EVALUATION OF THE ROLE OF D-DIMER IN ASSESSMENT OF SEVERITY AND OUTCOME OF ACUTE PANCREATITIS IN CHILDREN

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ABSTRACT:

Background: *Acute pancreatitis (AP) is the most common pathological entity affecting the pancreas in children. Multi-organ failure and/or pancreatic necrosis, can result from a severe episode of AP. Early severity stratification can result in aggressive treatment and prevent the development of persistent organ damage and multiple organ dysfunction. Multiple studies have evaluated the relationship between AP severity and d-dimer but most of these studies involved only adult population.*

Aim of work: *The aim of the present study is to find out the value of D-dimer as a marker of severity in acute pancreatitis.*

Patients and methods: *In this prospective cohort study 24 Children and adolescents were recruited with the diagnosis of AP among those attending gastroenterology unit Children's hospital, Ain Shams University. All patient's clinical data were recorded, and they have all withdrawn serum D-dimer in the 1st, 3rd, and 7th days of admission together with other laboratory markers, abdominal ultrasound, and CT scan. and those were correlated with the outcome of cases. NASPGHAN pancreas committee criteria were used to classify severity into mild, and moderately severe to severe disease.*

Results: *Age of patients ranged from 4-15 years; Eight patients developed moderately severe to severe disease while16 patients had mild disease. D-dimer levels were higher among patients with severe disease, yet this was not statistically significant, while serum calcium level was significantly lower among moderately severe to severe cases.*

Conclusion: *D-dimer, although high in acute pancreatitis cannot be relied upon as a marker of severity or prognosis in pediatrics.*

Key words*: acute pancreatitis, D-dimer, pediatrics, prognosis.*

INTRODUCTION:

Acute pancreatitis is the most common pathological entity affecting the pancreas in children[(1)].

Although it is a well-known disease concerning its clinical and treatment aspects in the adult population, most of the recommendations in pediatrics are derived from studies involving only adults[(2)].

Pancreatitis in children has been diagnosed more frequently in the past few decades, possibly due to an increase in health care provider awareness and etiologies of pancreatitis being identified, as well as more thorough evaluations of children[(3)].

Alcohol and gallstones account for more than 60% of cases of acute pancreatitis in adults. However, the etiology in children is

often drugs, infections, trauma, and anatomic anomalies such as choledochal cysts and abnormal union of the pancreatic-obiliary junction [4].

The INSPPIRE initiative has established that a diagnosis of AP requires two of the following three criteria: (1) characteristic abdominal pain (epigastric or right upper quadrant with or without radiation to the back), (2) serum amylase and/or lipase values 3 or more times the upper limit of normal, and (3) imaging findings (ultrasound, magnetic resonance imaging, or computed tomography [CT]) compatible with AP[5].

Significant morbidity and mortality due to numerous local and systemic complications, an intense inflammatory response that may progress to multiorgan failure and/or pancreatic necrosis, can result from a severe episode of AP [6].

Early severity stratification can result in aggressive treatment and prevent the development of persistent organ damage and multiple organ dysfunction, which are the two primary causes of mortality in patients with acute pancreatitis [7].

Several biochemical parameters, contrast enhanced computed tomography and multiple clinico-biochemical scores have been used to assess the severity of acute pancreatitis. An ideal prognostic method should be simple, cheap, routinely available, and highly accurate [8].

Multiple studies have evaluated the relationship between AP severity and d-dimer but most of these studies involved only adult population, results indicated that d-dimer is a good initial marker of severity with peak levels seen by day four [9].

Aim of work: The aim of the present study is to find out the value of D-dimer as a marker of severity in acute pancreatitis in pediatrics.

PATIENTS AND METHODS:

This prospective cohort study involved 24 Children and adolescent presented to the gastroenterology department Children's hospital Ain Shams University diagnosed as acute pancreatitis according to the INSPIRE criteria.

