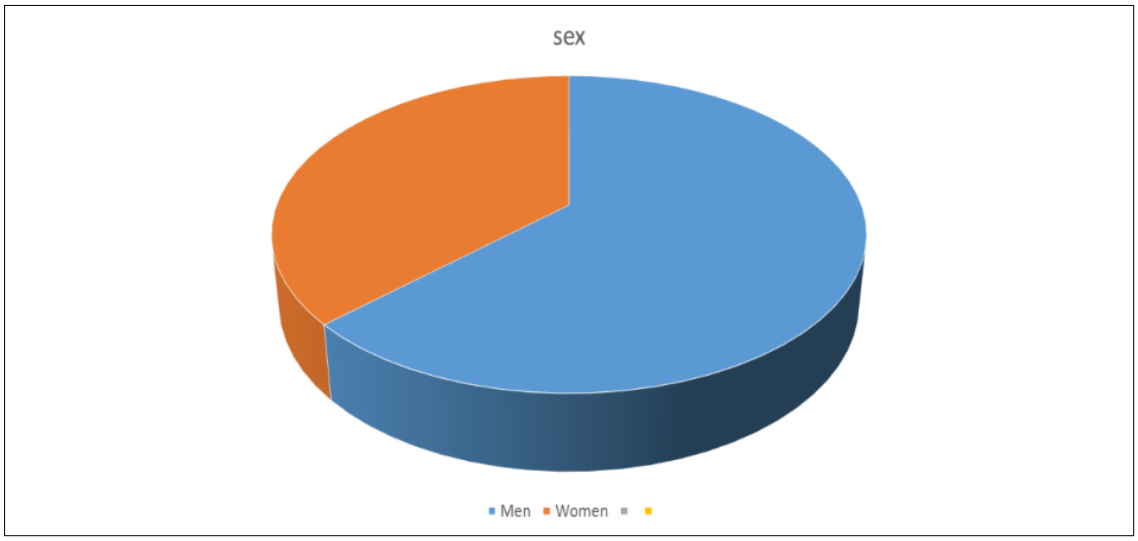

Figure 1: Distribution of patients by gender.

Clinical and Biological Data

Iatrogenic nephrotoxicity, particularly secondary to iodinated contrast agents, exists despite the use of less toxic products. End-stage chronic renal failure (CRF) represented the majority of our patients in 15 cases or 32.61 % of cases followed by early CRF in 11 cases or 23.93 % of cases and moderate CRF in 10 cases or 21.74 % of cases **(Table I).** Arterial hypertension (HTA) was the comorbidity the most frequent in our series, i.e. 24 cases (52.17 % of cases) followed by diabetes in 3 cases (6.52 % of cases) The same patient could have one, two or three associated comorbidities **(Table I).**

Table I: Stage of renal function at the time of administration of iodinated contrast product

Renal Function Status	Number of Cases	Percentage (%)
Normal renal function	0	0%
Early-stage chronic kidney disease	11	23.93%
Moderate chronic kidney disease	10	21.74%
Severe chronic kidney disease	10	21.74%
End-stage chronic kidney disease	15	32.61%
Total	**46**	**100%**

Table II: Distribution of patients according to comorbidities

Comorbidities	Number of Patients	Percentage %
Asthma	2	4.35 %
Diabetes	3	6.52 %
Heart failure	0	0 %
Liver failure	0	0 %
Multiple myeloma	0	0 %
Tumors	0	0 %
Stroke	1	2.17 %
Hypertension	24	52.17 %
None	17	36.96 %
Total	**46**	**100 %**

Scanographic Data

In our series, cerebral CT angiography was the most commonly performed examination in 15 cases, or 32.61 % of cases, followed by uro-CT in 10 cases, or 21.74 % of cases, and abdominal CT angiography in 10 cases, or 21.74 % of cases **(Table III).** The same patient could have two types of CT scan for different indications **(Figure 2 and 3).**

Table III: Distribution of patients according to the type of CT scan performed

Type of CT Scan	Number of Cases	Percentage (%)
Uro-CT	10	21.74 %
Thoracic Angio-CT	9	19.57 %
Cerebral Angio-CT	15	32.61 %
Abdominopelvic Angio-CT	10	21.73 %
Thoraco-abdominopelvic Angio-CT	2	4.35 %
Total	**46**	**100 %**

