

# Comparative Study between Nasogastric Versus Nasojejunal Feeding in Acute Severe Pancreatitis

#### Mohamed Gaber Ibrahium Mostafa Allam<sup>1,2\*</sup>

<sup>1</sup>Department of anesthesia, ICU and pain management, Faculty of Medicine, Ain Shams University, Cairo, Egypt; <sup>2</sup>King Abd Elaziz Specialist Hospital, Taif, Saudi Arabia

#### ABSTRACT

**Introduction and Aim of the Work:** Nutritional support is an important part in the management of severe acute pancreatitis as it significantly reduces mortality and morbidity by inhibiting bacterial translocation and maintaining integrity of the GIT mucosa. The way of delivering the nutrients through the GIT either by nasogastric (NG) or nasojejunal (NJ) route remains a point of debate. This study was designed to compare the NG vs. NJ routes as a way of nutrition in patients with acute severe pancreatitis.

**Patients and Methods:** 60 patients allocated in two groups. Group A received feeding through NG tube while group B received feeding through NJ tube. Effect of the two enteral feeding methods on the clinical condition of the acute severe pancreatitis, patient's tolerance to feeding, patient's general condition and on achieving satisfactory nutrition parameters of the patient were used to compare between the two methods.

**Results:** The results obtained showed no significant difference between delivering the feeding either by NG or NJ tube by comparing the four indicators used in these studies. The effect on clinical condition of patients with acute severe pancreatitis, patient's tolerance to feeding, patient's general condition, and achieving satisfactory nutrition parameters.

**Conclusion:** Both NG and NJ routes of feeding can be used in acute pancreatitis patients without any significant difference on clinical condition of patients with acute severe pancreatitis, patient's tolerance to feeding, patient's general condition, and both achieving satisfactory nutrition parameters.

Keywords: Severe acute pancreatitis; Nutrition; Nasogastric route; nasojejunal route

#### INTRODUCTION

Acute severe pancreatitis is common and potentially lethal disease. It is associated with significant morbidity and consumes enormous health care resources. Over the last 2 decades the treatment of acute pancreatitis has undergone fundamental changes based on new conceptual insights and evidence from clinical studies [1-3]. The incidence of acute pancreatitis In the United States 200,000 hospital admissions each year. In Europe the incidences of acute pancreatitis range from 4-45 per 100,000 patients a year [4-6]. Overall mortality in acute pancreatitis is approximately 5% [5-7]. Acute pancreatitis has a mild clinical course in about 80% of patients in whom the disease resolves spontaneously within about 1 week [1-2]. However, about 20%

of patients develop severe acute pancreatitis which is associated with mortality rates of 8% up to 39%. Pancreatitis necrosis occurs in around 15%-20% of patients [1-3].

In western countries the disease is mainly caused by gall stones 40%-50%, alcohol abuse 10%-40%, other causes 20%-30% include medication, endoscopic retrograde cholangio-pancreatography (ERCP), hyper-triglyceridemia, hypercalcemia and surgery in around 10% the etiology remains unknown [1-3].

Treatment includes pain management, fluid resuscitation, antibiotics, transfer of the patient to monitored area and nutrition [1-4].

**Correspondence to:** Dr. Mohamed Gaber Ibrahium Mostafa Allam, Department of anesthesia, ICU and pain management, Faculty of Medicine, Ain Shams University, Cairo, Egypt, Tel: 00966550179704; E-mail: mgaberallam@yahoo.com

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Nutritional support has a fundamental role in the management of acute severe pancreatitis as it prevents one of the lethal complications which is bacterial translocation. Bacterial translocation occurs with long time usage of total parenteral nutrition as the dryness of the gastrointestinal (GIT) mucosa due to cessation of GIT secretion cause minor cracks in it. And sensitize the gram-negative commensal to migrate to blood stream from these minor cracks and cause fatal gram-negative septicemia and multi-organ failure [8]. The way of delivering the caloric requirement of the patient remains a continuous issue of research [8-9]. A series of randomized trials comparing enteral vs. parenteral nutrition have been performed in patients with severe acute pancreatitis. These studies in general show a decrease in morbidity and mortality in patients given enteral nutrition early in the course of disease. The benefit was maximized when enteral feeds were started early (72 hours) and at least 50%-60% of needed calories delivered eternally. These patients showed significant reductions in the level of C-reactive protein and oxidant stress [10-12].

There is a consensus among the trials demonstrating decrease in infectious complications (sepsis, multi organ failure, need for operative intervention and infected pancreatic necrosis), length of ICU stay, and significant cost savings approximately 4 times when delivering equal calories through GIT (enteral feeding) [11-12].

Enteral feeding can be given through the nasogastric or the nasojejunal route. Many believe that the delivery of nutrients proximal to the duodeno-jejunal flexure will cause release of cholecystokinin (CCK) and exacerbating the inflammatory process in the pancreas as a result of stimulation of exocrine pancreatic secretion [7]. Various animal and human studies have shown an increase in exocrine pancreatic secretion in response to enteral feeding with greater response to intra-gastric feeding. However, none of these studies were carried out in acute pancreatitis. Animal studies have shown that pancreatic exocrine secretion in response to CCK stimulation is suppressed. In addition it is known that neural pathway affects pancreatic secretion in response to the presence of nutrients in the jejunum which causes significant CCK release also totally blocked in acute pancreatitis. On the other hand delivery of enteral feed distal to the ligament of Treitz does not prevent duodenal exposure to nutrient as a degree of reflux is inevitable [12-14]. This means that delivering of the nutrient in the jejunum can theoretically increase pancreatic secretion through both neural and hormonal (CCK) mechanisms.

#### Aim of the work

The aim of our study is to compare delivering of the nutrient in the stomach vs. in the jejunum as regards:

The effect of both methods on the clinical condition of patients with acute severe pancreatitis, patient's tolerance to feeding, patient's general condition and the effect of both methods on achieving satisfactory nutrition parameters.

#### PATIENTS AND METHODS

A total of 60 patients admitted to King Abdulaziz Specialists Hospital between June 2018 and April 2020 with both a clinical and biochemical presentation of acute pancreatitis with the following inclusion criteria: Abdominal pain  $\geq$  6 on the visual analogue scale without given analgesia, abdominal distension and tenderness with serum amylase and serum lipase at least 3 times the upper limit of the reference range (considering normal lipase level from 0-160 U/L, and normal amylase level from 0-100 U/L) with confirmed abdominal computerized axial tomography of grade D and E on Ranson and colleagues criteria [28] of inflamed pancreatic picture.

An acute physiology and chronic health evaluation (APACHE) II were done for all of them and only score of  $\geq 8$  included in our study.

King Abdulaziz Hospital research and ethical committee approved the project

A written consent for all the patients was obtained.

Children below age of 18 years and pregnant or lactating females were excluded

All patients were randomized into group A and group B, each group contains 30 patients, this randomization was made by numbers. Group A received nasogastric feeding (NG), while group B received nasojejunal feeding (NJ).

The insertion of NG tubes were performed by the ICU physician and the position was checked by auscultation of gas from 50 ml syringe by injecting 20 ml of air in the NG and by X-ray abdomen to be sure from the site of NG tube.

