



An Unusual Combination of Acute Pancreatitis Etiologies

Akut Pankreatit Etiyolojilerinin Sıradışı Bir Kombinasyonu

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Abstract

Among several etiologic factors of acute pancreatitis (AP), hypertriglyceridemia and chronic drug use are less frequent causes, responsible for approximately 10% of all cases. Our aim is to present a case of AP who had hypertriglyceridemia and was receiving carbamazepine.

Keywords: Akut pancreatitis, hypertriglyceridemia, carbamazepine use

Öz

Birçok farklı etiolojik faktörün neden olduğu akut pankreatit (AP) vakalarının %10'dan, hipertrigliseridemi ve kronik ilaç kullanımı sorumludur. Amacımız hipertrigliseritemisi olan ve karbamazepin kullanan hastada gelişen akut pankreatit vakasını sunmaktır.

Anahtar kelimeler: Akut pankreatit, hipertrigliseridemi, karbamazepin kullanımı

Introduction

Acute pancreatitis (AP) is one of the most common gastrointestinal disorder that needs hospitalization with a mortality rate of 30% in mild to severe forms (1). Typical abdominal pain, elevated pancreatic enzymes (amylase and lipase) and characteristic tomographic findings are the mainstay in the diagnosis. The most common causes of AP are cholelithiasis and alcohol consumption. However, hypertriglyceridemia, drug use and anti-microbial agents are also involved in the etiology of AP. Serum levels of triglyceride in hypertriglyceridemia related AP usually exceed 500 mg/dl or even 1000 mg/dl (2). The pathogenesis of hypertriglyceridemia related AP is lipotoxicity which is caused by breakdown of triglycerides to free fatty acids by pancreatic lipase (3). Additionally, more than hundreds of drugs including antihypertensives, antibiotics, antiinflammatory drugs and antiepileptics are accused for AP (1). We present a case of AP who had hypertriglyceridemia and were receiving carbamazepin for epilepsy.

Case Report

A 58-year women who had no chronic disorder, except epilepsy, admitted to Emergency department with

abdominal pain and nause-vomitting lasting for 5 hours. She has been using carbamazepin for 10 years, and also had a history of AP episode 5 years ago. Except right upper quadrant abdominal pain, she had no complaint. First blood sample of the patient was lipemic which precludes analysis of biochemical variables (Figure 1). At this sample, lipase:878 U/L amilase: 485U/L, triglyceride:1420 mg/dl and CRP:29 mg/L. The other biochemical variables and hemogram parameters were all in normal range. No sign of cholelithiasis or dilatation on choledoch was observed. Tomographic examination revealed interstitial pancreatitis and fluid collection around inflammation area, but no pancreatic necrosis or hemorrhage (Figure 2). The patient was diagnosed as drug and hypertriglyceridemia related AP. Antiepileptic medication was switched to levetirasetam which is less likely to cause pancreatic inflammation. Until abdominal pain of the patients recovered, oral nutrition was forgiven and intravenous hydration was administered. After an uneventfull hospitalization for 5 days, clinical and biochemical improvements were achieved, and the patient was discharged by recommending out-patient clinical follow-up.



Figure 1. Chylous appearance of serum sample indicating hypertriglyceridemia

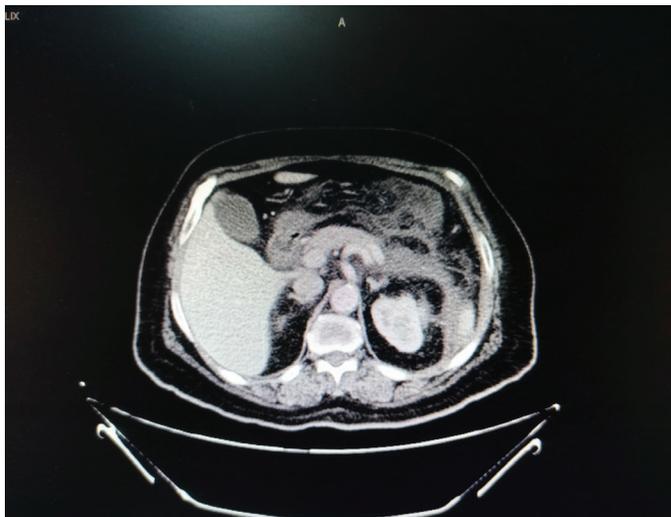


Figure 2. Tomographic examination showing interstitial pancreatitis and fluid collection around inflammation