The study protocol was approved by the Ethics Committee Number…., Faculty of Medicine, Ain Shams University, and comply with the regulations of the Egyptian Ministry of Higher Education and Helsinki declaration, 1964. and consent from parents or guardians of children in the study was obtained after clear explanation of the study objectives.

Full medical history for all patients with laying stress on the character of abdominal pain, its location, severity, radiation, onset, course, duration, history of back pain, frequency of vomiting and abdominal distention. Medication history of chemo-therapy and others, and finally any history of blunt trauma to the abdomen, recent surgical procedures, and gall stones.

In addition to full clinical evaluation of all patients, they were subjected to laboratory investigations on admission including: arterial blood gases, serum calcium, creatinine, complete blood count (CBC), liver enzymes, total bilirubin ,serum amylase, serum lipase, lipid profile ,D-dimer which was repeated again at 3rd and 7th day. D-dimer was measured by VIDAS® D-Dimer Exclusion II™ (DEX2).

Imaging done in the form of abdominal ultrasound and CT scan. Grading of CT was done based on balthazar scoring system criteria [1].

Patients were divided into 2 groups according to severity 66.7% of patients were suffering from mild disease, while 33.3% of patients had severe disease according to NASPGHAN pancreas committee criteria.

RESULTS:

This study included 24 patients, age of patients ranged from 4-15 years old, females constituted 66.7% of patients, pain was present in 100% of patients of whom radiation to the back occurred in 66.7% of patients, diarrhea in 4.2% and surprisingly low grade fever in 54.2% of patients, the duration of the acute episode of acute pancreatitis ranged from 17 days to 7 weeks, while duration of hospital admission ranged from 1day to 2 months.

The final etiology of pancreatitis was heterogenous where 45.8% were idiopathic ,12.5% were related to abdominal trauma ,25% (6 patients) related to medications valproic acid, L-asparaginase and NSAID's. Calculous cholecystitis occurred in 3 patients (12.5%), only one patient suffered from autoimmune pancreatitis.

Patients were divided into 2 groups according to severity 33,3% (n=8) of patients had severe disease (group A), 3 patients of that group had severe disease (persistent end organ failure) while 5 patients had moderately severe disease. Group B had 66.7% (n=16) of patients who were suffering from mild disease.

Table 1 shows comparison between both groups as regarding clinical data and radiological findings, and table 2 shows comparison of laboratory data.

This study found that the etiology of AP was mainly drug-related (37.5%) in group (A), followed by idiopathic (25)%, then trauma (12.5%) ,while in the mild to moderate group the most common etiological cause was idiopathic (56.3%), drugs (18.8%) and then trauma (12.5%), there was no statistical significance between both groups.

Interestingly, diabetes mellitus has occurred in 2 patients (25%) in group A, while it did not happen in the mild pancreatitis group (p value=**0.037)**

Three patients from group A (37.5%) had persistent end organ failure and one of those patients had died.

Intolerance to oral fluid intake during hospital stay ranged from 2-10 days in group A, while it took only 2-6 days in mild pancreatitis group which showed statistical significance (P value=0.020)

There was a positive correlation between serum lipase on admission and D-dimer level on day 3as shown in figure 1 (P value=0.048 and r =0.417)

The ROC curve shown in Fig 2 indicates that the best cut off value for calcium level to differentiate between moderately severe/severe cases and mild cases was found to be ≤8.5 mg/dl with sensitivity of 62.5%, specificity of 93.5%, positive predictive value (PPV) of 83.3%, negative predictive value (NPV) of 83.3% and area under the curve (AUC) of 73.0%

Statistical analysis:

Statistical terms such as range, mean ± SD were used for quantitative information. Inferential analysis was done for quantitative variables using independent *t* test in cases of two independent groups with normal distributed data. In qualitative information the level of significance estimated by *p* value less than 0.050 is significant, otherwise it is non- significant.

