Frequency and predictors of contrast-induced nephropathy after cardiac catheterization in diabetic patients

Thesis for partial fulfillment of master degree in cardiology
Submitted By

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M.B.B.Ch.

Under Supervision of

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Introduction

Contrast nephropathy (CN) is an important cause of iatrogenic acute renal failure and carries a significant risk for affected patient. CN is the third leading cause of acute renal failure in hospitalized patients. [1]

Cardiac catheterization and percutaneous intervention in diabetic patients is a relatively safe procedure but has a well – defined risk of morbidity and mortality. The use of contrast media in this procedure has been documented to be responsible for some risk. [2]

Despite advances in the percutaneous care, including stents and advanced anti-thrombotics, the risk of CN remains unchanged over last decade. CN was defined as an increase in the serum creatinine level of at least 44 u.mol /lit (0.5 mg/dl) above the base line value with in 48 hrs of exposure to contrast agent. [3]

CN appear to be the result of a synergistic combination of direct renal tubular epithelial cell toxicity and renal medullary ischemia. [4] Direct cytotoxicity in CN is suggested by histological changes of cell injury and enzymuria after contrast administration. [5] The nature of the contrast, associated ion, concentration, and concomitant hypoxia are all important to the degree of cellular damage, while the osmolality of the solution seems to be of secondary importance. [6]

An increase in serum creatinine (SCr) after percutaneous coronary intervention (PCI) is common and detrimental to prognosis. [7] Nephropathy after contrast exposure has been shown to be strongly associated with increased mortality [8], [9] and other adverse cardiac events. [11]

The most consistent predictor of contrast nephropathy is baseline renal dysfunction and hence, studies assessing preventive measures focus on this subgroup of patients. Diabetes mellitus also remains a consistent independent predictor of contrast nephropathy. [9], [10], [12], [13]

Although an initial study showed that patients with diabetes with preserved renal function were not at increased risk for contrast nephropathy. [14] Subsequent studies have shown diabetes to predict contrast nephropathy in populations with normal baseline creatinine. It is currently not routine practice to administer Reno-protective strategies in this subgroup of patients. Further study of this subpopulation may identify the necessity of these strategies. [11], [13]

The incidence of CIN increases in patients with certain condition, such as impaired kidney function [estimated Glomerular filtration rate (eGFR < 60 ml/min/1.73 m2)], and impaired left ventricular ejection fractions (<40%). [15] When diabetes is present in addition to one of the above, the incidence of CIN increases further. [16]

Based largely upon historical controls and data extolling the benefit of intravenous fluids, patients who are dehydrated or volume-depleted prior to CM administration also have an increased risk of CIN. Finally, any condition that impairs renal blood flow, such as hypotension, or Non-steroidal anti-inflammatory drugs, is also likely to increase the risk of CIN. [17]

Therefore, the first step in designing strategies to minimize the incidence of CIN is to correctly identify those individuals at greatest risk. [17]

AIM OF THE WORK

The aim of this work is to determine frequency and predictors of contrast-induced nephropathy after cardiac catheterization in diabetic patients. This study will be helpful in establishing the proportion of diabetic patients having contrast-induced nephropathy after cardiac catheterization in our community.

PATIENTS AND METHODS

*Inclusion Criteria:

This study will include:

- All diabetic patients after cardiac catheterization who underwent coronary angiography or percutaneous coronary intervention (PCI) from November 2008 to December 2009 in catheter lab of Ain Shams University.

*Exclusion Criteria:

- Patients on regular dialysis.
- Cardiogenic shock.
- Sever decompensated heart failure.
- Marked hypertension.
- Serum creatinine > 2 mg/dl.

Methods:

Every patient will be subjected to the following:

1-Proper history taking

With emphasis on risk profile of the patient

- Duration of diabetes.
- Severity of diabetes.
- Anti diabetic medications.
- Degree of control of diabetes.

2-Clinical examination (including local cardiac examination):

- Calculation of body mass index.
- Exclusion of the patients with the exclusion criteria.

3-ECG: For detection of patients with ischemic changes or arrhythmias.

4- Serum creatinine:

- at base line, before contrast exposure
- follow up of serum creatinine at the 3rd day and the 7th day of contrast exposure

7- Creatinine clearance:

Cockcroft-Gault formula is used to measure the estimated creatinine clearance eCrCl (ml/min)

$$eCrCL = \frac{(140 - Age) \times weight\ (kg) \times 0.85\ if\ (female)}{72 \times SCr\ (mg/dl)}$$

5- Cardiac catheterization

6-predictors of contrast induced nephropathy

Procedure related

**** Contrast media used***

(1) Ionic contrast media of high osmolality
(2) Ionic contrast media of low osmolality
(3) Nonionic contrast media

**** Amount of Contrast media used***

The amount of contrast will be individualized according to weight of patient and the length of the procedure with maximum amount of 4cc/kg.

**** Hydration:***

Type of hydration (saline or sodium bicarbonate)
Amount hydration

**** Use of N-Acetyl cysteine or not***

**** Length of procedure***

Patient related:

If one of the following is present and it's relation to CIN occurrence

*** Age** *** Sex**
*** Current smoker** *** body mass index**
*** Hypotension** *** Hypertension**
*** Renal insufficiency** *** Blood transfusion**
*** Oral hypoglycemics** *** Insulin**
*** Congestive heart failure** *** Hypercholesterolemia**
*** Peripheral vascular disease** *** Previous CABG**
*** Previous myocardial infarction**
***Volume depletion before the procedure**

- Duration of fasting
- Amount of water drink

***Nephrotoxins**

- ❖ Angiotensin-converting enzyme inhibitors (ACE)
- ❖ Angiotensin receptor blockers (ARBs)
- ❖ Non-steroidal anti-inflammatory drugs (NSAIDs)
- ❖ Lithium
- ❖ Cyclosporine
- ❖ Tacrolimus
- ❖ Aminoglycosides
- ❖ Amphotericin B
- ❖ Acyclovir
- ❖ Methotrexate
- ❖ Sulfa antibiotics
- ❖ Triamterene

STATISTICS

All the results will be subjected to adequate statistical analysis including mean and standard deviations, tabulated & will be discussed.

