Full Length Research Paper

Coagulation Profile (PT, APTT, fibrinogen level and platelets count) in Sudanese patients with acute pancreatitis

Sana Eltahir Abdalla1*, Hana Tajelsir Adam2 and Enaam Abdelrahman Abdelgadir3

Abstract

Pancreatitis is an uncommon disease characterized by inflammation of the pancreas. Acute pancreatitis affects about 50,000–80,000 each year. It is a condition that rises suddenly and may be quite severe, although patients usually have a complete recovery from an acute attack. The aim of this study is to measure the coagulation profile (prothrombin time, activated partial thromboplastin time, platelets counts and fibrinogen level) in Sudanese patients with acute pancreatitis. Fifty blood samples were collected from patients diagnosed as acute pancreatitis cases. Their PT, APTT and fibrinogen were estimated using coagulometer, and platelets were counted using Sysmex™ Kx21n. The mean prothrombin time of the patients’ group was 14.9± 3 sec. while it was 12.6± 1.4 sec in the control group and the mean activated partial thromboplastin time was 40.5±10.7 sec and 34± 1.9 sec in the patients and control groups respectively. The mean fibrinogen concentration in the patients’ group was 246.8± 105.2mg/dl, while it was 267± 52mg/dl in the control group. The mean platelets count was 244.9± 124.4x10³/l in patients’ group and 257.7± 55.7x10³/dl in control group. This study concluded from this study that prothrombin time and activated partial thromboplastin time were prolonged in acute pancreatitis and fibrinogen level was decreased. Moreover, the platelets counts showed normal values.

Keywords: Prothrombin time, Thromboplastin, Platelets, Fibrinogen.

INTRODUCTION

Blood coagulation is a complex process by which blood clot is formed. It is an important part of haemostasis where the blood clot occludes the blood vessel injury and hence stopping the bleeding. And thereafter repair of the damaged vessel begins. Disorders of coagulation can lead to haemorrhage or thrombosis. Coagulation begins almost instantly after an injury to the blood vessel endothelial lining (Gene, 2011). Platelets immediately form a plug at the site of injury; this is called primary haemostasis Secondary haemostasis occurs simultaneously where the coagulation factors respond in a complex cascade to form fibrin strands, which strengthen the platelet plug (Gene, 2011). The coagulation cascade in secondary homeostasis has two pathways which convert fibrinogen, a soluble protein, to insoluble strands of fibrin, which, together with platelets, form a stable thrombus (Heit, 2005). The intrinsic and extrinsic pathway model divides the initiation of coagulation into two distinct parts. The extrinsic pathway is thought to be responsible for the initial generation of activated Factor X (Xa), where as the extrinsic pathway leads to amplification of Factor Xa generation. Factor Xa plays a central role in the coagulation cascade because it occupies a point where the intrinsic and extrinsic pathways converge (Colman et al., 2002).

The other mechanism that occurs in equilibrium with
Table 1. Distribution of study population according to sex.

<table>
<thead>
<tr>
<th>Sex</th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Females</td>
<td>8</td>
<td>16</td>
</tr>
<tr>
<td>Males</td>
<td>42</td>
<td>84</td>
</tr>
<tr>
<td>Total</td>
<td>50</td>
<td>100</td>
</tr>
</tbody>
</table>

Table 2. The Mean PT and APTT in Acute Pancreatitis Patients and Control Group.

<table>
<thead>
<tr>
<th>Test</th>
<th>Sample</th>
<th>N</th>
<th>Mean</th>
<th>STD</th>
</tr>
</thead>
<tbody>
<tr>
<td>PT (sec) Control</td>
<td>50</td>
<td>12.6</td>
<td>1.4</td>
<td></td>
</tr>
<tr>
<td>Patients</td>
<td>50</td>
<td>14.9</td>
<td>3.8</td>
<td></td>
</tr>
<tr>
<td>APTT (sec) Control</td>
<td>50</td>
<td>34</td>
<td>1.9</td>
<td></td>
</tr>
<tr>
<td>Patients</td>
<td>50</td>
<td>40.5</td>
<td>10.7</td>
<td></td>
</tr>
</tbody>
</table>

Table 3. The Mean Fibrinogen in Patients with Acute Pancreatitis and Control Group.