Table 1: Comparison between 2 groups regarding clinical and radiological findings

		Group A: Moderately severe/severe pancreatitis	**Group B: Mild pancreatitis**	**Test value**	**P-value**
		No. = 8	**No. = 16**		
Gender	Male	2 (25.0%)	6 (37.5%)	0.375	0.540
	Female	6 (75.0%)	10 (62.5%)		
Age	Mean ± SD	9.00 ± 3.21	10.00 ± 3.90	-0.625	0.538
	Range	4 – 13	4 – 15		
Family history of similar condition	Yes	2 (25%)	0 (0.0%)	4.364	**0.037**
	No	6 (75%0	16(100%)		
Duration of symptoms in days before diagnosis	Median (IQR)	22.50 (6.50 – 30)	5.50 (4 – 7)	-2.468	**0.014**
	Range	3 – 32	1 – 8		
Present history					
Onset	Sudden	3(37.5%)	9(56.3%)	0.750	0.386
	Gradual	5(62.5%)	7(43.8%)		
Duration	Days	4(50.0%)	13(81.3%)	2.521	0.112
	Weeks	4(50.0%)	3(18.8%)		
Radiation of pain to the back	No	5 (62.5%)	3 (18.8%)	4.594	**0.014**
	Yes	3 (37.5%)	13 (81.3%)		
Vomiting	Yes	8 (100.0%)	14 (87.5%)	1.091	0.296
	No	0 (0.0%)	2 (12.5%)		
Diarrhea	Yes	1 (12.5%)	0 (0.0%)	2.087	0.149
	No	7 (87.5%)	16 (100.0%)		
Fever	Yes	5 (62.5%)	8 (50.0%)	0.336	0.562
	No	3 (37.5%)	8 (50.0%)		
Jaundice, difficulty of breathing, drowsiness	Yes	6 (75.0%)	5 (31.3%)	4.112	**0.043**
	No	2 (25.0%)	11 (68.8%)		
Examination					
Vital signs					
Heart rate (HR)	Normal	3 (37.5%)	14 (87.5%)	6.454	**0.011**
	Tachycardia	5 (62.5%)	2 (12.5%)		
RR (respiratory rate)	Normal	5 (62.5%)	15 (93.8%)	3.750	0.053
	Tachypnea	3 (37.5%)	1 (6.3%)		
Temperature	Normal	6 (75.0%)	16 (100.0%)	4.364	**0.037**
	High	2 (25.0%)	0 (0.0%)		
Blood pressure	Normal	6 (75%)	16 (100%)	6.281	**0.043**
	Low	2 (25.0%)	0 (0.0%)		
Oxygen Saturation	Mean ± SD	96.38 ± 1.51	96.75 ± 1.13	-0.688	0.499
	Range	95 – 99	95 – 98		
Abdominal examination					
Deep palpation of abdomen	Normal	0 (0.0%)	15(93.75%)	9.170	**0.027**
	Organomegaly	3 (37.5%)	1 (6.25%)		
	Mass	1 (12.5%)	0 (0.0%)		
Imaging					
Abdominal ultrasound	Normal	1 (12.5%)	4 (25.0%)	0.505	0.477
	pelvic fluid collection	7 (87.5%)	12 (75.0%)		
CT	Normal	3 (37.5%)	6 (37.5%)	0.000	1.000
	Abnormal	5 (62.5%)	10 (62.5%)		
CT grade	0	6 (75.0%)	13 (81.3%)	2.526	0.283
	1	2 (25.0%)	1 (6.3%)		
	2	0 (0.0%)	2 (12.5%)		

Table 2 comparison between 2 groups as regarding laboratory investigations:

Laboratory Investigations		**Group A**	**Group B**	**Test value**	**P-value**
		No. = 8	**No. = 16**		
ABG	Normal	6 (75.0%)	16 (100.0%)	4.364	**0.037**
	Metabolic acidosis	2 (25.0%)	0 (0.0%)		
Serum creatinine (mg/dl)	Median (IQR)	0.50 (0.40 – 0.85)	0.50 (0.50 – 0.60)	-0.223	0.823
	Range	0.30 – 2	0.30 – 5.10		
AST (IU/L)	Median (IQR)	59 (24 – 121.50)	27.50 (21.50 – 40.50)	-1.411	0.158
	Range	17 – 303	13 – 124		
ALT (IU/L)	Median (IQR)	64.50 (28 – 264.50)	17.00 (11.00 – 49.00)	-1.565	0.118
	Range	8 – 500	6 – 293		
Serum bilirubin (mg/dl)	Median (IQR)	1.10 (0.50 – 3.35)	0.60 (0.50 – 0.90)	-1.007	0.314
	Range	0.30 – 5.50	0 – 15.60		
Amylase (U/L)					
1st (on admission)	Median (IQR)	914 (174.50 – 1832)	474.50 (228 – 1200.50)	-0.521	0.603
	Range	30 – 2887	150 – 1417		
2nd (after a week)	Median (IQR)	214 (75 – 405)	172.50 (128 – 783)	-0.098	0.922
	Range	4 – 3665	56 – 1000		
Lipase (U/L)					
1st (on admission)	Median (IQR)	297 (100 - 865)	417 (162 - 700)	-0.032	0.974
	Range	18 – 2472	8 – 1540		
2nd (after a week)	Median (IQR)	64 (16- 132)	100 (94 - 148)	-1.278	0.201
	Range	5 – 319	72 – 2000		
Lipid profile					
Serum cholesterol (mg/dL)	Mean ± SD	165.25 ±23.25	170.31 ±37.09	-0.351	0.729
	Range	150 – 219	114 – 274		
Triglycerides (mg/dL)	Mean ± SD	111.25 ±44.92	107.06 ±41.43	0.227	0.822
	Range	70 – 211	31 – 200		
LDL (mg/dL)	Mean ± SD	112.00 ±17.43	116.68 ±29.10	-0.416	0.682
	Range	92 – 149	67 – 183		
HDL (mg/dL)	Mean ± SD	36.75 ±9.11	34.27 ±10.61	0.560	0.582
	Range	25 – 47	18 – 51		
Others					
Serum Ca (mmol/L)	Mean ± SD	8.49 ±1.43	9.45 ±0.63	-2.318	**0.030**
	Range	6.9 – 10.8	8 – 10.2		
CRP (mg/l)	Median (IQR)	6.75 (6 – 18)	34.50 (6 – 121)	-1.426	0.154
	Range	3 – 96	0.20 – 192		
WBCs (10^3/uL)	Median (IQR)	8.10 (6.05 – 12.40)	10.00 (5.45 – 13.75)	-0.398	0.691
	Range	4 – 19	3.40 – 24.80		
Hgb (gm/dL)	Mean ±SD	11.03 ±2.15	11.50 ±1.45	-0.642	0.527
	Range	8 – 14.9	8.9 – 13.7		
PLTs (10^3/uL)	Mean ±SD	246.25 ±135.56	334.69 ±151.74	-1.391	0.178
	Range	45 – 499	30 – 604		

D-Dimer (ug/l)					
1st day	Median (IQR)	1750 (260 – 7100)	640 (536 – 1055)	-0.399	0.690
	Range	200 – 10000	101 – 10000		
3rd day	Median (IQR)	655 (575 – 4829)	2077 (591 – 3300)	0.000	1.000
	Range	250 – 7000	250 – 5000		
7th day	Median (IQR)	839.50 (250 – 3201)	435 (200 – 556.50)	-1.204	0.229
	Range	200 – 6500	200 – 1480		
1st day Vs 3rd day	Median (IQR)	-18.36 (-33.50 – 146.67)	185.47 (-47.53 – 485.22)	-1.286	0.198
	Range	-81.25 – 213.64	-79.93 – 2494.06		
1st day Vs 7th day	Median (IQR)	-9.85 (-53.21 – 25.03)	-42.05 (-71.03 – -4.17)	-1.102	0.270
	Range	-76 – 59.67	-89.66 – 457.43		

Figure (1): Correlation between serum D-dimer on third day and serum lipase on admission.

