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Original Research Article

EVALUATION OF PREVALENCE OF CARDIAC DYSFUNCTION AND ITS ASSOCIATION WITH OUTCOME IN ACUTE PANCREATITIS PATIENTS

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Abstract

Background: Acute pancreatitis (AP) is defined as an acute inflammatory process of the pancreas with variable involvement of other regional tissue or remote organ system. Because of the frequent emergency, multimodality presentation, difficult preoperative diagnosis and management of complication, this challenging subject was taken up and study had planned to observe the prevalence of cardiac dysfunction and its association with outcome in AP. **Aims and Objectives:** To study the prevalence and association of cardiac dysfunctions with outcome in patients of acute pancreatitis. **Material and Methods:** Eligible 100 acute pancreatitis patients were enrolled according to the inclusion and exclusion criteria. Cardiovascular function was assessed by S.CK-MB, ECG and 2DEcho. Patients were managed and followed up during hospital stay for their outcome with CTSI (CT Severity index). **Results:** 96% patients were male and 80% were from 31-50 years of age group. 93% of patients had CTSI score of <6 (mild to moderate AP) while 7% cases had CTSI score >7 (severe pancreatitis). Mean S. CK MB was found to be 33.52 ±19.51 IU/L and in patients with severe pancreatitis, it was 52.57±26.97 IU/L. Sensitivity, specificity and positive predictive value (PPV) of positive S. CK MB value (>25 IU/ml) were 85.71%, 51.61% and 11.76%, respectively, in severe AP cases. Abnormal ECG changes were seen in 42% patients and all 42% patients had Sinus tachycardia. ST depression, QT prolongation, ST elevation and T wave inversion found in 42%, 33%, 21% and 21% cases, respectively. All 7 patients with severe AP have sinus tachycardia and QT prolongation, while 3 have ST depression, 2 have T inversion, 1 have ST-T changes and 1 have ST elevation. 35 patients with mild to moderate AP also have abnormal ECG findings. Sensitivity, specificity and PPV of abnormal ECG findings were 100%, 62.37% and 16.66%, respectively in severe AP cases. 20 out of 100 patient (20%) have abnormal 2D ECHO findings and in 12 out of these 20 have right wall motion abnormality (RWMA) with EF >50% with diastolic dysfunction and remaining 8 have RWMA with EF <50% with diastolic dysfunction. In 7 patients with severe AP, 4 patients have abnormal 2D ECHO findings in which 2 patients have RWMA with EF 56% with diastolic dysfunction grade 1, 1 patient have RWMA with EF 49% with diastolic dysfunction grade 2 and 1 have RWMA with EF 46% with diastolic dysfunction grade 2. 16 patients with mild to moderate AP also have abnormal 2D ECHO finding in which 10 patients have RWMA with EF 53% with diastolic dysfunction grade 1 and 6 patients have RWMA with EF 46% with diastolic dysfunction grade 2. Sensitivity, specificity and PPV of abnormal 2D ECHO findings were 57.14%, 82.14% and 20%, respectively, in severe AP cases. **Conclusion:** In our study association of high S.CRP and S. CK MB levels with severe acute pancreatitis had high sensitivity. Association of 2D ECHO abnormalities with severe AP was highly specific. Association of ECG changes with severe AP had 100% sensitivity. Most common ECG abnormality was sinus tachycardia.

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INTRODUCTION

Acute pancreatitis (AP) is defined as an acute inflammatory process of the pancreas with variable involvement of other regional tissue or remote organ system. The revised Atlanta classification define cardiovascular failure (CVSF) in acute pancreatitis as systolic blood pressure of <90 mmHg and no response to fluid therapy. Apart from hypotension, the cardiovascular manifestations in AP include cardiac rhythm disturbances, hyper-dynamic state with increased cardiac index, decrease myocardial contractility and low peripheral vascular resistance. Heart Ultra structural changes in experimental AP show microcirculation impairment with interstitial edema and cardiomyocyte hypoxia, intracellular edema and hypertrophy of cardiomycyte and collagenisation of myocardium stroma. Previously it was thought that a myocardial depressant factor may be responsible for impaired myocardial function and CVSF in AP. However, CVSF now is accepted to be a part of systemic inflammatory response secondary to cytokine Storm. Among the cardiovascular abnormalities studied in AP, the commonest are ECG changes, Echocardiographic changes and variable S. CK MB values. ECG changes are known to occur in 50% of AP patients and that include sinus tachycardia, atrial flutter and fibrillation, bundle branch block and ST-T wave changes. The changes are thought to be due to electrolyte disturbances, reflex mediated by the Vagus nerve, coronary vasospasm or myonecrosis due to the release of pancreatic proteolytic enzymes. Some reports show that cardiac specific enzyme (S.CK MB) being elevated in AP. Thandassery et al reported that 4% of their patients had a mild elevation of S. CP MB. But there is no prospective data on this marker of myocardial injury in consecutive patient of AP across all grades of severity. On echocardiography in patients with AP there result have been variable. While some study have reported no difference in left ventricular function in patients with AP as compared to control. Others have noted diastolic dysfunctions. Because of the frequent emergency, multimodality presentation, difficult preoperative diagnosis and management of complication, this challenging subject was taken up and study had planned to observe the prevalence of cardiac dysfunction and its association with outcome in AP.