The nasojejunal tube is silicone or polyurethane tube with an inner stylet that is positioned (under fluoroscopic guidance) beyond the ligament of Treitz.

Patients were placed in right lateral position and prokinetic (erythromycin 250 mg IV bolus) given to assist the passage of the tube through the pylorus.

Also, if difficulties were faced, endoscopy (using pediatric colonoscopy) was used to pass the feeding tube over an endoscopically placed guide wire.

Daily assessment for the proper site of both the NG & NJ were done by daily x-ray abdomen.

Feeding started to all patients in both groups on the third day from confirmation of the diagnosis of severe pancreatitis in those patients showed decreasing level of serum lipase and amylase.

Feeds were started by the rate of 20 ml/hour by continuous feeding pump (B- Braun) and increased by 20 ml/hour every 4 hours till reached 85 ml/hour. The caloric target was 2,040 kcal per day at least.

We used 'Insure' from 'Abbot' company, osmolarity 319 mosmol/L, osmolality: 376 mosmol/kg H2O which gives 1 kcal/ml and protein content 4 gm/100 ml, trace elements and vitamins.

The study was conducted on monitoring of four pillar for 13 days (data collected one day before feeding and every three days for 12 days). The first pillar comparing the effect of the two enteral feeding methods on the clinical condition of the acute

severe pancreatitis and this monitored clinically and laboratory by fixed parameters, clinically, by abdominal pain which assessed by visual analogue scale (VAS). and laboratory, by both markers of severity of pancreatic acini destruction which assessed by serum amylase, lipase and Markers of severity of interstitial pancreatic tissue destruction which assessed by the level of Lactate Dehydrogenase level (LDH), Aspartate aminotransferase (AST), C-reactive protein, prostacyclin and interleukin 8. The second pillar include effect of the two enteral feeding methods on the patient's tolerance and these monitored in our study by both post-feeding vomiting and post feeding attacks of osmotic diarrhea and also abdominal pain. Third pillar include effect of the two enteral feeding methods on the patient's general condition and these monitored by follows the APACHE II Score, hemodynamics of the patients in both groups (Mean arterial blood pressure and pulse) and arterial oxygen saturation (SpO<sub>2</sub>). Fourth pillar include effect of the two enteral feeding methods on achieving satisfactory nutrition parameters of the patients and both albumin level, radium blood sugar and electrolytes (sodium and potassium level) used as indicator for this.

All parameters observed one day before starting feeding and every 3 days for 12 days (duration of the study).

Three patients died from group B one patient died after 4 days and other two patients died after 7 and 8 days respectively from starting of NJ feeding from ARDS and multiple organ failure while two patients died from group A in the 5<sup>th</sup> day of feeding from the same cause.

#### Statistical analysis:

The Data was collected and entered into the personal computer. Statistical analysis was done using Statistical Package for Social Sciences (SPSS/version 20) software. Arithmetic mean, standard deviation, for categorized parameters chai square test was used while for numerical data t-test was used to compare the two groups. The level of significance was 0.05.

#### Sample size:

Depend on the research context, including the researcher's objectives and proposed analyses.

$$n = \frac{Z^2 P (1 - P)}{d^2}$$

The following formula was used to calculate the required sample size in this study; where n is the sample size, Z is the statistic

corresponding to level of confidence, P is expected prevalence, and d is precision (corresponding to effect size). The level of confidence was 95%. By using this equation, the sample size was 30 cases in each group (i.e. 60 cases in the two groups).

#### RESULTS

**Table 1:** Comparison between the two studied groups regarding thecause of severe pancreatitis.

	GROU	JP A	GROU	GROUP B		
	"n=30"		"n=30'	"n=30"		
	No.	%	No.	%		
Gall stone	27	90	28	93.3		
Idiopathic	3	10	1	3.3	0.316	
hyperparathyroidism	-	0	1	3.3		

 Table 2: Demographic data between the two studied groups regarding age and sex.

	GROUP A		GROU	- n valua	
Age group	"n=30"	"n=30"		"	p value
	No.	%	No.	%	
40 to 50 yrs	2	6.7	1	3.3	-
51 to 60 yrs	13	43.3	12	40	0.021
61 to 70 yrs	10	33.3	13	43.3	0.831
71 to 80 yrs	5	16.7	4	13.3	-
Sex					
Male	27	90	25	83.3	0.447
Female	3	10.00%	5	16.67%	0.447

p is significant if < 0.05

APACHE II Score	GROUP A		GROUP B		р
Al ACTIL II Stole					
One day before feeding	n=30	%	n=30	%	

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8-15	2	6.7	3	10	
16-20	3	10	2	6.7	0.921
21-25	9	30	8	26.7	0.921
>25	16	53.3	17	56.7	
1 <sup>st</sup> 3 days of feeding	n=30	%	n=30	%	
8-15	9	30	10	33.3	
16-20	4	13.3	3	10	0.057
21-25	8	26.7	7	23.3	0.957
>25	9	30	10	33.3	
2 <sup>nd</sup> 3 days of feeding	n=28	%	n=29	%	
8-15	11	39.3	9	31.03	
16-20	5	17.9	7	24.14	0.927
21-25	6	21.4	8	27.59	0.827
>25	6	21.4	5	17.24	
3 <sup>rd</sup> 3 days of feeding	n=28	%	n=27	%	
8-15	14	50	15	55.6	
16-20	10	35.7	10	37	0.877
21-25	2	7.1	1	3.7	0.077
>25	2	7.1	1	3.7	
4 <sup>th</sup> 3 days of feeding	n=28	%	n=27	%	
8-15	22	78.6	23	85.2	
16-20	4		2		0.970
21-25	2		2		0.879
>25	0		0		
P is significant if <0.05					

VAS for Abdominal Pain	GROUP A		GROUP B		Р
One day just before feeding	n=30	%	n=30	%	_
0-3	18	0.6	20	0.6	0.92

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4-6	10	0.33	9	0.3	
7-9	2	0.06	1	0.03	
10	0	0	0	0	
1 <sup>st</sup> 3 days post-feeding	n=30	%	n=30	%	
0-3	14	0.47	13	0.43	
4-6	13	0.43	12	0.4	0.740
7-9	3	0.1	5	0.17	0.749
10	0	0	0	0	
2 <sup>nd</sup> 3 days post-feeding	n=28	%	n=29	%	
0-3	15	53.6	16	55.17	
4-6	10	35.7	9	31.03	0.541
7-9	3	10.7	4	13.79	0.541
10	0	0	0	0.00%	
3 <sup>rd</sup> 3 days post-feeding	n=28	%	n=27	%	
)-3	21	75	22	81.5	
1-6	6	21.4	4	14.8	0.816
7.9	1	3.6	1	3.7	0.816
10	0	0	0	0	
4 <sup>th</sup> 3 days post- feeding	n=28	%	n=27	%	
0-3	21	75	23	85.2	
4-6	7	25	4	14.8	0.345
7-9	0	0	0	0	
10	0	0	0	0	
p is significant if <0.05					

 Table 5: Number of post-feeding attacks of vomiting in both groups.

