



A CASE OF POST-ERCP AKI

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ABSTRACT

Acute kidney injury (AKI) is a severe clinical syndrome reported in patients undergoing endoscopic procedures such as colonoscopy [1] or endoscopy of the upper gastrointestinal tract for variceal [2] and nonvariceal bleeding [3,4]. But, Little is known about acute kidney injury (AKI) post- ERCP. Here we present a case of Post-ERCP AKI which occurred 48 hours after the patient underwent the procedure for choledocholithiasis.

KEYWORDS :

INTRODUCTION:

Endoscopic retrograde cholangiopancreatography (ERCP) is considered a great innovation related to the management of individuals with pancreaticobiliary diseases, although it is a complex and technically challenging procedure that implies the highest risk for complications among all routine endoscopic procedures [1]. The incidence of AKI in patients who underwent ERCP has been poorly described in the literature. Chronic kidney disease (CKD) and end-stage renal disease (ESRD) have a known association with post-ERCP prolonged hospital stays, higher in-hospital mortality rates, and considerably larger hospital charges [5,6]. Furthermore, ESRD and CKD are associated with higher post-ERCP adverse events, including bleeding and post-ERCP pancreatitis [6,7]. By contrast, there are scarce data available concerning AKI post-ERCP and its potential impact on patient's outcome. Few studies have shown a prevalence of AKI post-ERCP ranging from 11.48% to 17% [8-10]. It is assumed that the occurrence of AKI after ERCP is an independent risk factor for in-hospital mortality [11].

Case Report:

A 55 year old, hypertensive, hypothyroid female with history of cholecystectomy done 3 years back presented to our department at SMHS hospital with two month history of complaints of recurrent biliary pain and jaundice. MRCP done initially showed dilated CBD with central IHBRD with 9-10mm calculi seen in common bile duct (CBD) with an aberrant Right hepatic duct and a diagnosis of choledocholithiasis was made. The patient underwent ERCP and CBD was cleared and cannulated. The initial baseline investigations before and after the procedure are tabulated.

Table: 1

FULL BLOOD COUNT						
BEFORE PROCEDURE			AFTER PROCEDURE			
TLC	HB	PLT	TLC	HB	PLT	
7.60	10.8	245	9.81	11.4	202	

Table: 2

KIDNEY FUNCTION TESTS (KFT)					
BEFORE PROCEDURE			AFTER PROCEDURE		
UREA	CRET	DAY2	DAY4	DAY5	DAY6
UREA/CRET	UREA/CRET	UREA/CRET	UREA/CRET	UREA/CRET	UREA/CRET

16	0.8	188/5.90	171/5.0	91/2.6	37/1.3
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Table: 3

OTHER INVESTIGATIONS						
BEFORE ERCP			AFTER ERCP			
Na+	142	134	143	145	149	145
K+	3.9	4.0	3.9	4.0	3.8	3.7
Bil	0.8		1.1	1.2		
AST	25		36	64		
ALT	27		70	94		
ALP	125		181	172		
Ca	9.1		9.3			
Po4	4.6		5.1			
PT/INR	12/1.0		12.6/0.97			

Table: 4

OTHER INVESTIGATIONS	
URINE	PUS CELLS 2-9; ALB 0.3; RBC 19.5 ALB/CRET RATIO = 118.92
ANA HEP 2	1+ (SPECKELED)
P ANCA	NEGATIVE
C ANCA	NEGATIVE
ANTIGBM	NEGATIVE
PHASE CONTRAST FOR RBC MORPHOLOGY	RBCS WITH 5% DYSMORPHIC CELLS
URINE FOR EOSINOPHILS	MANY EPITHELIAL CELLS SEEN. OCCASIONAL EOSINOPHILS SEEN. SOME NEUTROPHILS ALSO SEEN.

USG FOR KIDNEYS	RK 11.4X5 LK 10.9X4.5
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HIV1,2	NEGATIVE
HEPATITIS SEROLOGY B	NEGATIVE
HEPATITIS SEROLOGY C	NEGATIVE

It is clear from the above investigations that patient had AKI. By excluding other causes of AKI, finally the diagnosis of Post ERCP AKI was made.

DISCUSSION

Worldwide, AKI is a common complication in hospitalized patients, occurring in 20.0–31.7% of patients at diverse levels of in-hospital care [12], and being associated with significantly higher morbidity and short- and long-term

mortality [13]. The literature with regard to the incidence of AKI after ERCP exists in scarce amounts, despite the fact that nowadays ERCP is a common procedure used for treating diseases of the biliary and pancreatic ducts, and its utilization has increased 30-fold in the last decades [3]. Two retrospective studies reported an incidence of post-ERCP AKI around 17% [9,10]. The pathophysiology of post-ERCP AKI is complex and only partially understood. ERCP itself triggers a systemic inflammatory response [14,15], which plays an important role in renal damage associated with AKI, leading to microvasculature dysfunction and alteration in tubular cells' functions [16]. The following parameters were regarded as independent predictors for AKI: lower eGFR level, higher nonrenal CCI score(Charlson comorbidity index score), choledocholithiasis as indication for ERCP(as in our patient), and elevated levels of total bilirubin. Post ERCP AKI is also associated with increased hospital stay. In patients with post-ERCP AKI, the rate of in-hospital mortality is 7.76% compared with 0.3% in the non-AKI group. This finding is similar to the discovery in a large ERCP procedures database from the United States, in which post-ERCP AKI was associated with an in-hospital mortality rate of 7% as compared to 0.6% in ERCP patients without AKI [12].

CONCLUSION

Patients undergoing ERCP are vulnerable to development of AKI. For these patients, it is very important to intervene early and to provide an adequate hydration, while avoiding fluid overload, and also to withdraw or not to add possible nephrotoxins.

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