Aims and Objectives: to study the prevalence and association of cardiac dysfunctions with outcome in patients of acute pancreatitis.

MATERIALS AND METHODS

The study was conducted in the department of medicine and gastroenterology after approval from institutional ethical committee of JLN Medical College and Hospital Ajmer. After taking informed consent from patients, study was performed in 100 patients those admitted in various medical and gastroenterology wards.

Inclusion Criteria

Acute pancreatitis as defined by clinical symptoms and elevated serum amylase or lipase (more than thrice the upper limit of normal range) or imaging evidence of acute pancreatitis 2. Patients presenting within 7 days of onset of pain. 3. Age more than 18 year.

Exclusion Criteria

1 Patients with severe pre-existing diseases like coronary artery disease, congestive heart failure of any etiology, chronic kidney disease, chronic liver disease. 2 Patients with any evidence of chronic pancreatitis any previous episode of acute pancreatitis.

Qualifying patients were undergone detailed history, clinical examination, biochemical and radiological investigation. Following investigation were performed in all patients e.g. CBC, blood sugar, blood urea, S. Creatinine, S. Sodium and potassium, S. Calcium and phosphorus, LFT, Lipid profile, S. amylase and lipase, urine complete, ECG, USG abdomen. Special investigations: 1. S. CK MB was done on admission and on after 3 days. 2 2D ECHO 3 TMT (if required) 4 CECT abdomen was done on or after 3 days to assess the severity of AP. severity of AP was defined according to CTSI (CT Severity index). Acute physiology and chronic health evaluation II (APACHE II) score was derived at admission.

RESULTS

In our study 80% patients were from 31-50 years of age group and 96% of patients were male. 93% of patients had CTSI score of <6 (mild to moderate AP) while 7% cases had score >7 (severe AP). 76% have APACHE II score between 4 to 9 and 7% have score >13. All the 7 patients those have APACHE II score > 13 also have CTSI score >7. [Table 1]

In all studied patients, S. CK MB values were ranging between 10- 96 IU/ml (Mean + SD 33.52 +19.51). In severe AP cases range was 24 -96 IU/ ml (52.57 +26.97) while in mild to moderate AP cases it was 18- 88 IU/ ml (32.08 +18.23). 6 out of 7 (86%) with CTSI score >7 (severe AP) have positive S. CK MB value i.e. >25 IU/ml. However, 45 patients with CTSI score <6 (mild to moderate AP) also have positive S. CK MB value >25 IU/ml. Sensitivity, specificity and positive predictive value (PPV) of positive S. CK MB value (>25 IU/ml) were 85.71%, 51.61% and 11.76%, respectively in severe AP cases. [Table 2]

In our study 42 patients out of 100 (42%) have abnormal ECG findings and among these 42 patients, all 42 patients have sinus tachycardia. ST depression seen in 42%, QT prolongation in 33%, ST elevation and T inversion seen in 21% each. All 7 patients with severe AP have sinus tachycardia and QT prolongation, while 3 have ST depression, 2 have T inversion, 1 have ST-T changes and 1 have ST

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elevation. 35 patients with mild to moderate AP also have abnormal ECG findings. Sensitivity, specificity and PPV of abnormal ECG findings were 100%, 62.37% and 16.66%, respectively in severe AP cases. [Table 3]