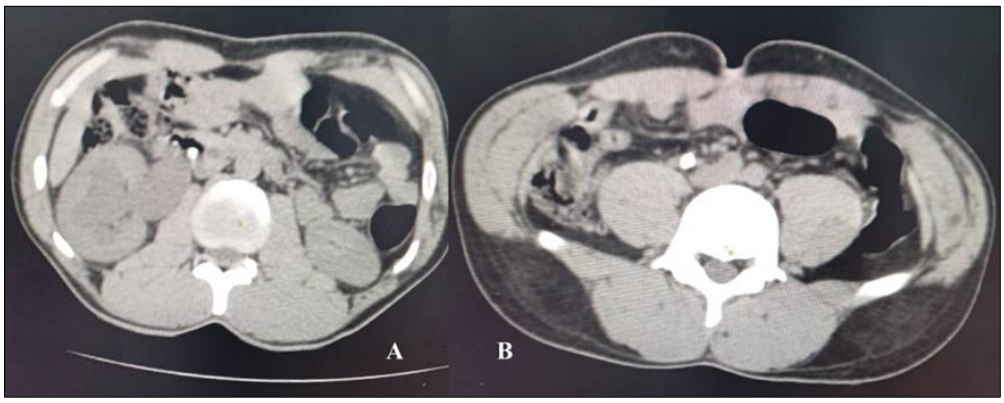

Figure 2 (A and B): Uro-CT in axial reconstruction without injection of PDC showing an obstructive right lumbar ureteral lithiasis with moderate ureterohydronephrosis on impaired renal function

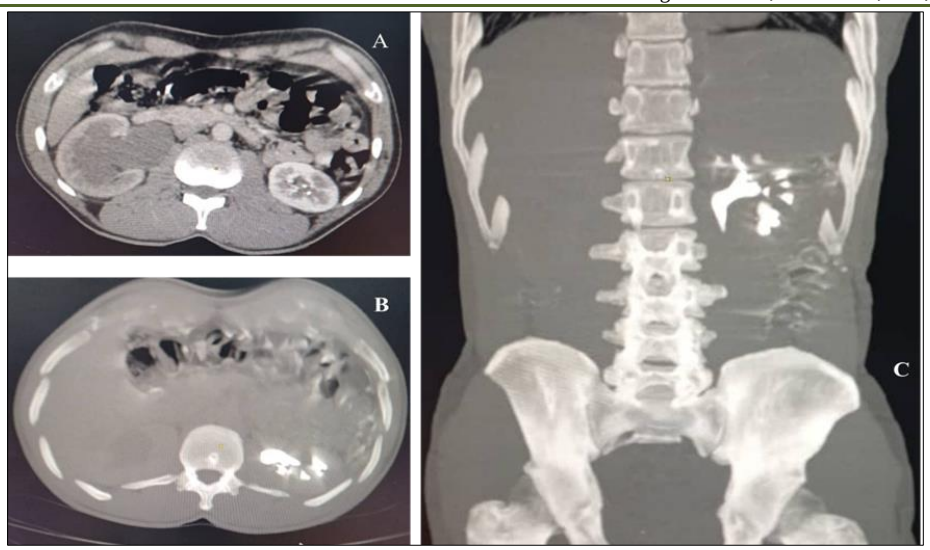

Figure 3 (A, B and C): Uro-CT in axial reconstruction in parenchymal window, nephrographic phase (A) and bone window in late phase (B) and in coronal reconstruction in bone window in late phase (C) objectifying a moderate right ureterohydronephrosis with delayed excretion in a patient with acute renal failure