<table>
<thead>
<tr>
<th>Fibrinogen (mg/dl)</th>
<th>N</th>
<th>Mean</th>
<th>STD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>50</td>
<td>267</td>
<td>52</td>
</tr>
<tr>
<td>Patients</td>
<td>50</td>
<td>246.8</td>
<td>105.2</td>
</tr>
</tbody>
</table>

Table 4. The Mean of Platelets in Patients of Acute Pancreatitis and Control Group.

<table>
<thead>
<tr>
<th>Test (x10^9/l)</th>
<th>N</th>
<th>Mean</th>
<th>STD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Platelet control</td>
<td>50</td>
<td>257.7</td>
<td>55.7</td>
</tr>
<tr>
<td>Platelet patient</td>
<td>50</td>
<td>244.9</td>
<td>124.4</td>
</tr>
</tbody>
</table>

clot formation is fibrinolysis, which is a process that prevents blood clots from growing and becoming problematic. This process has two types: primary fibrinolysis and secondary fibrinolysis. The primary type is a normal body process, whereas secondary fibrinolysis is the breakdown of clots due to a medicine, a medical disorder, or some other cause (Dugdale et al. 2011).

In fibrinolysis, a fibrin clot is broken down by the enzyme plasmin that cuts the fibrin mesh at various places, leading to the production of circulating fragments that are cleared by other proteases or by the kidney and liver (Cesarman-Maus and Hajjar, 2005).

Pancreatitis is inflammation of the pancreas that can be acute or chronic. Either form is serious and can lead to complications. In severe cases, bleeding, infection, and permanent tissue damage may occur (Frossard et al., 2008). The disease is a syndrome with discrete episode that may cause varying degrees of injury to the pancreas, adjacent and distant organs (Furie and Furie, 2005).

Acute pancreatitis (AP) is a potentially lethal disorder with no specific medical treatment. AP is characterized by a spectrum of symptoms, ranging from a local inflammatory process to more severe form (acute necrotizing pancreatitis) (Pandol et al., 2007).

In addition, pancreatitis may be further classified into acute interstitial and acute hemorrhagic disease. In the first type, the gland architecture is preserved but is edematous. Inflammatory cells and interstitial oedema are prominent within the parenchyma. Haemorrhagic disease is characterized by marked necrosis, haemorrhage of the tissue, and fat necrosis (Furie and Furie, 2005). There is marked pancreatic necrosis along with vascular inflammation and thrombosis (Frossard et al., 2008). Severe acute pancreatitis may cause dehydration and low blood pressure. The heart, lungs, or kidneys may fail and if bleeding occurs in the pancreas, shock and even death may follow (Furie and Furie, 2005).

Disturbances of blood coagulation may occur in acute pancreatitis. Acute pancreatitis (AP) is characterized by changes in both coagulation and pro inflammatory activities. In the pathogenesis of pancreatic necrosis, the pancreatic perfusion and hypoxia seem to play an important role. There is accumulating evidence that
microvascular disturbances (vasoconstriction, shunting, increase permeability, inadequate perfusion and increase blood viscosity and coagulation) are significant events in the progression of acute pancreatitis (Cuthberston and Christophi, 2006).

Objectives

The objective of this study was to estimate the prothrombin time (PT), activated partial thromboplastin time (APTT), fibrinogen level and platelets count in patients with acute pancreatitis.

METHODS

This is a descriptive case control study conducted in Khartoum Teaching Hospital in Khartoum State (Sudan), patients with acute pancreatitis were included, while any patients with bleeding disorder was excluded from the study, a written consent was taken from all patients. Designed questionnaire was used to collect data from the patients.