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مقدمةالرسالة:

اعتلال الكلى الناتج عن الصبغة المستخدمة هو من أهم أسباب الفشل الكلوى الحاد الناتج عن استخدام الادوية و يعتبر أيضا ثالث سبب مؤدى الى الفشل الكلوى الحاد الذى يحدث للمرضى فى المستشفيات.

تعتبر القسطرة القلبية والقسطرة التداخلية أمنه الى حد ما و لكن لها بعض المخاطر و قد تتسبب فى بعض الامراض و قد تؤدى الى الوفاه و أكثر هذه المخاطر يرجع الى الصبغه المستخدمة.

و بالرغم من التقدم فى مجال العناية بالقسطرة التداخلية (الدعامات—الأدوية المضادة للتجلط) فان معدل حدوث الاعتلال الكلوى الناتج عن الصبغة المستخدمة لم يتغير كثيرا فى العقد الأخير.

يعرف اعتلال الكلى الناتج عن الصبغة المستخدمة على أنه ارتفاع نسبة الكرياتينين بالدم أكثر من 5, مجم /ديسليتر أعلى من المعدل الطبيعى فى خلال 48 ساعة من التعرض للصبغة.

ينتج اعتلال الكلى الناتج عن الصبغة المستخدمةمن:
1. التسمم المباشر للخلايا المبطنة للأنابيب الكلوية
2. نقص الدم الواصل الى النخاع الكلوى

ويستدل على التسمم المباشر للخلايا من التلف و التغيرات التى تحدث للخلايا والانزيمات الموجوده فى البول.

العوامل المهمة التى تؤثر على درجة تلف الخلايا
1. طبيعة الصبغة المستخدمة
2. الايونات المصاحبة للصبغة
3. نقص كمية الاكسجين المصاحبة

أما أوزموزية المحاليل فتأتى فى الدرجة الثانيه من الأهمية و أصبحت الزيادة فى نسبة الكرياتينين بالدم بعد القسطرة القلبية شائعة وتؤثر فى الحالة الصحية للمريض وتكون مصحوبة بزيادة نسبة الوفيات.

أكثر العوامل التى تنبىء بحدوث اعتلال الكلى الناتج عن الصبغة المستخدمة هو اختلال وظائف الكلى مسبقا و يظل مرض السكرى أيضا من العوامل المهمة التى تنبىء بحدوث اعتلال الكلى الناتج عن الصبغة المستخدمة.

وتظهر دراسات مبدئية أن مرض السكرى مع وظائف الكلى الطبيعية تكون معهم نسبة حدوث اعتلال الكلى ضئيلة ولكن دراسات أخرى تثبت أن مرض السكرى ينبىء بحدوث اعتلال الكلى الناتج عن الصبغة المستخدمة. و تجد دراسات تثبت أنه ليس من الضرورى اتخاذ اجراءات روتينية لحماية الكلى ودراسات أخرى حول ضرورة اتخاذ هذه الاجراءات لحماية الكلى.

تزداد نسبة حدوث اعتلال الكلى الناتج عن الصبغة المستخدمة فى :

- اختلال وظائف الكلى
- ضعف انقباض عضلة القلب
- مرض السكرى
- الجفاف ونقص كمية السوائل بالدم
- أى حالة تقلل من سريان الدم للكلى كما في:
  * انخفاض ضغط الدم
  * أدوية مضادات الالتهاب

ولهذا تعتبر أول خطوة فى استراتيجية تقليل حدوث اعتلال الكلى الناتج عن الصبغة المستخدمة هو معرفة العوامل التى تنبىء بحدوثه.

الهدف من الرسالة:

دراسة معـــدل اعتلال الكلى الناتج عن الصبغة المستخدمة مـا بعـد القسطـــره القلبيـــه والعوامل التى تنبئ و تؤثر فى حدوثه فى مرضى السكر.

المرضى و الوسائل

المرضى الذين سيشملهم البحث:

- تشمل الدراسة كل مرضى السكرممن خضعوا لقسطرة القلب فى الفترة ما بين نوفمبر 2008 الى ديسمبر 2009 بقسم قسطرة القلب بجامعة عين شمس.

المرضى سيتم إستبعادهم من البحث:

- مرضى الغسيل الكلوى
- الهبوط الحاد فى الدورة الدموية
- احتشاء عضلة القلب الحاد الغير مستقر
- ارتفاع ضغط الدم الشديد
- ارتفاع نسبة الكرياتينين فى الدم أكثر من 2 مجم لكل ديسليتر

سوف تشتمل الدراسة على الخطوات التالية:

1. استعراض التاريخ المرضى للمرضى
2. الفحص الطبى الاكلينيكى الشامل لكل مريض
3. رسم القلب الكهربائى
4. قياس نسبة الكرياتينين فى الدم قبل القسطرة و فى اليوم الثالث واليوم السابع بعد القسطره
5. حساب تصفية الكرياتينين
6. اجراء القسطرة التشخيصية أو التداخلية للشرايين التاجية
7. تسجيل العوامل التى سوف تنبىء بحدوث اعتلال الكلى