Number of vomiting attacks	GROUP A GROUP B		р		
One day just before feeding	n=30	%	n=30	%	
First 8 hours	6	20	5	16.67	0.933
Second 8 hours	5	16.67	5	16.67	0.933

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Third 8 hours	3	10	2	6.67	
1 <sup>st</sup> 3 days post- feeding	n=30	%	n=30	%	
0-3 attacks/day	10	33.33	20	66.67	
4-6 attacks/day	14	46.67	10	33.33	0.041*
7-9 attacks/day	5	16.67	0	0	0.041
>9 attacks/day	1	3.33	0	0	
2 <sup>nd</sup> 3 days post-feeding	n=28	%	n=29	%	
0-3 attacks/day	16	57.1	22	81.5	
4-6 attacks/day	10	35.7	7	25.9	0.020*
7-9 attacks/day	2	7.1	0	0	0.039*
>9 attacks/day	0	0	0	0	
3 <sup>rd</sup> 3 days post-feeding	n=28	%	n=27	%	
0-3 attacks/day	28	100	22	81.5	
4-6 attacks/day	0	0	4	14.8	0.124
7-9 attacks/day	0	0	1	3.7	0.124
>9 attacks/day	0	0	0	0	
4 <sup>th</sup> 3 days post feeding	n=28		n=27		
0-3 attacks/day	28	100	22	81.5	
4-6 attacks/day	0	0	4	14.8	0.124
7-9 attacks/day	0	0	1	3.7	0.124
>9 attacks/day	0	0	0	0	

 Table 6: Number of post-feeding attacks of diarrhea in both groups.

Post feeding diarrh (motion/day)	nea GROUP A		GROUP	В	
1 <sup>st</sup> 3 days post-feeding	n=30	%	n=30	%	
0-3 motion/day	19	0.633	8	0.27	
4-6 motion/day	10	0.333	10	0.33	0.004*
7-9 motion/day	1	0.033	9	0.3	0.004*
>9 motion/day	0	0	3	0.1	
2 <sup>nd</sup> 3 days post-feeding	n=28	%	n=29	%	
0-3 motion/day	22	78.6	12	41.38	0.035*

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4-6 motion/day	6	21.4	13	44.83	
7-9 motion/day	0	0	4	13.79	
>9 motion/day	0	0	0	0	
3 <sup>rd</sup> 3 days post-feeding	n=28	%	n=27	%	
0-3 motion/day	28	100	17	63	
4-6 motion/day	0	0	8	29.6	0.004*
7-9 motion/day	0	0	2	7.4	0.004*
>9 motion/day	0	0	0	0	
4 <sup>th</sup> 3 days post-feeding	n=28	%	n=27	%	
0-3 motion/day	28	100	22	81.5	
4-6 motion/day	0	0	4	14.8	0.124
7-9 motion/day	0	0	1	3.7	0.124
>9 motion/day	0	0	0	0	

 Table 7: Pulse in beat/min in both groups.

Pulse (beat/min)	GROUP A		GROUP B		Р
	20	0/			
One day just before feeding	n=30	%	n=30	%	
80-90 beat/min	12	0.4	13	0.43	
91-100 beat/min	10	0.333	9	0.3	0.954
101-120 beat/min	8	0.267	8	0.27	0.954
>120 beat/min	0	0	0	0	
1 <sup>st</sup> 3 days post-feeding	n=30	%	n=30	%	
80-90 beat/min	9	0.3	8	0.27	
91-100 beat/min	13	0.43	12	0.4	0.851
101-120 beat/min	8	0.27	10	0.33	0.851
>120 beat/min	0	0	0	0	
2 <sup>nd</sup> 3 days post-feeding	n=28	%	n=29	%	
80-90 beat/min	8	28.6	7	24.14	
90-100 beat/min	14	50	13	44.83	0.709
100-120 beat/min	6	21.4	9	31.03	

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>120 beat/min	0	0	0	0	
3 <sup>rd</sup> 3 days post-feeding	n=28	%	n=27	%	
80-90 beat/min	12	42.9	11	40.7	
91-100 beat/min	10	35.7	11	40.7	0.021
101-120 beat/min	6	21.4	5	18.5	0.921
>120 beat/min	0	0	0	0	
4 <sup>th</sup> 3 days post-feeding	n=28	%	n=27	%	
80-90 beat/min	22	78.6	20	74.1	
91-100 beat/min	6	21.4	7	25.9	0.025
101-120 beat/min	0	0	0	0	0.925
>120 beat/min	0	0	0	0	

Table 8: Mean arterial blood pressure in mm /Hg in both groups.

	GROUP A		GROUP B		Р
MABP (mm/Hg)	No	%	No	%	
One day just before feeding	n=30	%	n=30	%	
80-85 mm/Hg	17	0.57	16	0.533	
86-90 mm/Hg	7	0.23	8	0.267	0.052
91-95 mm/Hg	6	0.2	6	0.2	0.952
>95 mm/Hg	0	0	0	0.00%	
1 <sup>st</sup> 3 days post-feeding	n=30	%	n=30	%	
80-85 mm/Hg	16	0.53	17	0.567	
86-90 mm/Hg	8	0.27	6	0.2	0.021
91-95 mm/Hg	6	0.2	7	0.233	0.821
>95 mm/Hg	0	0	0	0.00%	
2 <sup>nd</sup> 3 days post-feeding	n=28	%	n=29	%	
80-85 mm/Hg	14	50	15	51.72	
86-90 mm/Hg	7	25	6	20.69	0.022
91-95 mm/Hg	7	25	8	27.59	0.922
>95 mm/Hg	0	0	0	0.00%	
3 <sup>rd</sup> 3 days post-feeding	n=28	%	n=27	%	

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80-85 mm/Hg	15	53.57	14	51.85		
86-90 mm/Hg	9	32.14	10	37.04	0.800	
91-95 mm/Hg	4	14.29	3	11.11	0.899	
>95 mm/Hg	0	0	0	0.00%		
4 <sup>th</sup> 3 days post-feeding	n=28	%	n=27	%		
80-85 mm/Hg	20	71.43	19	70.37		
86-90 mm/Hg	8	28.57	7	25.93	0.020	
91-95 mm/Hg	0	0	1	3.7	0.989	
>95 mm/Hg	0	0	0	0		

 Table 9: Oxygen saturation in both groups.

Arterial Oxygen Saturation	GROUP A		GROUP B		p
(SPO <sub>2</sub> %)	No	%	No	%	
One day just before feeding	n=30	%	n=30	%	_
85-90%	5	0.17	6	0.2	
91-95%	19	0.63	18	0.6	0.943
>95%	6	0.2	6	0.2	_
1 <sup>st</sup> 3 days post-feeding	n=30	%	n=30	%	
85-90%	8	0.27	7	0.23	
91-95%	11	0.37	13	0.43	0.869
>95%	11	0.37	10	0.33	_
2 <sup>nd</sup> 3 days post-feeding	n=28	%	n=29	%	
85-90%	1	3.57	2	6.9	
91-95%	12	42.86	14	48.28	0.736
>95%	15	53.57	13	44.83	_
3 <sup>rd</sup> 3 days post-feeding	n=28	%	n=27	%	
85-90%	1	3.57	1	3.7	
91-95%	8	28.57	8	29.63	0.995
>95%	19	67.86	18	66.67	_
4 <sup>th</sup> 3 days post-feeding	n=28	%	n=27	%	
85-90%	0	0	0	0	0.997

91-95%	6	21.43	6	22.22
>95%	22	78.57	21	77.78

Table 10: Random blood sugar in mg% in both groups.