In our study 20 out of 100 patient (20%) have abnormal 2D ECHO findings and in 12 out of these 20 have right wall motion abnormality (RWMA) with EF >50% with diastolic dysfunction and remaining 8 have RWMA with EF <50% with diastolic dysfunction. In 7 patients with severe AP, 4 patients have abnormal 2D ECHO findings in which 2 patients have RWMA with EF 56% with diastolic dysfunction grade 1, 1 patient have RWMA with EF 49% with diastolic dysfunction grade 2 and 1 have RWMA with EF 46% with diastolic dysfunction grade 2. 16 patients with mild to moderate AP also have abnormal 2D ECHO finding in which 10 patients have RWMA with EF 53% with diastolic dysfunction grade 1 and 6 patients have RWMA with EF 46% with diastolic dysfunction grade 2. Sensitivity, specificity and PPV of abnormal 2D ECHO findings were 57.14%, 82.14% and 20%, respectively, in severe AP cases. [Table 4]

Table 1: Correlation of S. CK MB with CTSI score

CTSI score	S. CK MB values (in IU/ml)		Mean	Standard deviation
	Minimum	maximum		
≤6	10	88	32.08	18.23
≥7	24	96	52.57	26.97

Table 2: Correlation of ECG findings with CTSI score

CTSI score	ECG findings	
	Normal [N(%)]	Abnormal [N(%)]
≤6	58 (62.4%)	35 (37.6%)
≥7	0 (0%)	7 (100%)

Table 3: Correlation of 2D ECHO findings with CTSI score

CTSI score	2D ECHO findings	
	Normal [N(%)]	Abnormal [N(%)]
<6	77 (82.8%)	16 (17.2%)
>7	80 (82%)	20 (20%)

Table 4: Sensitivity, Specificity and Positive predictive value (PPV) of S. CK MB, ECG findings and 2D ECHO findings to predict severity of acute pancreatitis

Parameter	Sensitivity (%)	Specificity (%)	PPV (%)
S. CK MB	85.71	51.61	11.76
ECG findings	100	62.37	16.66
2D ECHO findings	57.14	82.14	20

DISCUSSION

AP is the most frequent gastrointestinal cause for hospitalisation and one of the leading cause of in hospital death. Because of the frequent emergency, multimodality presentation, difficult pre-operative diagnosis and management of complication, this challenging subject was taken up for the present study in 100 patients admitted in various medical and gastro ward.

In present study 80% of the cases were in the age group of 31- 50 years. In the study conducted by Prasad et al, Juthika Abhijeet Deharar kar et al and Lee and papachirastu reported 70%, 32% and 42% of their study population, respectively being from 31-40-year age group. Surjit Kumar Das et al reported 63% of cases from 22-39-year age group.

In present study we observed that maximum patients (96%) were male. Surjit Kumar Das et al and Jitin Yadav et al also reported male dominance in 96% and 70%, respectively. This male predominance could be because alcohol was observed to be the main etiology in our study. However, Yilmaz Bilgic et al and Y I Kibar et al reported female predominance in 66% and 65% respectively that might be because of gallstone being the main etiology in their study.

In present study 7% patients had CTSI score >7 (severe AP). Prasad et al also found that 8% patients had CTSI score >7. Viswanath Sahu et al, Thomas Bollen et al and shetalgonapati et al observed that 15-20%, 15-20% and 17% patients, respectively had CTSI score >7.

In present study 7% patients had APACHE II score >13 and all these 7 patients also had CTSI score >7 (severe AP). In the studies conducted by Prasad et al and gurleyik et al, 15% and 12% respectively had APACHE II score >8.

In our study, the mean S.CRP level was 33.58 mg/L with SD 32.48 and range being from 4.92- 138 mg/dl. Among patients with severe AP, minimum and maximum values of serum CRP were 118 and 138 mg/dl, respectively and mean + SD was 125.29 + 6.84. This showed that the patients with severe AP had higher serum CRP value and their association was found to be significant ($P < 0.001$). Bingzhao et al and parmar et al also reported statistically significant association ($P < 0.05$). Kaplan et al, Pizzelli et al, Trivikaram et al, Vijay Kumar et al and Joshi et al also reported higher serum CRP level in severe AP.

