

ACUTE PANCREATITIS WITH NORMAL AMYLASE AND LIPASE LEVELS

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ABSTRACT

Acute pancreatitis (AP) is caused most commonly by various biliary tract diseases or heavy alcohol intake. The diagnosis is most commonly based upon characteristic history of acute abdomen accompanied with rise in levels of pancreatic enzymes namely amylase and lipase in serum. Imaging, particularly computed tomography (CT scan) is the investigation of choice to make a definitive diagnosis. However, the rise in serum pancreatic enzyme levels is not an absolute criterion to diagnose AP. We are reporting one such case of a young female who presented with acute abdomen, had normal pancreatic enzyme levels, but was detected to be suffering from AP on CT scan of abdomen.

Key Words: Acute pancreatitis, Serum Lipase, Serum amylase

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INTRODUCTION

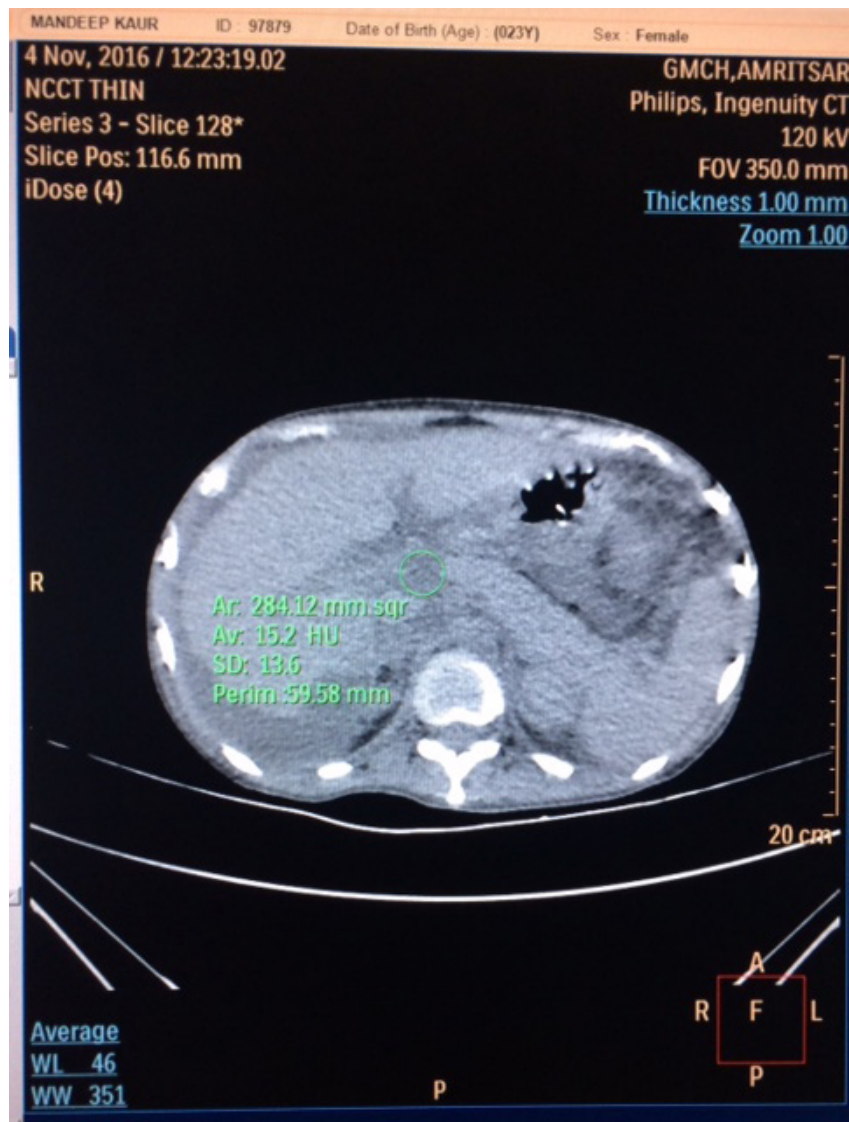
Serum amylase and lipase levels are the most commonly requested investigations in the emergency department for the diagnosis of acute pancreatitis (AP). The 3 fold or more increase in their levels is often seen in vast majority of patients with AP within 24 hours of the onset of the disease. But, their role in the diagnosis and prognosis has frequently been the subject of confusion and controversy as AP can and does occur with normal levels of amylase and lipase. We are reporting one such case of a young female who presented with acute abdomen, had normal pancreatic enzyme levels, but was detected to be suffering from AP on CT scan of abdomen.