Post feeding Random blood	GROUP A		GROUP B	GROUP B		
sugar (mg%)	No	%	No	%		
1 <sup>st</sup> 3 days post-feeding	n=30	%	n=30	%		
≤ 80 mg%	0	0	1	0.03		
81-150 mg%	24	0.8	22	0.73	0.929	
>150 mg%	6	0.2	7	0.23		
2 <sup>nd</sup> 3 days post-feeding	n=28	%	n=29	%		
≤ 80 mg%	0	0	0	0		
81-150 mg%	21	75	22	75.86	0.997	
>150 mg%	7	25	7	24.14		
3 <sup>rd</sup> 3 days post-feeding	n=28	%	n=27	%		
≤ 80 mg%	0	0	0	0		
81-150 mg%	23	82.14	22	81.48	0.998	
>150 mg%	5	17.86	5	18.52		
4 <sup>th</sup> 3 days post-feeding	n=28	%	n=27	%		
≤ 80 mg%	0	0	0	0		
81-150 mg%	22	78.57	23	85.19	0.816	
>150 mg%	6	21.43	4	14.81		

Table 11: Post feeding albumin level in gm/dL in both groups.

	GROUP A		GROUP B		р
Post feeding Albumin level (gm/dL)	No	%	No	%	
1 <sup>st</sup> 3 days post-feeding	n=30	%	n=30	%	
$\leq 2 \text{ gm/dL}$	4	0.13	2	0.07	
2.14 gm/dL	24	0.8	26	0.87	0.688
>4 gm/dL	2	0.07	2	0.07	
2 <sup>nd</sup> 3 days post-feeding	n=28	%	n=29	%	
≤ 2 gm/dL	0	0	0		0.931

2.1-4 gm/dL	18	64.29	20		
>4 gm/dL	10	35.71	9		
3 <sup>rd</sup> 3 days post-feeding	n=28	%	n=27	%	
≤ 2 gm/dL	0	0	0	0	
2.1-4 gm/dL	14	50	14	51.85	0.990
>4 gm/dL	14	50	13	48.15	
4 <sup>th</sup> 3 days post-feeding	n=28	%	n=27	%	
≤ 2 gm/dL	0	0	0	0	
2.1-4 gm/dL	9	32.14	9	33.33	0.925
>4 gm/dL	19	67.86	18	66.67	

 Table 12: Post feeding serum Sodium level in mEq/L in both groups.

$\mathbf{D}_{\mathbf{r}} = \mathbf{f} \left\{ \mathbf{r} \in \mathbf{J} : \mathbf{r} \in \mathbf{N} : \mathbf{I}_{\mathbf{r}} = \mathbf{I} \left\{ \mathbf{r} \in \mathbf{F} = \mathbf{J} \right\}$	GROUP	GROUP A		i	Р
Post feeding Na level (mEq/L)	No	%	No	%	
1 <sup>st</sup> 3 days post-feeding	n=30	%	n=30	%	
≤ 120 mEq/L	3	0.10	2	0.07	
121-145 mEq/L	21	0.70	20	0.67	0.775
>145 mEq/L	6	0.20	8	0.27	
2 <sup>nd</sup> 3 days post-feeding	n=28	%	n=29	%	
≤ 120 mEq/L	0	0.00	0	0.00	
121-145 mEq/L	22	78.57	23	79.31	0.997
>145 mEq/L	6	21.43	6	20.69	
3 <sup>rd</sup> 3 days post-feeding	n=28	%	n=27	%	
≤ 120 mEq/L	0	0.00	0	0.00	
121-145 mEq/L	26	92.86	25	92.59	0.969
>145 mEq/L	2	7.14	2	7.41	
4 <sup>th</sup> 3 days post-feeding	n=28	%	n=27	%	
≤ 120 mEq/L	0	0.00	0	0.00	
121-145 mEq/L	28	100.00	27	100.00	0.979
>145 mEq/L	0	0.00	0	0.00	

Table 13: post feeding potassium level in mEq/L in both groups.

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Post feeding Potassium level	GROUP A		GROUP B	GROUP B		
(mEq/L)	No	%	No	%		
1 <sup>st</sup> 3 days post-feeding	n=30	%	n=30	%		
$\leq 3 \text{ mEq/L}$	10	0.33	9	0.3		
3.1-5.5 mEq/L	13	0.43	14	0.47	0.956	
>5.5 mEq/L	7	0.23	7	0.23		
2 <sup>nd</sup> 3 days post-feeding	n=28	%	n=29	%		
≤ 3 mEq/L	4	14.29	5	17.24		
3.1-5.5 mEq/L	19	67.86	20	68.97	0.891	
>5.5 mEq/L	5	17.86	4	13.79		
3 <sup>rd</sup> 3 days post-feeding	n=28	%	n=27	%		
$\leq 3 \text{ mEq/L}$	0	0	0	0		
3.1-5.5 mEq/L	26	92.86	25	92.59	0.999	
>5.5 mEq/L	2	7.14	2	7.41		
4 <sup>th</sup> 3 days post-feeding	n=28	%	n=27	%		
$\leq 3 \text{ mEq/L}$	0	0	0	0		
3.1-5.5 mEq/L	28	100	27	100	0.979	
>5.5 mEq/L	0	0	0	0		

 Table 14: Serum lipase level in both groups.

	GROUP A		GROUP B		Р
Serum lipase	No	%	No	%	
One day just before feeding	n=30	%	n=30	%	
300-600 U/L	11	0.37	12	0.4	
601-900 U/L	16	0.53	15	0.5	
901-1200 U/L	3	0.1	2	0.07	0.878
>1200 U/L	0	0	1	0.03	
1 <sup>st</sup> 3 days post-feeding	n=30	%	n=30	%	
300-600 U/L	10	0.33	9	0.3	
601-900 U/L	17	0.57	16	0.53	0.892
901-1200 U/L	2	0.07	3	0.1	

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>1200 U/L	1	0.03	2	0.07	
2 <sup>nd</sup> 3 days post-feeding	n=28	%	n=29	%	
300-600 U/L	14	50	17	58.62	
601-900 U/L	10	35.71	9	31.03	0.012
901-1200 U/L	3	10.71	2	6.9	0.913
>1200 U/L	1	3.57	1	3.45	
3 <sup>rd</sup> 3 days post-feeding	n=28	%	n=27	%	
300-600 U/L	21	75	23	85.19	
601-900 U/L	4	14.29	2	7.41	0.702
901-1200 U/L	2	7.14	1	3.7	0.783
>1200 U/L	1	3.57	1	3.7	
4 <sup>th</sup> 3 days post-feeding	n=28	%	n=27	%	
300-600 U/L	23	82.14	24	88.89	
601-900 U/L	2	7.14	1	3.7	0.0202
901-1200 U/L	2	7.14	1	3.7	0.8802
>1200 U/L	1	3.57	1	3.7	