Discussion

Acute inflammation of pancreatitis is associated with over 270,000 hospitalizations and costs up to 2.6 billion dollars per year (4). The outcome of AP may range from self-resolving disorder to multiorgan failure, or even death. Hypertriglyceridemia related AP is at the third rank, following gallstones and alcohol, with a frequency of 1-7% of patients with AP (2). Bessembinders et al showed that AP risk of patients with triglyceride >1000 mg/dl is 8% (5). They also demonstrated that the risk logarithmically rises if

there is comorbidity like diabetes, obesity, alcohol intake or chronic drug use. Although the clinical presentation of hypertriglyceridemia related AP is similar to other causes related AP, the risk of complications is higher (6). Clinicians should be aware of the fact that high triglyceride levels (>500 mg/dl) may suppress the release of pancreatic enzymes and may cause confusion in the diagnosis of AP (7). Nevertheless, authors recommend similar therapeutic approach to patients with hypertriglyceridemia induced AP, including cessation of oral intake, fluid repletion, opioid analgesia and rapidly decreasing serum triglyceride levels. In the past few decades, some authors have suggested plasmapheresis or combination of insulin and heparin in the management of hypertriglyceridemia induced AP (8). And also minimizing other risk factors of AP is essential like discontinuation of drugs which may trigger recurrence of AP.

On the otherhand, because it is difficult to establish a cause and effect relationship between drug and pancreatitis, controversies exist in the diagnosis and the treatment of drug induced AP. Therefore, case reports rather than randomized clinical trials dominate the literature. Majority of reports support the belief that cessation of suspected drug plays crucial role, and the patients experience rapid symptom relief. The Naranjo Algorithm (Table 1) is used to determine the probability of a change in the clinical status of a patient that is related to drug use rather than other factors (9). Our patient had a final score of 6 meaning that an adverse drug reaction which caused AP is probable. Thus, carbamazepine was switched to a newer and relatively safer antiepileptic drug 'Levetiracetam' (10). In our patient, symptoms declined after levetiracetam therapy initiated.

Drug or hypertriglyceridemia related AP is less frequently observed which may cause delay in the diagnosis of the disorder. The diagnosis of AP is established after excluding other etiologic factors. Carbamazepine induced pancreatitis is related to clinical situation and additional risk factors of AP, but not the dose or the duration of the medication.

Hypertriglyceridemia as seen in this case, may enhance the risk of AP for the patients who have been receiving carbamazepin. Although some cases require individualization in the management of the disorder, general approach to AP with different etiologies is similar. Patients with high risk of AP should be identified, and the risk factors like high triglyceride should be appropriately treated or drugs associated with high incidence of AP should be discontinued. In conclusion, clinicians should

Table 1. NARANJO algorithm (probability scoring of acute pancreatitis)

Interpretation of the total score

9 or more, ADR highly probable,

5 to 8 ADR is probable,

1 to 4, ADR is possible,

0 or less, ADR is doubtful

1. Are there previous conclusive reports on this reaction?	+1	0
2. Did the adverse event appear after the suspected drug was administered?	+2	-1
3. Did the adverse reaction improve when the drug was discontinued or a specific antagonist was administered?	+1	0
4. Did the adverse reaction reappear when the drug was re-administered?	+2	-1
5. Are there alternative causes (other than the drug) that could on their own have caused the reaction?	-1	+2
6. Did the reaction reappear when a placebo was given?	-1	+1
7. Was the drug detected in the blood (or other fluids) in a concentration to be toxic?	+1	0
8. Was the reaction more severe when the dose was increased, or less severe when the dose was decreased?	+1	0
9. Did the patient have a similar reaction on the same or similar drugs in any previous exposure?	+1	0
10. Was the adverse event confirmed by any objective evidence?	+1	0

NARANJO: The adverse drug reaction probability scale, ADR: Adverse drug reaction

consider initiating antiepileptic or other drug groups with low risk for pancreatitis to patients who had a history or additional risk factor for AP.

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