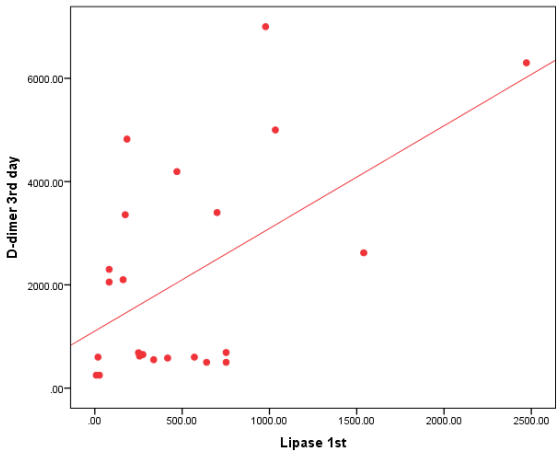
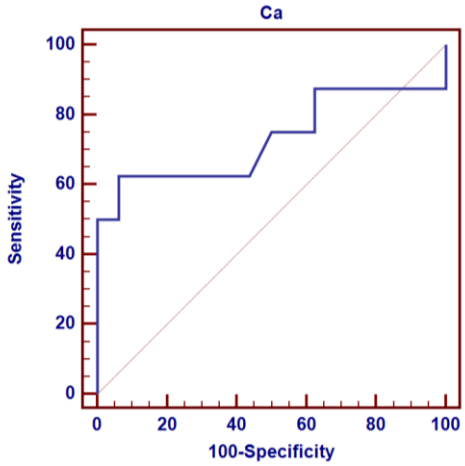


Figure (2): Receiver operating characteristics (ROC) curve for serum calcium level to differentiate between moderately severe to severe and mild cases.



DISCUSSION:

Our study that there is significant difference between the 2 groups in the duration before diagnosis (P-value 0.014), Median (IQR) 22.50 (6.50 – 30) days in the moderately severe to severe group. Median (IQR) 5.50 (4 – 7) days in the mild pancreatitis group.

This finding show that early severity stratification can result in aggressive treatment and prevent the development of persistent organ damage and multiple organ dysfunction, which are the two primary causes of mortality in patients with acute pancreatitis [(9).]

Also, the late diagnosis may be due to diagnosis not in mind during evaluation, this need increase in the awareness of the disease. Due to increased cases all over the world (US, United Kingdom) [(5)].

Epigastric pain radiating to the back was found in 66.7% of our patients. NASPGHAN pancreas committee stated that the ‘‘classic’’ presentation of epigastric pain radiating to the back occurs in only 1.6%-5.6% of pediatric patients [(10)].

In this study all patients underwent abdominal ultrasound and CT abdomen. Abnormal ultrasound was seen in 19.7% while, 62.5% of patients had abnormal CT abdomen in the form of pancreatic edema, necrosis, and pseudocyst.

Duration of hospital stay ranged from 1 to 60 days (median 9 days), this comes in agreement with **Grzybowska-Chlebowczyk et al. (2017)** who found that the hospitalization period ranged from 4 to 48 day (average 13.8 days) [(11)].

Our study found that 2 patients (25%) of group A developed diabetes mellitus (DM) after the acute pancreatitis attack resolved. **Das et al.,2014** concluded that patients with AP often develop DM after discharge from hospital and have more than two fold increased risk of DM over 5 years further studies needed to determine the optimal strategy for detection and if the risk of developing DM can be reduced [(12)].

Ibrahim et al in 2011 in his study done on 50 pediatric patients, forty-eight children underwent abdominal US, 19 of these were read as abnormal, and only two of these patients were found to have abnormal CT scans at the same admission [(13)].

Our study found that only one patient had disturbed lipid profile (high cholesterol, LDL, triglycerides, and low HDL), while 14 patient had low levels of HDL**. Hong et al. (2017)** also found no relation between HDL, LDL, and severity of AP. On the contrary, **Khan et al. (2013)** concluded that low serum cholesterol, HDL and LDL were associated with severe AP[(14&15)].