#### Table 15: Serum amylase level in U/L in both groups.

Samura annulasa	GROUP A		GROUP B		Р
Serum amylase	No	%	No	%	
One day just before feeding	n=30	%	n=30	%	
<100	0	0	0	0	
100-250 U/L	4	0.13	5	0.17	
251-400 U/L	12	0.4	11	0.37	0.959
401-500 U/L	9	0.3	8	0.27	
>500 U/L	5	0.17	6	0.2	
1 <sup>st</sup> 3 days post-feeding	n=30	%	n=30	%	
<100	0	0	0	0	
100-250 U/L	2	0.07	3	0.1	0.926
251-400 U/L	14	0.47	12	0.4	
401-500 U/L	10	0.33	10	0.33	

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>500 U/L	4	0.13	5	0.17	
2 <sup>nd</sup> 3 days post-feeding	n=28	%	n=29	%	
<100	1	3.57	2	6.9	
100-250 U/L	5	17.86	6	20.69	
251-400 U/L	11	39.29	10	34.48	0.949
401-500 U/L	9	32.14	8	27.59	
>500 U/L	2	7.14	3	10.34	
3 <sup>rd</sup> 3 days post-feeding	n=28	%	n=27	%	
<100	7	25	8	29.63	
100-250 U/L	13	46.43	12	44.44	
251-400 U/L	3	10.71	4	14.81	0.943
401-500U/L	3	10.71	2	7.41	
>500 U/L	2	7.14	1	3.7	
4 <sup>th</sup> 3 days post-feeding	n=28	%	n=27	%	
<100	13	46.43	15	55.56	
100-250 U/L	10	35.71	9	33.33	
251-400 U/L	3	10.71	2	7.41	0.949
401-500 U/L	2	7.14	1	3.7	
>500 U/L	0	0	0	0	

#### Table 16: Serum LDH level in U/L in both groups.

LDH (U/L)	GROUP A		GROUP B	GROUP B	
	No	%	No	%	
One day just before feeding	n=30	%	n=30	%	
≥ 200 U/L	0	0	0	0	
201-400 U/L	6	20	5	16.67	
401-600 U/L	7	23.33	6	20	0.962
601-700 U/L	11	36.67	12	40	
>700 U/L	6	20	7	23.33	
1 <sup>st</sup> 3 days post-feeding	n=30	%	n=30	%	
≥ 200 U/L	0	0	0	0	0.991

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201-400 U/L	1	3.33	1	3.33	
401-600 U/L	10	33.33	11	36.67	
601-700 U/L	10	33.33	10	33.33	
>700 U/L	9	30	8	26.67	
2 <sup>nd</sup> 3 days post-feeding	n=28	%	n=29	%	
≥ 200 U/L	0	0	0	0	
201-400 U/L	6	21.43	7	24.14	
401-600 U/L	10	35.71	11	37.93	0.958
601-700 U/L	9	32.14	9	31.03	
>700 U/L	3	10.71	2	6.9	
3 <sup>rd</sup> 3 days post-feeding	n=28	%	n=27	%	
≥ 200 U/L	2	7.14	3	11.11	
201-400 U/L	9	32.14	10	37.04	
401-600 U/L	10	35.71	9	33.33	0.892
601-700 U/L	7	25	5	18.52	
>700 U/L	0	0	0	0	
4 <sup>th</sup> 3 days post-feeding	n=28	%	n=27	%	
≥ 200 U/L	12	42.86	11	40.74	
201-400 U/L	6	21.43	8	29.63	
401-600 U/L	8	28.57	7	25.93	0.87
601-700 U/L	2	7.14	1	3.7	
>700 U/L		0			

Table 17: Serum AST level in U/L in both groups.

AST (U/L)	GROUP A		GROUP B		Р
	No	%	No	%	
One day just before feeding	n=30	%	n=30	%	
≥ 40 U/L	0	0	0	0	
41-200 U/L	3	10	2	6.67	0.946
201-800 U/L	10	33.33	9	30	

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	GROUP A		GROUP B		р
Cable 18: ESR in both groups.					
>1000 U/L	0	0	0	0	
801-1000 U/L	5	17.86	4	14.81	
201-800 U/L	9	32.14	8	29.63	0.927
41-200 U/L	9	32.14	11	40.74	
≥ 40 U/L	5	17.86	4	14.81	
4 <sup>th</sup> 3 days post-feeding	n=28	%	n=27	%	
>1000 U/L	4	14.29	3	11.11	
801-1000 U/L	5	17.86	6	22.22	
201-800 U/L	9	32.14	8	29.63	0.965
41-200 U/L	10	35.71	10	37.04	
≥ 40 U/L	0	0	0	0	
3 <sup>rd</sup> 3 days post-feeding	n=28	%	n=27	%	
>1000 U/L	6	21.43	5	17.24	
801-1000 U/L	6	21.43	6	20.69	
201-800 U/L	9	32.14	10	34.48	0.979
41-200 U/L	7	25	8	27.59	
≥ 40 U/L	0	0	0	0	
2 <sup>nd</sup> 3 days post-feeding	n=28	%	n=29	%	
>1000 U/L	10	33.33	9	30	
801-1000 U/L	8	26.67	8	26.67	
201-800 U/L	11	36.67	12	40	0.992
41-200 U/L	1	3.33	1	3.33	
≥ 40 U/L	0	0	0	0	
1 <sup>st</sup> 3 days post-feeding	n=30	%	n=30	%	
>1000 U/L	7	23.33	8	26.67	

$\mathbf{ESD}$ ( $\mathbf{rr}$ , $\mathbf{rr}$ /L $\mathbf{r}$ )	GROUP A		GROUP B		р
ESR (mm/hr)	No	%	No	%	
One day just before feeding	n=30	%	n=30	%	

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≥ 30 mm/h	0	0	0	0	
31-50 mm/h	2	6.67	1	3.33	
51-70 mm/h	8	26.67	7	23.33	0.919
71-90 mm/h	12	40	13	43.33	
>90 mm h	8	26.67	9	30	
1 <sup>st</sup> 3 days post-feeding	n=30	%	n=30	%	
≥ 30 mm/h	0	0	0	0	
31-50 mm/h	0	0	0	0	
51-70 mm/h	10	33.33	11	36.67	0.992
71-90 mm/h	9	30	9	30	
>90 mm/h	11	36.67	10	33.33	
2 <sup>nd</sup> 3 days post-feeding	n=28	%	n=29	%	
≥ 30 mm/h	0	0	0	0	
31-50 mm/h	3	10.71	4	13.79	
51-70 mm/h	10	35.71	11	37.93	0.969
71-90 mm/h	8	28.57	8	27.59	
>90 mm/h	7	25	6	20.69	
3 <sup>rd</sup> 3 days post-feeding	n=28	%	n=27	%	
≥ 30 mm/h	0	0	0	0	
31-50 mm/h	9	32.14	10	37.04	
51-70 mm/h	10	35.71	9	33.33	0.889
71-90 mm/h	6	21.43	4	14.81	
>90 mm/h	3	10.71	4	14.81	
4 <sup>th</sup> 3 days post-feeding	n=28	%	n=27	%	
> 30 mm/h	6	21.43	5	18.52	
31-50 mm/h	7	25	9	33.33	
51-70 mm/h	8	28.57	7	25.93	0.923
71-90 mm/h	6	21.43	5	18.52	
>90 mm/h	1	3.57	1	3.7	

Table 19: Serum C-reactive protein level in mg/L in both groups.