Fifty Sudanese patients with acute pancreatitis were enrolled in this study. The clinical diagnosis of pancreatitis was confirmed by the relevant investigations that included serum amylase and lipase, abdominal X-Ray and CT scan. Fifty normal people that matched the patients’ group were included as a control group.

Venous blood was collected by venepuncture in plastic containers containing 0.109 M sodium citrate in ratio of nine part of blood and one part of anticoagulant (the blood volume adjusted according to the amount of citrate in the container). Platelet poor plasma (PPP) was separated according to the recently recommended double centrifugation method, the resulting (PPP) was tested immediately.

PT, APTT and fibrinogen level were measured using automated coagulometer, and platelets were counted using Sysmex™ Kx21n, a fully automated haematological analyzer.

RESULTS

The age range of the patients’ group was 30-55 years with a mean of 40 years. According to sex, males were 42 (84%) and females were eight (16%) as seen in Table (1).

The mean value of PT in the patients’ group was 14.9± 3 sec. and it was 12.6± 1.4 sec in the control group.

The mean value of APTTT in the patients’ group was 40.5±10.7 sec while it was 34± 1.9 in the control group as shown in Table (2).

The mean value of APTT in the patients’ group was 40.5±10.7 sec while it was 34± 1.9 in the control group as shown in Table (2).

DISCUSSION

This study showed that, there is decreased plasma fibrinogen, prolonged PT, and prolonged APTT. These findings may be due to circulating factors originating from the inflamed pancreas that may damage various organs by variable mechanisms. The pancreatic disorders are responsible for significant alteration in coagulation system. Increased tendency to thrombosis (usually as deep vein thrombosis and occasionally widespread micro thrombi) is often recorded in acute pancreatitis (Furie and Furie, 2005).

These findings agreed with previous study replicates the findings of DIC secondary to acute pancreatitis conducted by Agarwal et al which found the following, altered plasma fibrinogen, raised PT , raised PTTK and raised T.T. Secondary fibrinolysis as detected by shortened euglobulin lysis time occurred in all (Agarwal et al., 1982). These findings also in agreement with Saif NW who stated that various hematological abnormalities including fall in serial values of hemoglobin or hematocrit, coagulation factor abnormalities, leukocytosis, acute hemolytic anemia, thrombocytopenia, and thrombotic thrombocytopenic purpura or hemolytic uremic syndrome have been reported in patients with acute pancreatitis. Similarly, abnormalities of blood coagulation factors consistent with disseminated intravascular coagulopathy (DIC) have also been noticed in patients with pancreatitis. The results of his study revealed abnormal prothrombin time and partial prothrombin time. Coagulation work-up revealed thrombin time 24.3 sec fibrinogen 110 mg/dl, D-dimers >1 and < 2, and fibrin degradation products >22 (Saif, 2005).

The levels of DIC parameters (level of platelets and higher levels of D-dimer) and thrombin-antithrombin complex upon admission have been found to be associated with increase severity poor prognosis of acute pancreatitis (Maeda et al., 2006). A four-fold increase in D-dimer levels has been shown to be a marker of complicated acute pancreatitis (Salmon et al., 2003).

In this study platelet counts showed normal values. The role of platelets in pathophysiology of disease has not been elucidated yet. There is activation of platelets during acute pancreatitis and alteration of platelet numbers and indexes between onset and remission of the disease which reflect the bone marrow response (Mimidis et al., 2004). Platelets are directly involved in the systemic inflammatory process of acute pancreatitis which leads to consumption of platelets and are compensated by an immediate bone marrow response.
A low platelet count as a component of disseminated intravascular coagulation is known to occur in severe and complicated pancreatitis (Mimidis et al., 2004). This finding supported the findings of the role of platelets in experimental acute pancreatitis as concluded by Br Surg who stated that Platelets play a crucial role in AP by regulating neutrophil infiltration in the pancreas (Abdulla et al., 2011).

CONCLUSION

This study concluded that PT and APTT were prolonged in patients with acute pancreatitis with decrease fibrinogen level and normal Platelets count.

REFERENCES

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