Cross Current International Journal of Medical and Biosciences
Abbreviated Key Title: Cross Current Int J Med Biosci
ISSN: 2663-2446 (Print) & Open Access
DOI: 10.36344/ccijmb.2023.v05i02.001

Volume-5 | Issue-2 | Jul-Aug, 2023 |

Case Report

Non-Pancreatic Hyperlipasemia and Chronic End Stage Renal Disease: A Case Report and Review of the Literature

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| **Received:** 27.05.2023 | **Accepted:** 04.07.2023 | **Published:** 13.07.2023 |

Abstract: The association of elevated pancreatic lipase during chronic end-stage renal disease is poorly studied in the literature. We report the case of a 51-year-old female patient which has an hyperlipasemia associated with chronic end-stage renal disease outside of acute pancreatitis. Knowledge of this association will help the clinician in the management of this patients.

Keywords: End stage renal disease - Pancreatic hyperlipaemia – Incidence- A case report.

INTRODUCTION

Elevated pancreatic enzymes, particularly hyperlipasemia, are often suggestive of acute pancreatitis. However, several studies report a high incidence of elevated pancreatic enzymes during renal failure outside of acute pancreatitis. We report a case of non-pancreatic hyperlipasemia associated with chronic end-stage renal disease.

OBSERVATION

Patient information:

We describe the case of 51-year-old female initially admitted to the nephrology department of the Mohammed V Military Hospital in Rabat for kidney failure.

The patient presented with asthenia and vomiting after taking analgesics for 20 days. The whole evolving in a context of apyrexia.

She had no history of diabetes, high blood pressure or known heart disease. Furthermore, she had intermittent edema of the lower legs without hematuria and a history of pregnancy-induced hypertension during her last two pregnancies.

Clinical Results

The clinical examination on admission revealed a conscious patient with preservation of the general state, hemodynamically stable and without epigastralgic abdominal pain.

Diagnostic assessment

The biological result on admission showed normochromic normocytic anemia at 7.7 g/dL, end-stage G5 chronic renal failure with uremia at 2.24 g/L (0.15-0.38), creatinine at 85 mg/L and DFG estimated by CKD-EPI at 5 ml/min/1.73m^2, uricemia at 107 mg/L (39-78) and calcemia at 56 mg/L(80-105). Alkaline phosphatases at 114U/L (32-91), bicarbonates at 10 mmol/L (21-28), parathyroid hormone at 829 pg/mL (15-68), lipasemia at 1712 IU/L (<78) with CRP at 7.1 (<5). The urine ionogram showed a preserved diuresis. The Albuminuria Creatinuria Ratio was 222 mg/g with a Proteinuria Creatinuria Ratio at 646 mg/g.

An abdominal computed tomography scan (CT-scan) revealed polycystic liver and kidney disease without lithiasis with some renal cysts showing hemorrhagic content. The pancreas has a normal size with respect to its lobulations.

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Citation: Leila Laamara, Asmaa Biaz, Sanama Bagadema Don Carleone, Samira ELMachtani idrissi, Sanae Bouhsain, Driss El Kabbaj, Abdellah Dami (2023). Non-Pancreatic Hyperlipasemia and Chronic End Stage Renal Disease: A Case Report and Review of the Literature. *Cross Current Int J Med Biosci*, *5*(2), 30-32.

The diagnosis was a non-pancreatic hyperlipasemia associated with chronic end-stage renal failure with liver and kidney polycystic disease without lithiasis.

Therapeutic intervention

Parenteral rehydration was initiated with intravenous calcium supplementation at 60 mg of calcium gluconate daily, transfusion of 02 units of red blood cells, and antihypertensive therapy at a dose of 10 mg daily for blood pressure control.

The patient was put on peritoneal dialysis while waiting for kidney transplantation. A biological control 1 month later showed an improvement with a decrease of the lipasemia to 314 IU/L.

DISCUSSION

Acute pancreatitis (AP) is a common disease and is the leading cause of emergency department visits for acute abdominal pain [1]. Its positive diagnosis is based on two of the following three criteria: Abdominal pain, elevation of serum lipase three times the upper limit of normal and suggestive imaging [2].

However, several studies report a high incidence of increased pancreatic lipase outside of acute pancreatitis during kidney failure, which usually does not exceed three times the normal level [3], with a correlation between the concentration of lipasemia and the severity of renal failure, with a maximum that can reach up to five times the normal level [4]. In our patient, this level is 1712 IU/L (20xN), which is related to the severity of the renal damage.

Lipase is a glycoprotein mainly secreted by the pancreas. Its plasma level reflects the balance between its production, catabolism and purification. The clearance of lipase enzyme is largely carried out by the kidney. Glomerular filtration is mainly responsible for the elimination of lipase enzyme, the majority of the filtered lipase enzyme is reabsorbed by the renal tubules with an intrarenal degradation. Indeed, the increase in lipasemia during chronic renal failure is explained by reduced glomerular filtration [5, 6], rather than by its increased release from the pancreas [7].

Although the molecular weight of lipase precludes it from having dialytic clearance [8], the plasma level of lipase decreases after dialysis in 56% of cases, respectively [4]. The mechanism for this decrease is unknown.

The association of elevated pancreatic lipase during end-stage renal disease is poorly studied in the literature. A study conducted by *W. Junge et al* on 28 patients with CKD showed a higher-than-normal plasma lipase concentration in 15 patients outside acute pancreatitis [6]. This same study had conducted an experiment on rats to study the contribution of the kidney in the elimination of lipase. After intravenous bolus injection of lipase in rats with normal renal function, serum activity of the enzyme decreased rapidly. The half-life of lipase was 18.1 min, with only traces of lipase activity found in the urine. In contrast, in animals with ligated kidneys, the serum half-life of lipase enzyme was 3 times longer [6].

CONCLUSION

Lipase is a specific marker for the diagnosis of acute pancreatitis; however, very high levels of lipase can occur during renal failure without pancreatic pathology in relation to the severity of renal involvement. Knowledge of this association will assist the clinician in the management of this patients.

Conflicts of Interest: The authors declare that there are no conficts of interest regarding the publication of this article.

ACKNOWLEDGMENTS

I would like to thank Professor Biaz Asmae, for his invaluable guidance and support throughout the research process. Her expertise, insights, and encouragement were invaluable in helping me to complete this work.

I also want to thank Professor Samira ELMachtani idrissi it was an immense honor to have been able to work alongside you and benefit from your experience, Professor Abdellah DAMI, for your kindness and your warm welcome, Professor Sanae BOUHSAIN Your seriousness, your competence and your sense of duty have left an enormous mark on us.

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