

Research Article

ESTIMATION AND ASSESSMENT OF PROTEIN C/S AMONG SUDANESE PATIENTS IN ALJAZEERA STATE WITH CHRONIC RENAL FAILURE

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ARTICLE INFO

Article History:

Received 09th, September 2015
Received in revised form
13th, October 2015
Accepted 26th, November 2015
Published online 30th, December 2015

Keywords:

Chronic kidney disease,
cardiovascular disease,
Protein.

ABSTRACT

Background:

Chronic renal failure: Chronic kidney disease (CKD) describes abnormal kidney function and/or structure. There is evidence that treatment can prevent or delay the progression of CKD, reduce or prevent the development of complications, and reduce the risk of cardiovascular disease (CVD). The definition of CKD is based on the presence of kidney damage (ie albuminuria) or decreased kidney function (ie glomerular filtration rate (GFR) <60 ml/minute per 1.73 m²) for three months or more, irrespective of clinical diagnosis.

Protein C & S

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INTRODUCTION

Protein C, also known as autoprothrombin IIA and blood coagulation factor XIV, MW 62KDa and a key component of the natural anticoagulant pathway, is a zymogen, the activated form of which plays an important role in regulating anticoagulation, inflammation, cell death, and maintaining the permeability of blood vessel walls in humans and other animals. Activated protein C (APC) performs these operations primarily by proteolytically inactivating proteins Factor V_a and Factor VIII_a. APC is classified as a serine protease as it contains a residue of serine in its active site. In humans, protein C is encoded by the *PROC* gene, which is found on chromosome 2.

The zymogenic form of protein C is a vitamin K-dependent glycoprotein that circulates in blood plasma. Its structure is that of a two-chain polypeptide consisting of a light chain and a heavy chain connected by a disulfide bond. The protein C zymogen is activated when it binds to thrombin, another protein heavily involved in coagulation, and protein C's activation is greatly promoted by the presence of thrombomodulin and endothelial protein C receptors (EPCRs). Because of EPCR's role, activated protein C is found primarily near endothelial cells (i.e., those that make up the walls of blood vessels), and it is these cells and leukocytes (white blood cells) that APC affects.

Because of the crucial role that protein C plays as an anticoagulant, those with deficiencies in protein C, or some kind of resistance to APC, suffer from a significantly increased risk of forming dangerous blood clots (thrombosis).

Protein C may be measured by

- **Immunological:** by means of an ELISA assay Remember this measures only the amount of Protein C present and NOT its functional activity.
- **A clot-based functional APTT assay:** The time to clot formation after addition of a Protein C activator is determined and from this the amount of Protein C present can be determined.
- **A Chromogenic assay:** Protein C is activated using (commonly) Protac™, an extract of the venom of *Akistrodon contortrix contortrix* and the concentration of Protein C is determined from the rate of colour change in the test sample due to cleavage of a chromogenic substrate.
- **A thrombin:** Generation-based test has also been shown to detect Protein C deficiency.

MATERIALS AND METHODS

Commercial kits are readily available for Protein Assays ,We used Enzymes Linked Immuno Sorbent Assay (ELISA),and also we used a reference standard calibrated against the current International Standard for Protein C.

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Most ELISA assays use either monoclonal or polyclonal antibodies against Protein C. blood sample collection in Trisodium citrate and separated to plasma sample and saved in cold condition until we do examination by ELIZA.

Reference Ranges

The plasma concentration of protein C in a healthy baby is in the region of 40 IU/dL. Levels increased with age and reach levels of ~60 IU/dL but may not reach true adult

RESULTS

60 persons were participates in this study; 50 patient with chronic kidney disease were represent case group while 10 healthy participant represent control group. 35(58.3%) of all group were female, whereas 25 (41.7%) were male (Table 1). All participate fall in age ranged of 15-88 years the mean age (51) (Table 2).

Table 1.

Gender	Frequency	Percent	p.value
Case	Valid		
	Male	20	40.0
	Female	30	60.0
	Total	50	100.0
Control	Valid		
	Male	5	50.0
	Female	5	50.0
	Total	10	100.0

Table 2.

Variable	Case	Control	p.value
	mean±SD	mean±SD	
Age	54.21±21	32.8±17	0.004
Protein C	149.8±18.2	94.1±25	0.000

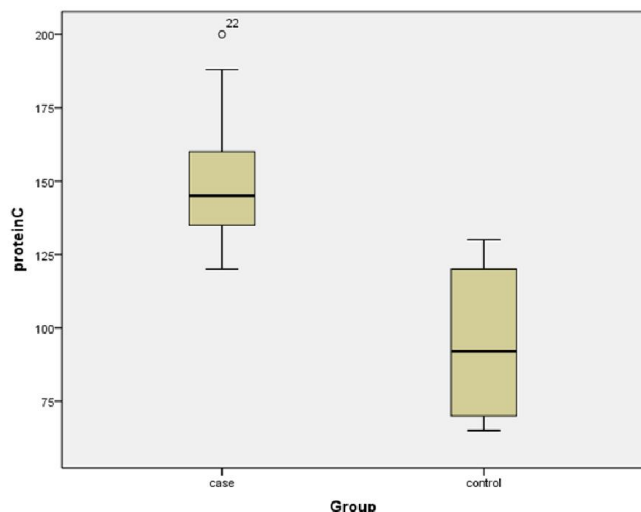


Figure 2. scatter plot shows correlation between protein C in chronic kidney disease patient and normal health persons (control)

According to other chronic disease 9(18%) of CKD patient have diabetes mellitus; while 10(20%) have hypertension (Table 3 and 4 respectively). Most of case group was make dialysis tow times per week 36(72%) (Table 5). The mean concentration of serum level of protein C among CKD patient was (149.8 IU/dL) while control group was (94.1 IU/dL). The serum level of protein C were significantly elevated in patient with CKD when compare with control group (p.value = 0.000).

Table 3.

DM	Frequency	Percent	p.value	
Case	Valid			
	yes	9	18.0	
	no	41	82.0	
	Total	50	100.0	
Control	Valid	no	10	100.0

Table 4.

HTN	Frequency	Percent	p.value	
Case	Valid			
	yes	10	20.0	
	no	40	80.0	
	Total	50	100.0	
Control	Valid	no	10	100.0

Table 5.

Group	Valid	NO of dialysis		p.value
		Frequency	Percent	
Case	Valid	1	14	28.0
		2	36	72.0
		Total	50	100.0
Control	Valid	0	10	100.0

(Table 6, Figure1). Statistical correlation showed insignificant association of serum level of CKD with gender, diabetes mellitus and hypertension in CKD patients (p.value 0.566, 0.151 and 0.125 respectively) (Table 1, 3, 4 respectively).

Conclusion

In this study the serum level of protein C was significant with age and number of dialysis (p.value< 0.004, 0.000 respectively) (Table 2, 5 respectively)

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