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In all studied patients, S. CK MB values were ranging between 10- 96 IU/ml (Mean + SD 33.52 +19.51). In severe AP cases range was 24 -96 IU/ ml (52.57 +26.97) while in mild to moderate AP cases it was 18- 88 IU/ ml (32.08 +18.23). Prasad et al, Albrecht and low et al and Korantzopoulou et al reported that S.CK MB levels gets elevated in severe AP. On the other hand, Pizzelli et al revealed no association between S. CK MB and severity of AP. Sensitivity, specificity and positive predictive value (PPV) of positive S. CK MB value (>25 IU/ml) were 85.71%, 51.61% and 11.76%, respectively in severe AP cases. This in line with Bingzhao et al (sensitivity 83.7% and specificity 66.7%) and Ankit Mahajan et al (sensitivity 83% and specificity 66%).

In our study 42 patients out of 100 (42%) have abnormal ECG findings and among these 42 patients, all 42 patients have sinus tachycardia. ST depression seen in 42%, QT prolongation in 33%, ST elevation and T inversion seen in 21% each. This is in line with Ankit Mahajan et al and Prasad et al. Buch et al in a prospective study of 54 patients showed that 31 (57%) patients had transient ECG abnormalities. Most common ECG changes in their study were non specific T wave changes in 25 cases and accelerated atrial or nodal rhythm in 8 cases. Authors implicated that these ECG changes were mostly due to underlying ischemic heart disease exacerbated by the stress of AP and or as a consequence of an imbalance of the autonomous nervous system. Other study by Rubio Tapia et al suggested that this is attributed by electrolyte alteration. Pizzelli et al reported that the most frequently observed abnormality was ST segment depression with T wave inversion. Hsu et al, RO et al Yu and Regart et al revealed that AP might actually be complicated by myocardial infarction secondary to hypovolemia, cogulation abnormalities or coronary arteries spasm.

All 7 patients with severe AP have sinus tachycardia and QT prolongation, while 3 have ST depression, 2 have T inversion, 1 have ST-T changes and 1 have ST elevation. 35 patients with mild to moderate AP also have abnormal ECG findings. Sensitivity, specificity and PPV of abnormal ECG findings were 100%, 62.37% and 16.66%, respectively in severe AP cases. This is in line with Ankit Mahajan et al, Atul et al and Nadkarni et al. In cases of severe pancreatitis this abnormal ECE findings were present with Sensitivity of 100% and specificity of 62.37%.

In our study 20 out of 100 patient (20%) have abnormal 2D ECHO findings and in 12 out of these 20 have right wall motion abnormality (RWMA) with EF >50% with diastolic dysfunction and remaining 8 have RWMA with EF <50% with diastolic dysfunction. This is in line with Gyongyosi et al, Ankit et al and Pizzelli et al, while variyam and Shah did not found any abnormal finding in 2D ECHO.

In 7 patients with severe AP, 4 patients have abnormal 2D ECHO findings in which 2 patients have RWMA with EF 56% with diastolic dysfunction grade 1, 1 patient have RWMA with EF 49% with diastolic dysfunction grade 2 and 1 have RWMA with EF 46% with diastolic dysfunction grade 2. 16 patients with mild to moderate AP also have abnormal 2D ECHO finding in which 10 patients have RWMA with EF 53% with diastolic dysfunction grade 1 and 6 patients have RWMA with EF 46% with diastolic dysfunction grade 2. Sensitivity, specificity and PPV of abnormal 2D ECHO findings were 57.14%, 82.14% and 20%, respectively, in severe AP cases. This association between severe AP and 2D ECHO findings was found to be statically significant. This in line with Atul et al and Nadkarni et al.

CONCLUSION

In our study association of high S.CRP and S. CK MB levels with severe acute pancreatitis had high sensitivity. Association of 2D ECHO abnormalities with severe AP was highly specific. Association of ECG changes with severe AP had 100% sensitivity. Most common ECG abnormality was sinus tachycardia.

Limitation

Limitation of the study is that thought to be excluded patients with documented history of Cardiac disease, the pre-orbit cardiac status of enrolled patients was not reliable known follow up cardiac evaluation have helped us better to know the Dynamics of such changes if you cases of diastolic dysfunction could have been contributed by patients with hypertension as they were not excluded and lastly the sample size would have been increase.