Serum calcium level ranged between 6.9 to 10.8mg/dl, which was significantly lower among the moderately severe to severe pancreatitis patients. **Pokharel et al., 2017** study concluded that low serum calcium can predict severe acute pancreatitis [(16)]. **Edakkepuram et al**. **(2017)** also concluded that hypocalcemia can predict severity of AP equal to but not superior to BISAP score.

The best cut off value for calcium level to differentiate between moderately severe/severe cases and mild cases was found to be ≤8.5 mg/dl with sensitivity of 62.5%, specificity of 93.5%, PPV of 83.3%, NPV of 83.3%. This is slightly different from the cut off value suggested by **Gutiérrez-Jiménez et al. (2014)** which is 7.5 mg/dl, with sensitivity 67%, specificity 82%, PPV 27%, NPV 96% and he stated that thoses were similar to Ranson and APACHE II prognostic scales [(17)].

D- dimer levels were higher in the moderately severe/severe group than in the mild pancreatitis group Median (IQR) of 1750 (260 – 7100) versus 640 (536 – 1055) on admission. However, this was not statistically significant even on day 3 and day 7. This could be related to the small sample size.

To the best of our knowledge, only one study was conducted in pediatrics, by **Boskovic et al. (2014)** which included 36 patients and classified the severity of AP according to Pediatric acute pancreatitis severity score. This study found that D-dimer was significantly higher among the severe group [(18)].

Other studies were conducted upon adult population, where many studies compared serum D-dimer level to other severity scoring system and concluded that D-dimer is a good prognostic factor

Maeda et al. in 2006 showed less favorable results of a d-dimer test than the antithrombin III level in a prediction of prognosis of pancreatitis, however the aggravated coagulation parameters predicted a fatal outcome in AP patients[19]. **Radenovic et al in 2009** verified D-dimer as a novel marker for predicting organ failure, with a sensitivity of 90% and NPV of 96% for a cut-off level of 414.00 microg/L. [20]. **Ke et al in 2011,** recommended that D-dimer might replace currently accepted single-variable predictors with higher predictive precision [21].

Due to scanty information about serum D-Dimer level in AP in pediatrics, hematologic parameters were not included in the revised Atlanta criteria, or in any pancreatic studies[22,23,24,25,26,27].

Conclusion:

Higher D-dimer levels were related to more severe disease, yet this was not statistically significant. Large multi-center studies involving the pediatric population is highly needed to reach reliable results.

REFERENCES:

1. Ricardo Restrepo, Heidi E. Hagerott, Sakil Kulkarni,Mona Yasrebi,Edward Y. Lee, 2016. American Journal of Roentgenology.
2. Bai HX, Lowe ME, Husain SZ.2011, What have we learned about acute pancreatitis in children? J Pediatr Gastroenterol Nutr.
3. Lopez MJ.2002 The changing incidence of acute pancreatitis in children: a single-institution perspective. J Pediatr.
4. Suzuki M, Sai JK, Shimizu T. 2014 ,Acute pancreatitis in children and adolescents. World J Gastrointest Pathophysiol 2014.
5. Monica Shukla-Udawatta, MD; Shailender Madani, MD; and Deepak Kamat, MD, PhD, 2017. FAAP PEDIATRIC ANNALS • Vol. 46, No. 5.
6. Whitcomb DC.2006, Clinical practice. Acute pancreatitis. N Engl J Med. 354(20): 2142-50.
7. Alexander Brun MD, Neelam Gidwaney MD, Department of Medicine, Robert Wood Johnson School of Medicine New Brunswick, NJ.2012 ,Practical Gastroenterology .
8. Mamun, A., Datta, I., Rahman, M. A., & Hoque, M. N. (2019). Serum D-dimer is a Predictor of Severity and Outcome of Acute Pancreatitis. BIRDEM Medical Journal, 9(1), 44-54.
9. Alexander Brun MD, Neelam Gidwaney MD, Department of Medicine, Robert Wood Johnson School of Medicine New Brunswick, NJ, 2012.Practical Gastroenterology .
10. Maisam Abu-El-Haija,Soma Kumar, Flora Szabo, Steven Werlin, Darwin Conwell, Peter Banks, and Veronique D. Morinville, June 2018 on behalf of the NASPGHAN Pancreas Committee JPGN . Volume 64, Number 6
11. Grzybowska-Chlebowczyk, Urszula & Jasielska, Martyna & Flak-Wancerz, Anna & Wiecek, Sabina & Gruszczynska, Katarzyna & Chlebowczyk, Wojciech & Woś, Halina. (2017). Acute pancreatitis in children. Gastroenterology Review.
12. Das, S. L., Singh, P. P., Phillips, A. R., Murphy, R., Windsor, J. A., & Petrov, M. S. (2014). Newly diagnosed diabetes mellitus after acute pancreatitis: a systematic review and meta-analysis.
13. Medhat Mohamed Ibrahim, Khaled Gabr, Mohamed Abdulrazik, Hany Fahmy and Yousef El-Booq, 2011. Annals of Pediatrics Surgery
14. Hong, W., Lin, S., Zippi, M., Geng, W., Stock, S., Basharat, Z., Cheng, B., Pan, J., & Zhou, M. (2017). Serum Albumin Is Independently Associated with Persistent Organ Failure in Acute Pancreatitis.