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Consistive marketing 11 (/1)	GROUP A		GROUP B		Р
C-reactive protein level (mg/L)	No	%	No	%	
One day just before feeding	n=30	%	n=30	%	
≤ 100 mg/L	0	0	0	0	
101-200 mg/L	5	16.67	4	13.33	
201-250 mg/L	8	26.67	7	23.33	0.958
251-300 mg/L	12	40	13	43.33	
>300mg/L	5	16.67	6	20	
1 <sup>st</sup> 3 days post-feeding	n=30	%	n=30	%	
≤ 100 mg/L	0	0	0	0	
101-200 mg/L	2	6.67	2	6.67	
201-250 mg/L	10	33.33	11	36.67	0.991
251-300 mg/L	9	30	9	30	
>300 mg/L	9	30	8	26.67	
2 <sup>nd</sup> 3 days post-feeding	n=28	%	n=29	%	
≤ 100 mg/L	0	0	0	0	
101-200 mg/L	6	21.43	7	24.14	
201-250 mg/L	10	35.71	11	37.93	0.969
251-300 mg/L	8	28.57	8	27.59	
>300mg/L	4	14.29	3	10.34	
3 <sup>rd</sup> 3 days post-feeding	n=28	%	n=27	%	
≤ 100 mg/L	2	7.14	3	11.11	
101-200 mg/L	9	32.14	10	37.04	
201-250 mg/L	10	35.71	9	33.33	0.876
251-300 mg/L	6	21.43	4	14.81	
>300	1	3.57	1	3.7	
4 <sup>th</sup> 3 days post-feeding	n=28	%	n=27	%	
≤ 100 mg/L	11	39.29	10	37.04	
101-200 mg/L	7	25	9	33.33	
201-250 mg/L	7	25	6	22.22	0.906
251-300 mg/L	3	10.71	2	7.41	

>300 mg/L 0 0 0 0
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#### Table 20: Serum Pro-calcitonin level in ng/ml in both groups.

Pro-calcitonin level (ng/ml)	GROUP A		GROUP B	GROUP B		
	No	%	No	%		
One day just before feeding	n=30	%	n=30	%		
0.5-1.5 ng/ml	0	0	0	0		
1.6-2.0 ng/ml	6	20	5	16.67		
2.1-2.5 ng/ml	8	26.67	7	23.33	0.964	
2.6-3.0 ng/ml	9	30	10	33.33		
>3 ng/ml	7	23.33	8	26.67		
1 <sup>st</sup> 3 days post-feeding	n=30	%	n=30	%		
0.5-1.5 ng/ml	0	0	0	0		
1.6-2.0 ng/ml	4	13.33	5	16.67		
2.1-2.5 ng/ml	5	16.67	6	20	0.959	
2.6-3.0 ng/ml	12	40	11	36.67		
>3 ng/ml	9	30	8	26.67		
2 <sup>nd</sup> 3 days post-feeding	n=28	%	n=29	%		
0.5-1.5 ng/ml	1	3.57	2	6.9		
1.6-2.0 ng/ml	7	25	9	31.03		
2.1-2.5 ng/ml	9	32.14	9	31.03	0.943	
2.6-3.0 ng/ml	7	25	6	20.69		
>3 ng/ml	4	14.29	3	10.34		
3 <sup>rd</sup> 3 days post-feeding	n=28	%	n=27	%		
0.5-1.5 ng/ml	4	14.29	5	18.52		
1.6-2.0 ng/ml	13	46.43	12	44.44		
2.1-2.5 ng/ml	5	17.86	4	14.81	0.953	
2.6-3.0 ng/ml	4	14.29	5	18.52		
>3 ng/ml	2	7.14	1	3.7		
4 <sup>th</sup> 3 days post-feeding	n=28	%	n=27	%		
0.5-1.5 ng/ml	9	32.14	10	37.04	0.823	

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1.6-2.0 ng/ml	12	42.86	13	48.15
2.1-2.5 ng/ml	5	17.86	3	11.11
2.6-3.0 ng/ml	2	7.14	1	3.7
>3 ng/ml	0	0	0	0

Table 21: Serum Interleukin 8 level in Pg/ml in both groups.

Interleukin 8 level (Pg/ml)	GROUP A		GROUP B		Р
interleukin 8 level (Pg/mi)	No	%	No	%	
One day just before feeding	n=30	%	n=30	%	
5-10 pg/ml	0	0	0	0	
10.1-20 pg/ml	8	26.67	9	30	
20.1-40 pg/ml	9	30	8	26.67	0.962
40.1-60 pg/ml	9	30	8	26.67	
>60 pg/ml	4	13.33	5	16.67	
1 <sup>st</sup> 3 days post-feeding	n=30	%	n=30	%	
5-10 pg/ml	0	0	0	0	
10.1-20 pg/ml	0	0	0	0	
20.1-40 pg/ml	4	13.33	5	16.67	0.918
40.1-60 pg/ml	17	56.67	17	56.67	
>60 pg/ml	9	30	8	26.67	
2 <sup>nd</sup> 3 days post-feeding	n=28	%	n=29	%	
5-10 pg/ml	0	0	0	0	
10.1-20 pg/ml	2	7.14	3	10.34	
20.1-40 pg/ml	11	39.29	13	44.83	0.910
40.1-60 pg/ml	11	39.29	10	34.48	
>60 pg/ml	4	14.29	3	10.34	
3 <sup>rd</sup> 3 days post-feeding	n=28	%	n=27	%	
5-10 pg/ml	2	7.14	3	11.11	
10.1-20 pg/ml	6	21.43	7	25.93	0.040
20.1-40 pg/ml	10	35.71	9	33.33	0.949
40.1-60 pg/ml	8	28.57	7	25.93	

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>60 pg/ml	2	7.14	1	3.7	
4 <sup>th</sup> 3 days post-feeding	n=28	%	n=27	%	
5-10 pg/ml	8	28.57	9	33.33	
10.1-20 pg/ml	13	46.43	13	48.15	
20.1-40 pg/ml	5	17.86	4	14.81	0.922
40.1-60 pg/ml	2	7.14	1	3.7	
>60 pg/ml	0	0	0	0	