CASE PRESENTATION

A 23 years old, married for the last 8 months, non-alcoholic, non-pregnant female reported to the casualty

of Govt. Medical College and Guru Nanak Dev Hospital, Amritsar, with complains of severe pain abdomen and shortness of breath from last 5 days. There was no history of tuberculosis, bronchial asthma, cardiovascular or any other chronic illness or poisoning. There was no history of constipation, obstipation or diarrhoea. The history of any septic abortion was also negative.

As she was severely dyspnoeic, she was referred to emergency of our institute. We received the patient in dehydrated and cyanosed state with severe respiratory distress, acute abdomen and shock. She was conscious, apprehensive, co-operative and well oriented to time, space and person. She had mild pallor, no jaundice, no clubbing, no lymphadenopathy and no oedema. The pulse was 90/ minute, regular and very feeble. The systolic blood pressure was 80 mm Hg, while the diastolic pressure was un-recordable. Her respiratory rate was 42/ minute; regular with abdomino-thoracic character. There was use of accessory muscles of respiration. The

Figure 1: Contrast enhanced computed tomography showing acute pancreatitis

oxygen saturation was 72% on room air. Her random blood sugar level was 125 mg/dl. She was afebrile and her rest of the general physical examination was unremarkable. Electrocardiograph showed tachycardia with ST segment depression in lead V1-V6.

On respiratory examination, the trachea was found to be slightly shifted to the left. The breath sounds were absent in the mammary, infra-mammary, mid-axillary, mid-scapular and infra-scapular regions of the right side of the chest. The vocal resonance and tactile vocal fremitus was decreased over the same areas and they were found to be stony dull on percussion. Normal vesicular breath sounds were heard over the left side along with normal palpation and percussion notes.

On abdominal examination, all the quadrants were found tense and extremely tender. Guarding, rigidity

and rebound tenderness was present. Sluggish bowel sounds were heard. Other than tachycardia, her cardiovascular and neurological examination revealed no abnormality.

A working diagnosis of peritonitis with reactionary right-sided pleural effusion along with circulatory shock was made and the patient was put on high flow oxygen, intravenous fluids, Ryle's tube aspiration with nil per orally, intravenous ceftriaxone and metronidazole. The laboratory investigations revealed total leucocyte count to be 9750/mm³ with the differential being polys 60%, Lymphos 35%, Monocytes 3% and Eosinophils 2%. The ESR was 34 mm at the end of first hour. The Blood urea read 114 mg/dl and serum creatinine was 2.1 mg/dl. The liver function tests were in the normal range. X-rays of abdomen showed no multiple air fluid levels or gas under the diaphragm. The X-ray chest revealed

moderate right sided pleural effusion. The USG of abdomen showed only mild ascites as the only abnormality. Serum amylase was found to be 78 U/L (normal range 25-98 U/L) and serum lipase 149 U/L (normal range 10-190 U/L). Serum calcium was 9.2 mg/dl (normal range 8.5-10.5 mg/dl) and calcium-ionised being 4.61 mg/dl (normal range 4.40-5.60 mg/dl). Therapeutic pleural tapping was done and around 1500 ml of yellowish and turbid pleural fluid was drained. The aspirate was sent for examination and it showed exudate character. Ascitic fluid analysis also revealed exudative picture with no evidence of spontaneous bacterial peritonitis. The ascitic fluid adenosine deaminase levels were found in normal range as 12.9 U/L (normal being less than 40 U/L).