Canadian journal of gastroenterology & hepatology, 2017, 5297143.

15. Khan, K. M., Desai, C. S., Kalb, B., Patel, C., Grigsby, B. M., Jie, T., Gruessner, R. W., & Rodriguez-Rilo, H. (2013). MRI prediction of islet yield for autologous transplantation after total pancreatectomy for chronic pancreatitis. Digestive diseases and sciences, 58(4), 1116–1124.
16. Ashik Pokharel, Prem Raj Sigdel, Suman Phuyal, Prasan Bir Singh Kansakar, Pradeep Vaidya,2017. Surgery Research and Practice.
17. Gutiérrez-Jiménez AA, Castro-Jiménez E, Lagunes-Córdoba R.2014, Calcio sérico total y calcio corregido como predictores de severidad en pancreatitis aguda. Revista de Gastroenterología de México.
18. Boskovic, A., Pasic, S., Soldatovic, I., Milinic, N., & Stankovic, I. (2014). The role of D-dimer in prediction of the course and outcome in pediatric acute pancreatitis. Pancreatology : official journal of the International Association of Pancreatology (IAP) ... [et al.], 14(5), 330–334.
19. Maeda K, Hirota M, Ichihara A, Ohmuraya M, Hashimoto D, Sugita H, et al.2006 Applicability of disseminated intravascular coagulation parameters in the assessment of the severity of acute pancreatitis. Pancreas.32(1):87-92.
20. Radenkovic, D., Bajec, D., Ivancevic, N., Milic, N., Bumbasirevic, V., Jeremic, V., Djukic, V., Stefanovic, B., Stefanovic, B., Milosevic-Zbutega, G., & Gregoric, P. (2009). D-dimer in acute pancreatitis: a new approach for an early assessment of organ failure. Pancreas.
21. Ke L, Ni H, Tong Z, Li WQ, Li N, Li JS, et al. 2012 ,D-dimer as a marker of severity in patients with severe acute pancreatitis. J Hepatobiliary Pancreat Sci .
22. Coffey MJ, Nightingale S, Ooi CY. 2013, Serum lipase as an early predictor of severity in pediatric acute pancreatitis. J Pediatr Gastroenterol Nutr.
23. 23-Lautz TB, Turkel G, Radhakrishnan J, et al. 2012 ,Utility of the computed tomography severity index (Balthazar score) in children with acute pancreatitis. J Pediatr Surg .
24. DeBanto JR, Goday PS, Pedroso MR, et al. 2002, Acute pancreatitis in children. Am J Gastroenterol.
25. Banks P. A. (2016). Acute Pancreatitis: Landmark Studies, Management Decisions, and the Future. Pancreas, 45(5), 633–640.
26. Suzuki M, Shimizu T, Kudo T, Suzuki R, Ohtsuka Y,Yamashiro Y, Shimotakahara A, Yamataka A. 2006 , Usefulness of nonbreath-hold 1-shot magnetic resonance cholangiopancreatography for the evaluation of choledochal cyst in children. J Pediatr Gastroenterol Nutr.
27. Abu-El-Haija, M., Kumar, S., Szabo, F., Werlin, S., Conwell, D., Banks, P., Morinville, V. D., & NASPGHAN Pancreas Committee (2017). Classification of Acute Pancreatitis in the Pediatric Population: Clinical Report From the NASPGHAN Pancreas Committee. Journal of pediatric gastroenterology and nutrition, 64 (6), 984–990.