#### DISCUSSION

# As regards the effect of both enteral feeding methods on the clinical condition of the acute severe pancreatitis:

As regard the clinical assessment of the First indicator, results showed that abdominal pain which evaluated in our study by VAS revealed that most of the patients in both groups had VAS from (0-3) one day before starting of feeding and this result enable us to start enteral feeding safely as abdominal pain decreased from >6 on the VAS (considered one of our inclusion criteria) to 3 on the third day after confirming the diagnosis of acute severe pancreatitis. On the other hand, most of patients in both groups had VAS from 0-6 on the  $1^{st}\xspace$  and  $2^{nd}\xspace$  three days post feeding and 0-3 in the 3<sup>rd</sup> and 4<sup>th</sup> three days post feeding. With no significant difference between the two groups in all the studied duration. These results prove that both methods of feeding not affecting the abdominal pain and not increasing the local inflammatory reaction in the pancrease which clinically assessed in our study by evaluating the abdominal pain. And there was no significant difference between VAS recorded in all patients in both groups one day before feeding and after feeding. And this result proves our former theory written in the introduction that both neuronal reflexes and hormonal effect (release of CCK in response to presence of feeding in the gut) that increase pancreatic secretion in response to enteral feeding are completely attenuated or even abolished in acute severe pancreatitis. As the inflamed pancreas not respond to feeding in the gut. As regard laboratory assessment of the inflammatory process in the pancreatic acini, results of pancreatic enzymes showed that most of the patients in both groups had lipase level <900 U/L and amylase level of <500 U/L one day before starting of feeding (both showed more than 3 folds of their normal range and this considered one of the inclusion criteria). This proves the reliability of our sample that included in our study. On the other hand, most of patients in both groups had decreasing level of serum lipase and amylase even after starting enteral feeding without significant difference between the two groups all over the studied duration. These results prove that both methods of feeding not affecting markers of local tissue destruction of pancreatic acini and not increasing the local inflammatory reaction in the pancreatic acini which laboratory assessed in our study by serum lipase and amylase. Moreover, there was no significant difference between the recorded serum levels of both lipase and amylase one day before feeding and

after starting of feeding. And again, this prove that starting of feeding does not increase the inflammatory process which already going on in the pancreatic acini and does not affect the fading of the inflammation in this area, as both serum lipase and amylase showed decreasing level even after starting feeding. As regard laboratory assessment of the inflammatory process in the interstitial pancreatic tissue, results of markers of tissue destruction (C-reactive protein, ESR, LDH, Pro-calcitonin, AST, and interleukin 8) showed that most of the patients in both groups had C-reactive protein level from 201-300mg/L, ESR level from 31-90 mm/h, LDH level from 201-700U/L, Procalcitonin level from 1.6-3 ng/ml, AST level from 41-1000 U/L and Interleukin 8 level from 11-60 pg/ml one day before starting of feeding. And most of patients in both groups had almost the same level of those indicators after starting of feeding. These results prove that both methods of feeding not affecting markers of inflammation in the interstitial pancreatic tissue and not increasing the inflammatory reaction in the interstitial pancreatic tissue which laboratory assessed in our study by those markers. Moreover, no significant difference was found between the level of those markers one day before feeding and after starting of feeding. this prove that starting of feeding does not increase the inflammatory process which already going on in the interstitial pancreatic tissue and does not affect the fading of the inflammation in this area.

## As regards the effect of both enteral feeding methods on the patient's tolerance to feeding:

In this pillar we recorded numbers of attacks of both postfeeding vomiting and post-feeding diarrhea as indicators. There was significant higher number of post-feeding vomiting in group A compared to group B in the first 6th days after starting of feeding. This indicate that NG feeding triggering post-feeding nausea and/or vomiting more than NJ feeding this could be explained by gastroparesis that occur from prolonged (3 days) fasting and also direct relation of gastric distension with nutrient with the occurrence of vomiting after feeding with NG more than feeding by NJ more over stress ulcer occur during stressful condition which is responsible on post feeding vomiting are more common to occur in the stomach than to occur in jejunum. There was significant higher number of postfeeding diarrhea in group B compared to group A allover the studied duration this indicate that NJ feeding associated by higher number of diarrhea than NG feeding and this explained

by either mal-absorption or osmotic diarrhea which considered a well-known complication of jejunal feeding.

# As regards the effect of the two enteral feeding methods on the patient's general condition:

APACHE II Score, hemodynamics of the patients in both groups (Mean arterial blood pressure and pulse) and arterial oxygen saturation used as indicator for general condition of the patients. For the APACHE II score most of the patients in both groups had score from 16 till >25 one day before feeding without significant difference between the two groups. Still the score showed no significant difference between the two groups after feeding rather there was improvement in that score with progress of feeding in both groups in all periods of the study with no significant difference. This proves that both methods of feeding do improve the general condition without significant difference and feeding by any of those methods do improve the general condition of the acute severe pancreatitis patients. For hemodynamics and oxygenation most of the patients in both groups had MABP between 80-95 mm/Hg, pulse from 80-100 beat/min and arterial oxygen saturation from 91->95% before feeding and all over the studied duration after feeding without significant difference between the two groups. Which prove no significant difference between the two methods of feeding as regard patient's hemodynamics and oxygenation and prove that feeding in acute pancreatitis do not compromise the hemodynamics and oxygenation of those patients.

# As regards effect of the two enteral feeding methods on achieving satisfactory nutrition parameters of the patients:

Post-feeding random blood sugar, albumin level and blood electrolytes used as indicators

Most of the patients had post-feeding RBS from 81-150 mg%, albumin level 2.1-4 gm/dL in the first 6 days after feeding and >4 gm/dL in the last 6 days of studied period, sodium level from 121-145 mEq/L all over the studied period, potassium level from <3 -5.5 mEq/L in the first 6 days after feeding and 3.1-5.5 mEq/L in the last 6 days of studied period. Which prove no significant difference between the two methods of feeding in achieving satisfactory nutrition parameters and also prove that feeding in those patients is very important to achieve satisfactory caloric input, maintain normal electrolyte balance and prevent fatal secondary bacterial translocation.

In summary all these results prove that enteral feeding by either method did not increase inflammatory process in acute severe pancreatitis. And do not affect or interfere with the natural process of fading of this severe inflammation. Rather improve the general condition of the patients and maintain electrolyte balance and caloric requirement. And prevent the development of fatal secondary bacterial translocation. And prove that delivery of nutrients either proximal (by NG) or distal to the duodeno-jejunal flexure (by NJ) do not affect the exocrine pancreatic secretion and does not increase acute inflammation in pancreas and thus does not increase local abdominal signs and post-feeding abdominal pain [7]. Moreover last studies prove that CCK release which can be done by either hormonal pathway carried out by presence of nutrients in the jejunum or by local axon reflex carried out by presence of nutrients in the duodenum both are suppressed in inflamed pancreas and thus both methods do not increase the exocrine function of inflamed pancreas. Added to these the fact that delivery of nutrients to jejunum does not prevent duodenal exposure to nutrient as a degree of reflux is inevitable [12-14].

Still many authors find that naso-jejunal feeding is the accepted way of enteral nutrition in acute severe pancreatitis (James Robert, Anthony et al. in 2011) [7]. NJ feeding was first published as an only source of enteral feeding by kalferent [22] windso [23] and the others [24,26] in acute severe pancreatitis and this description was supported by Stanga Z, Giger U et al. done in 2005 [6].

On the other hand, our results support the last published research work done by C.E. forsmark, J, Baillie et al. 2007 [25] in 2007 and in this study they find no statistical difference between enteral feeding by NG and NJ in severe acute pancreatitis this also supported by L. Gianotti, R, meieretal [27] study which done in 2009 and reach the same conclusion that NG feeding is a safe, simple, reliable less complicated way of enteral feeding in acute severe pancreatitis when compared with NJ feeding [19,20].

But the final agreement between all authors now that enteral feeding in acute severe pancreatitis is a must for nutritional support, given the satisfactory caloric requirement and prevent complication of total parenteral nutrition for those patients. Enteral feeding should be started with careful monitoring local abdominal signs, markers of destruction of pancreatic acini, and markers of destruction of interstitial pancreatic tissue. If there is any rise in all/one of those markers feeding should be stopped.