DISCUSSION

The frequency of CIPN was 11% of drug-induced IRAs in the literature. It is more important among patients with risk factors such as: pre-existing renal failure, diabetes, sodium depletion, real or effective hypovolemia (heart failure, decompensated cirrhosis, nephrotic syndrome), associated nephrotic drug, arterial hypotension [1-9]. Our study involved 46 patients out of 3420 cases of renal damage, all with chronic renal failure, representing a frequency of 1.34% of cases. Its incidence varies in the literature from 0 to 25% depending on the studies [10-16]. The risks are all the greater when the PCI is hyperosmolar, when it is administered arterially, when the dose is high and when the administration is repeated at close intervals. PCIs must therefore be used at the lowest possible dose. The use of low osmolarity PCIs and iso-osmolar PCIs is associated with less nephrotoxicity. This should be preferred in patients with pre-existing chronic renal failure or one or more other risk factors. The search for renal failure is therefore important. A serum creatinine measurement should be performed before each PCI injection and, since this value is often misleading, the glomerular filtration rate should be estimated [3]. In our study, all patients had chronic renal failure with a GFR less than 30 ml/min. The risk is considered very low or nonexistent in the general population with a GFR greater than 30 ml/min [6, 7].

Mechanism of Nephrotoxicity of Iodinated Contrast Agents

The osmolarity of PCI causes, on the renal artery, after a brief vasodilation, vasoconstriction, a decrease in renal blood flow and glomerular filtration rate [17]. Some studies in diabetics and patients with a GFR less than 30 min/min had shown a significant increase in AKI. Davenport found 36.4% of AKI in patients with a GFR less than 30% according to the AKIN classification versus 19.4% in non-injected patients with the same renal function. The presence of pre-existing renal insufficiency therefore seems to be a major risk factor for worsening renal function in the event of PCI injection [18, 19]. All our patients had IR before PCI injection. Several vasoactive substances participate in this vasoconstriction: prostaglandins, natriuretic factor, adenosine, endothelin, vasopressin, noradrenaline, angiotensin and free radicals. They are responsible for a decrease in local blood flow and a decrease in tissue oxygenation, which is at the origin of a reduction in GFR, mediated by an increase in adenosine concentration [6-8]. A decrease in sodium reabsorption also participates in the reduction of GFR. This erythrocyte toxicity can have a clinical impact in situations of reduction in local blood flow by "sludge" effect. In addition, tubular obstruction can also participate in the development of renal failure, the contrast product promoting the precipitation of Tamm Horsfall protein (glycoprotein isolated from normal urine), myeloma light chains, and uric acid [9, 10].

Prevention of Nephrotoxicity to Iodinated Contrast Media (ICM)

Prevention also involves eliminating risk factors, namely pre-existing renal failure, diabetes, sodium depletion, real or effective hypovolemia, associated nephrotic drugs, arterial hypotension, hyperosmolar contrast media, high dose of iodinated contrast media, administration of iodinated contrast media at close intervals [11]. We recommend a few methods to reduce the risk of nephrotoxicity. Avoid iodine when possible or close examinations, by using another contrast media such as CO2 or a non-ionic iodinated contrast media and low dose of ICM; Choose MRI, Doppler ultrasound; Stop diuretics and NSAIDs 24 hours before the examination. Prevention is essentially based on hydration using isotonic saline (1 ml/kg/h, 4

hours before and 12 hours after the injection of the iodinated contrast product) or sodium bicarbonate (bolus 3 ml/kg one hour before the examination and perfusion of 1 ml/kg for 6 hours afterwards) [13, 14].

Interest of a Hemodialysis Session after the Injection of Iodinated Contrast Product in the Chronic Hemodialysis Patient

The problem of nephrotoxicity does not arise in the chronic hemodialysis patient (except for the maintenance of a residual diuresis) due to the terminal stage of renal failure. Iodinated contrast products being osmo-active agents, they can cause hydroelectrolytic abnormalities (hyponatremia, hyperkalemia, metabolic acidosis) and/or a polemical expansion in oligo-anuric hemodialysis patients and/or suffering from heart failure. A volume overload by iodinated contrast products can easily cause acute pulmonary edema. The following preventive measures have been proposed in chronic hemodialysis patients, namely the injection of a minimum dose of iodinated contrast product, the use of iso-osmolar contrast products, the performance of a control blood ionogram 1-4 hours after the procedure in order to control natremia and kalemia and to carry out a hemodialysis session as soon as possible.

CONCLUSION

In patients with chronic renal failure, iodinated contrast agents may be responsible for worsening renal function. Nephropathy induced by iodinated contrast agents may be due to vasoconstriction, renal ischemia and direct cellular toxicity of the iodinated contrast agent.

Ethical Considerations: informed consent was obtained from all patients and anonymity was maintained.

Conflict of Interest: none

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