Contrast enhanced computed tomography (CECT) of the patient, done on the third day of admission, revealed sub-acute intestinal obstruction with thickened gut wall, moderate ascites, bilateral pleural effusion (right more than the left) with collapse of the right lung and right sided adnexal cyst. The head and neck regions of the pancreas were found to be hypo-echoic and were not separately identified from the peri-pancreatic haze. The findings were suggestive of acute pancreatitis (Figure 1).

The patient was shifted to intensive care unit on revised antibiotic treatment with intravenous imipenem (500 mg) 8 hourly. The patient kept on deteriorating and went into multi-organ failure. She developed acute respiratory distress syndrome on the fifth day and unfortunately, she expired the following day.

DISCUSSION

In clinical settings, the diagnosis of acute pancreatitis (AP) is heavily relied upon the clinical picture and elevated pancreatic enzymes. Most commonly requested of them are serum amylase and lipase. Enzymes including elastase, P-isoamylase, chymotrypsin or immunoreactive trypsin, when are more tedious in estimation, expensive and at the same time they are also not much better when it comes to diagnosis¹. Whereas with the first attribute, which requires great degree of skill, it is often difficult to reach the correct diagnosis in the "pandora box" relying on itself alone; the second factor is misleading at times. When it comes to the diagnosis of acute pancreatitis, there is no doubt that serum amylase and lipase are the most important tests². Due to their technical simplicity and easy availability combined with high sensitivity, amylase estimation remains the most frequently requested laboratories to be summoned when the diagnosis of AP is considered. When it comes to disadvantages, it has a low specificity.

Lipase estimation has better sensitivity and specificity than amylase, but even it has been reported to be present in normal values in confirmed cases of AP³⁻⁶.

Normal levels of them have been reported in around 19% of total AP cases in one study⁴. An ideal laboratory test in the evaluation of a patient with acute pancreatitis (AP) should, in addition to accurately establishing the diagnosis of AP, provide early assessment of its severity and identify the aetiology⁵. None of the enzyme assays meet those criteria with absolute certainty and have a predictive role in determining the severity or etiology of AP. When it comes to predicting the clinical course of the AP patients with pancreatic enzyme levels, neither they correlate with the severity of the disease nor can they predict disease accurately. Thus both can be misleading. AP is not invariably associated with elevated serum amylase^{6,7}. Multiple factors may contribute to the absence of increased enzymatic levels on admission. They may include either the inability of inflamed pancreases to produce amylase, or return of enzyme levels to normal before hospitalization^{8,9}. Interestingly, AP does not appear to behave differently whether serum amylase normal or elevated, and treatment principles remains the same in either subset, if any¹⁰.

Evaluation has been done regarding the relative merits of different serum pancreatic enzymes, ultrasonography, and computerized tomography in the diagnosis and management of AP. The ultrasonography plays a vital role in the evaluation of the biliary tract in AP. Besides that to estimate the presence and extent of pancreatic necrosis, the contrast-enhanced computed tomography (CECT) is quite useful¹¹. It promptly helps to categorise the high risk patients who are prone to systemic and local complications. Though the utility of the CECT in routine cases of acute pancreatitis—where enzyme elevations are low—may be helpful but its use in clinically mild cases is questionable. Every patient who poses a diagnostic dilemma or is substantially ill must get a CECT done. A normal CT in this group of patients virtually excludes the diagnosis of clinically severe acute pancreatitis.

In the last decade or so, lot of research has been done to detect newer serological and urinary markers which have better diagnostic and prognostic values. Serum C-reactive protein at 48 hours, urinary trypsinogen activation peptides within 12-24 hours of onset¹⁴, serum levels of interleukins 6 and 8 are the areas attracting latest interest to predict severity and prognosis¹⁵.

CONCLUSION

Serum estimation of amylase and lipase helps in diagnosis making of most AP cases due to their simplicity and relative ease in measurement. But, AP is not always associated with increased levels of serum amylase and lipase. These markers can be misleading. If the clinical diagnosis of AP is strongly suspected while the enzyme levels are in normal range, CECT should be performed without delay.

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