تقييم دور د-ديمر في تقييم شدة ونتائج التهاب البنكرياس الحاد عند الأطفال

سمر مصطفى محمد ,مروة طلعت الديب ، يسرا محمد محسن ،

قسم أمراض الجهاز الهضمي للأطفال

التهاب البنكرياس الحاد هو المرض الأكثر شيوعًا الذي يؤثر على البنكرياس عند الأطفال. (١) يمكن أن ينتج فشل العديد من الأعضاء و تآكل البنكرياس عن نوبة شديدة من . (٢) يمكن أن يؤدي التقسيم الطبقي المبكر إلى علاج قوي ومنع تطور التلف المستمر للأعضاء واختلال وظائف الأعضاء المتعددة. قيمت دراسات متعددة العلاقة بين شدة د ديمر ولكن معظم هذه الدراسات شملت السكان البالغين فقط (٣)٢).

هدف العمل: الهدف من هذه الدراسة هو معرفة قيمة تحليل د-ديمر المخبري كعلامة تنبؤية لتقييم شدة ونتائج التهاب البنكرياس الحاد .

المرضى والطرق:

في هذه الدراسة المستقبلية تم تقديم ٢٤ طفل و / أو مراهق لقسم أمراض الجهاز الهضمي مستشفيات طب الأطفال جامعة عين شمس تم تشخيص إصابتهم بالتهاب البنكرياس الحاد ، تم استعادة جميع البيانات السريرية للمرضى وخضعوا جميعًا لمصل د ديمر في الأيام الأول والثالث والسابع من القبول وقد ارتبط ذلك بنتيجة الحالات ، حيث تم تقسيم المرضى إلى مجموعتين (أ ، ب) حسب شدة المرض ، كان ٦٦.٧٪ من المرضى يعانون من مرض خفيف ، بينما كان ٣٣.٣٪ من المرضى يعانون من مرض شديد وفقًا لمعايير INSPIRE٤

النتائج :تراوحت أعمار المرضى بين ٤-١٥ سنة ، شكلت الإناث ٦٦.٧٪ من المرضى ، وتراوحت مدة الإصابة بالتهاب البنكرياس الحاد من ١٧ يومًا إلى ٧ أسابيع. انتشر ألم القناة الهضمية إلى الظهر عند ٦٦.٧٪ من المرضى ، بينما كانت الحمى موجودة في ٥٤.٢٪ من المرضى ، وكشف التاريخ الدوائي للمرضى قبل هجوم التهاب البنكرياس أن ٢ مريض (٨.٣٪) تلقيا العلاج الكيميائي ، وتلقى ٨ مرضى المسكنات. بينما تلقى ١٣ مريضًا (٥٩٪) أدوية ستيرويد ومريض واحد فقط كان يتناول أدوية مضادة للصرع ، أصيب ٨ مرضى بمرض شديد و ١٦ مريضًا كانوا مصابين بمرض خفيف.:

الخلاصة: د-ديمر كعلامة شدة في التهاب البنكرياس الحاد لا يعتمد وحده ويحتاج إلى معلمات مختبرية وشعاعية أخرى.