REFERENCES

1. Bradley TH, Morgan DE. Current concepts: Acute necrotizing pancreatitis. N Engl J Med 1999; 340:18 :141 2- 17.
2. Banks PA, Freeman ML. Practice Parameters Committee of the American College of Gastroenterology. Practice guidelines in acute pancreatitis. Am J Gastroenterol 2006; 101:2379-400.
3. Muniraj, Thiruvengadam G, Mahesh T, Sudha R, Karthik R, Seema D et al. (2012). Acute Pancreatitis. Disease-a-month: DM. 58. 98-144.
4. Lankisch, P. G., Apte, M., & Banks, P.A. (2015). Acute pancreatitis. The Lancet, 3 86(9988), 85- 96.
5. Banks PA, Bollen TL, Dervenis C, Gooszen HG, Johnson CD, Sarr MG et al. Classification of acute pancreatitis-2012: revision of the Atlanta classification and definitions by international consensus. Gut. 2013 Jan 1; 62(1):102-11
6. Pezzilli R, Barakat B, Billi P, Bertaccini B.o. Electrocardiographic abnormalities in acute pancreatitis Eur J E . M d · metg e .1999; 6:27- 9.
7. Ro TK, Lang RM, Ward RP. Acute pancreatitis mimicking myocardial infarction: evaluation with myocardial contrast Echocardiography. J Am Soc Echocardiogr. 2004; 17:387- 90.
8. Gyongyosi M, Takacs T, Czako L. Noninvasive monitoring of hemodynamic changes in acute pancreatitis in rabbits. Dig Dis Sci. 1997; 42:955--61.
9. Prasada R, Dhaka N, Bahl A, Yadav T. D., & Kochhar R. Prevalence of cardiovascular dysfunction and its association with outcome in patients with acute pancreatitis. Indian Journal of Gastroenterology 2018, 37(2), 113-119.
10. Thandassery RB, Choudhary N, Bahl A, Kochhar R. Characterization of cardiac dysfunction by echocardiography in early severe acute pancreatitis. Pancreas, 2017; 46:626-30.