#### CONCLUSION

Both NG and NJ routes of feeding can be used in acute pancreatitis patients without any significant difference on clinical condition of patients with acute severe pancreatitis, patient's tolerance to feeding, patient's general condition, and both achieving satisfactory nutrition parameters.

#### REFERENCES

- 1. Banks PA, Freeman ML. Practice guidelines in acute pancreatitis. Am J Gastroenterol. 2006;101:2379-2400.
- 2. Spanier BW1, Dijkgraaf MG, Bruno MJ. Epidemiology, aetiology and outcome of acute and chronic pancreatitis: An update. Best Pract Res Clin Gastroenterol. 2008;22:45-63.
- 3. Yadav D, Lowenfels AB. Trends in the epidemiology of the first attack of acute pancreatitis: a systematic review. Pancreas. 2006;33:323-330.
- Fagenholz PJ, Ferna C, Harris NS, Pelletier AJ, Camargo Jr CA. Direct medical costs of acute pancreatitis hospitalizations in the United States. Pancreas. 2007;35:302-307.
- Shaheen NJ1, Hansen RA, Morgan DR, Gangarosa LM, Ringel Y, Thiny MT, Russo MW, Sandler RS. The burden of gastrointestinal and liver diseases, 2006. Am J Gastroenterol. 2006;101:2128-2138.
- 6. Stanga Z1, Giger U, Marx A, DeLegge MH. Effect of jejunal longterm feeding in chronic pancreatitis. JPEN J Parenter Enteral Nutr. 2005;29:12-20.
- 7. James Robert Anthony Skipworth, Dimitri Aristotle Raptis, Shalini Wijesuriya, Zudin Puthucheary, Steven WM Olde

Damink, Charles Imber, Massimo Malagò, Arjun Shankar. The Use of Nasojejunal Nutrition in Patients with Chronic Pancreatitis. 2011;12-6.

- Guan D, Ohta H, Green GM. Rat pancreatic secretory response to intraduodenal infusion of elemental vs polymeric defined formula diet. Journal of Parenteral and Enteral Nutrition. 1994;18:335-339.
- 9. Al Omran M, AlBalawi ZH, Tashkandi MF, Al Ansary LA. Enteral versus parenteral nutrition for acute pancreatitis. Cochrane database of systematic reviews. 2010.
- 10. Ammori BJ, Leeder PC, King RF, Barclay GR, Martin IG, Larvin M, McMahon MJ. Early increase in intestinal permeability in patients with severe acute pancreatitis: correlation with endotoxemia, organ failure, and mortality. Journal of gastrointestinal surgery. 1999;3:252-262.
- Rahman SH, Ammori BJ, Holmfield J, Larvin M, McMahon MJ. Intestinal hypoperfusion contributes to gut barrier failure in severe acute pancreatitis. Journal of Gastrointestinal Surgery. 2003;7:26-36.
- 12. Petrov MS, Pylypchuk RD, Uchugina AF. A systematic review on the timing of artificial nutrition in acute pancreatitis. British Journal of Nutrition. 2008;101:787-793.
- 13. Bakker OJ, van Santvoort HC, van Brunschot S, Ali UA, Besselink MG, Boermeester MA, Bollen TL, Bosscha K, Brink MA, Dejong CH, van Geenen EJ. Pancreatitis, very early compared with normal start of enteral feeding (PYTHON trial): design and rationale of a randomised controlled multicenter trial. Trials. 2011;12:73.
- 14. Takács T, Hajnal F, Németh J, Lonovics J, Pap Á. Stimulated gastrointestinal hormone release and gallbladder contraction during continuous jejunal feeding in patients with pancreatic pseudocyst is inhibited by octreotide. International journal of pancreatology. 2000;28:215-220.
- 15. Neumann DA, DeLegge MH. Gastric versus small-bowel tube feeding in the intensive care unit: a prospective comparison of efficacy. Critical care medicine. 2002;30:1436-438.
- 16. Abou-Assi S, Craig K, O'Keefe SJ. Hypocaloric jejunal feeding is better than total parenteral nutrition in acute pancreatitis: results of a randomized comparative study. The American journal of gastroenterology. 2002;97:2255-62.
- 17. Oláah A, Pardavi G, Beláagyi T, Nagy A, Issekutz Á, Mohamed GE. Early nasojejunal feeding in acute pancreatitis is associated with a lower complication rate. Nutrition. 2002;18:259-62.

- Eatock FC, Brombacher GD, Steven A, Imrie CW, McKay CJ, Carter R. Nasogastric feeding in severe acute pancreatitis may be practical and safe. International Journal of Pancreatology. 2000;28:23-29.
- 19. Montejo JC, Grau T, Acosta J, Ruiz-Santana S, Planas M, Garcíade-Lorenzo A, Mesejo A, Cervera M, Sánchez-Álvarez C, Núñez-Ruiz R, López-Martínez J. Multicenter, prospective, randomized, single-blind study comparing the efficacy and gastrointestinal complications of early jejunal feeding with early gastric feeding in critically ill patients. Critical care medicine. 2002;30:796-800.
- 20. Boivin MA, Levy H. Gastric feeding with erythromycin is equivalent to transpyloric feeding in the critically ill. Critical care medicine. 2001;29:1916-1919.
- 21. Nakad A, Piessevaux H, Marot JC, Hoang P, Geubel A, Van WS, Reynaert M. Is early enteral nutrition in acute pancreatitis dangerous? About 20 patients fed by an endoscopically placed nasogastrojejunal tube. Pancreas. 1998;17:187-193.
- 22. Kalfarentzos F. Enteral nutrition is superior to parenteral nutrition in severe acute pancreatitis: results of a randomised prospective trial. Br J Surg. 1997;83:349-53.
- 23. Windsor AC, Kanwar S, Li AG, Barnes E, Guthrie JA, Spark JI, Welsh F, Guillou PJ, Reynolds JV. Compared with parenteral nutrition, enteral feeding attenuates the acute phase response and improves disease severity in acute pancreatitis. Gut. 1998;42:431-435.
- 24. The use of enteral feeding in patients with severe acute pancreatitis( P.A. Banks ML. Freeman, R. Fass et al. Practicle guidelines in acute pancreatitis, American journal of gastroenterology, vol 101,no10pp2379-2400,2006)
- Forsmark CE, Baillie J. AGA Institute technical review on acute pancreatitis. Revista de gastroenterologia de Mexico. 2007;72:257-81.
- Gianotti L, Meier R, Lobo DN, Bassi C, Dejong CH, Ockenga J, Irtun O, MacFie J. ESPEN guidelines on parenteral nutrition: pancreas. Clinical Nutrition. 2009;28:428-35.
- Gianotti L, Meier R, Lobo DN, Bassi C, Dejong CH, Ockenga J, Irtun O, MacFie J. ESPEN guidelines on parenteral nutrition: pancreas. Clinical Nutrition. 2009;28:428-435.
- 28. Ranson JH, Shamamian P. Diagnostic standards for acute pancreatitis. World J Surg. 1997;21:136-142.