ISSN (O): 2687-5365; ISSN (P): 2753-6556

11. Van Westerloo DJ, Bruno MJ, van der Poll T: New insights into the pathophysiology and severity assessment of acute pancreatitis. In: Vincent JL (ed.), Yearbook of Intensive Care and Emergency Medicine. Berlin, Springer, 2003; 823- 87
12. Marx C: Adreno-cortical insufficiency: An early step in the pathogenesis of severe acute pancrentitis and development of necrosis? Do we have a new treatment option? Crit Care Med, 2006; 34: 1269 18.
13. Yadav D, Lowenfels AB: Trends in the epidemiology of the first attack of acute pancreatitis: a systematic review. Pancreas, 2006; 33: 323- 30
14. Owyang C: Pancreatitis. In: Goldman L (ed.), Cecil Medicine, 23rd ed. Saunders, An Imprint of Elsevier, 2007. Accessed through www. mdconsult. Com
15. Carroll JK, Herrick B, Gipson T et al: Acute pancreatitis: Diagnosis, prognosis, and treatment. Am Fam Physician, 2007; 75: 1513-20
16. Yadav D and A. B. Lowenfels. "The epidemiology of pancreatitis and pancreatic cancer". Gastroenterology, vol. 144, no. 6, pp. 1252-1261, 2013.
17. Peery F, E. S. Dellon, J. Lund et al., "Burden of gastrointestinal disease in the United States: 2012 update," Gastroenterology, vol. 143, no. 5, pp. 1179-1187.e3, 2012
18. Bilgic Y, Akbulut S, Kutlu 0, Colak C, Yilmaz C, Secldn Y et al. Could platelet indices be prognostic biomarkers for mild or severe acute pancreatitis. Int J Clin Exp Med. 2016 Jan 1; 9(7):13918-25.
19. Deherkar JA, Pandey A, Deshmukh S. C-reactive protein levels in acute pancreatitis and its clinical significance. Int Surg J 2019; 6:3328-34.
20. 20. Lee PJ, Papachristou GI. New insights into acute pancreatitis. Nat Rev Gastroenterol Hepatol 2019; 16:479-96.
21. Das SK, Das S. Clinical profile of patients with acute pancreatitis in a tertiary care centre in Tripura: A retrospective study. Asian Journal of Medical Sciences. 2020 Nov 1; 11 (6):96-l OO.
22. Jitin Yadav, Sanjay kumar yadav, satish kumar. Predicting morbidity and mortality in acute pancreatitis in an Indian population a comparative study of the BISAP score Ransons' score and CT severity index.
23. Kibar Yi, Albayrak F, Arabul M, Dursun H, Albayrak Y, Ozturk Y. Resistin: new serum marker for predicting severity of acute pancreatitis. Journal of International Medical Research. 2016 Apr; 44(2):328-37.
24. Sahu B, Abbey P, Anand R, Kumar A, Tomer S, Malik E. Severity assessment of acute pancreatitis using CT severity index and modified CT severity index: Correlation with clinical outcomes and severity grading as per the Revised Atlanta Classification. The Indian journal of radiology & imaging. 2017 Apr; 27(2):152.
25. Bollen TL, Singh VK, Maurer R, Repas K, van Es HW, Banks PA, et al. Comparative evaluation of the modified CT severity index and CT severity index in assessing severity of acute pancreatitis. Am J Roentgenol. 2011; 197:386-92.
26. Sahu B, Abbey P, Anand R, Kumar A, Tomer S, Malik E. Severity assessment of acute pancreatitis using CT severity index and modified CT severity index: Correlation with clinical outcomes and severity grading as per the Revised Atlanta Classification. Indian J Radio Imaging. 2017 Apr-Jun; 27(2):152-160. doi: 10.4103/ijri.IJRI_300_16. PMID: 28744075; PMCID: PMC5510312
27. Gurleyik G, Cirpici OZ, Aktekin A, Saglam A. The value of Ranson and APACHE II score and serum level of C-reactive protein in early diagnosis of severity of acute pancreatitis. Ulus Travma Acil Cerrahi Derg. 2004; 10(2):83-8.
28. Parmar D, Parihar S. To evaluation of C-reactive protein in acute pancreatitis by semi quantitative method and its correlation with prognosis: a hospital based study. Int J Med Res Prof. 2015; 1(3):206-8.
29. Zhao B, Sun S, Wang Y, Zhu H, Ni T, Qi X et al. Cardiac indicator CK MB might be a predictive marker for severity and organ failure development of acute pancreatitis. Ann Transl Med 2020.
30. Kaplan M, Ates I, Oztas E, Yuksel M, Akpinar MY, Coskun 0. A new marker to determine prognosis of acute pancreatitis: PLR and NLR combination. J Med Biochem. 2018; 37:21-30.
31. Pezzilli R, Billi P, Miniero R, Fiocchi M, Cappelletti 0, Maria A. Serum interleukin-6, interleukin-8 and beta 2microglobulin in early assessment of severity of acute pancreatitis: comparison with serum C reactive protein. Dig Dis Sci. 1995; 40(11):2341-8.
32. Albrecht CA, Laws FA. ST-segment elevation pattern of acute myocardial infarction induced by acute pancreatitis. Cardiel Rev. 2003; 11: 147- 51.
33. Mahajan A, Vijant Singh. Study of ECG changes and Echocardiographic changes in patients of acute pancreatitis. Med Pulse International Journal of Medicine. September 2019; 11(3): 58- 62
34. Nadkarni N, Bhasin DK, Rana SS. Diastolic dysfunction, prolonged QTc interval and pericardial effusion as predictors of mortality in acute pancreatitis. J Gastroenterol Hepatol. 2012; 27: 1576- 80
35. Buch J, Buch A, Sclunidt A. Transient ECG changes during acute attacks of pancreatitis. Acta Cardiol. 1980; 35:381-90
36. 36. Atul et al. Cardiac involvement in acute pancreatitis and its effect on morbidity and mortality.
37. Rubio-Tapia A, Garcia-Leiva J, Asensio-Lafuente E, Robles-Diaz G, Vargas-Vorackova F. Electrocardiographic abnormalities in patients with acute pancreatitis. J Clin Gastroenterol. 2005; 39:815-8
38. Hsu PC, Lin TH, Su HM, Lin ZY, Lai WT, Sheu SH. Acute necrotizing pancreatitis complicated with ST elevation acute myocardial infarction: a case report and literature review. Kaohsiung J Med Sci. 2010; 26:200-5.
39. Yu AC, Riegert-Johnson DL. A case of acute pancreatitis presenting with electrocardiographic signs of acute myocardial infarction. Pancreatology. 2003; 3 :515- 7
40. Gyongyosi M, Takacs T, Czako L. Noninvasive monitoring of hemodynamic changes in acute pancreatitis in rabbits. Dig Dis Sci 1997; 42:955-61
41. Variyam EP, Shah A. Pericardial effusion and left ventricular function in patients with acute alcoholic pancreatitis. Arch Intern Med. 1987; 147:923-5.

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