

The speed of eating and functional dyspepsia

Khalid A. Jasim Al-Khazraji*

CABM, FRCP, FACP MD

Abstract:

Background: dyspepsia is a common complaint, affecting about 26-41% of the general population. Investigations fail to identify organic cause in 50%–60% of functional dyspepsia. Little informations are available about whether quick eating is one of the risk factors for functional dyspepsia (FD).

Objectives: To study the association between quick eating and FD.

Materials and methods: The study was performed on 132 resident doctors in the medical city/Baghdad, by introducing a questionnaire derived from the Rome III criteria for diagnosis of FD and its subtypes and calculating the average daily meal duration for each one. The subjects were all Arabian, Iraqi, white resident doctors sharing the same type and amount of food. We excluded those with history of alcoholism, chronic NSAID use, documented organic gastrointestinal lesion, or with alarm features of dyspepsia, chronic systemic disease or previous history of dyspepsia.

Results: The prevalence of uninvestigated dyspepsia (UD) was 37.88%, post-prandial distress syndrome (PPD): 16.67%, and epigastric pain syndrome (EPS): 12.12%. Quick eating (independently on the BMI) was significantly associated with higher prevalence of FD and EPS, but not PPD. Higher BMI was associated with higher prevalence of FD and PPD (not EPS), smoking was associated with higher prevalence of FD and both subtypes, while age and gender had no effect on the prevalence of each.

Conclusion: prevalence of UD increases in quick eaters regardless the BMI, Smoking and higher BMI also increase the UD prevalence.

Key words: functional dyspepsia, uninvestigated dyspepsia, speed of eating, PPD, EPS.

JFac Med Baghdad
2016; Vol.58, No .4
Receive June. 2016
Accepted Nov..2016

Introduction:

Dyspepsia is a common term usually used to describe combinations of upper gut symptoms such as epigastric pain, burning, fullness, discomfort, early satiety, nausea, vomiting and belching. It was first recorded in mid-18th century and since then it has been widely used (1). Studies from United States, Great Britain, and other parts of the world have shown the prevalence of dyspepsia to be between 26% and 41%. (2,3) While only 20%–25% of these individuals seek medical care, dyspepsia accounts for 2%–5% of all consultations in primary care.(4)For gastroenterologists, dyspepsia accounts for between 20%-40% of consultations.(5) In 50%–60% of cases of dyspepsia, no cause is identified even after performing investigations, and patients are considered to have functional dyspepsia (7,8).The majority of people having dyspepsia do not seek doctors' opinion because they either ignore the symptoms or use self-medication(9), that's why the epidemiology of dyspepsia in the community is not easily predictable. Functional dyspepsia, previously known as non-ulcer or idiopathic dyspepsia, in absence of structural abnormality on upper GI endoscopy and metabolic or systemic causes explaining the symptoms.(10) The term "uninvestigated dyspepsia" refers to those patients who have the dyspeptic symptoms which are not yet investigated 11.

Materials and methods:

The study was performed on 139 resident doctors in the medical city in Baghdad. Among the 132 doctors completed the study,were 114 males and 18 females. All the doctors included in the study were Arabian Iraqi, white, and reside in the doctors' residence house and depend completely on the meals provided there. The exclusion criteria included having previously confirmed diagnosis of an organic cause explaining the symptoms, being a current or an ex-alcoholic, having a chronic comorbid illness(namely diabetes mellitus, chronic kidney disease, or psychiatric disease), having history of alarm features (Weight loss, Bloody stool, Anemia, Family history of inflammatory bowel disease, Colon cancer, and Celiac disease) and history of non-steroidal anti-inflammatory drug use for any cause. So participants were sharing the same amount, contents and timing of meals, and almost the same daily amount of hospital exercise as they work together, the same quality of life, in addition to be of the same race.Choosing this target group and these exclusion criteria were made to abolish the effect of other factors that may influence the prevalence of functional dyspepsia including dietary, racial, psychological factors and also the comorbidity, drugs, and alcoholism. Other factors including age, gender, smoking, and body mass index were dealt with in the study statistically. The seven doctors who were excluded from the study, four of them because they were using almost regular NSAIDs for different causes, one doctor was black, and the other two doctors were excluded

*Dept. of Medicine, College of Medicine, University of Baghdad.
drkhalidgit1959@gmail.com

because they have been investigated and diagnosed previously as to have peptic ulcers. Study protocol: This is a cross-sectional study, the survey was performed over 10 days period during October 2011 by introducing a questionnaire including demographic informations about age, gender, and smoking history, in addition to other questions designed to diagnose uninvestigated dyspepsia and its subtypes i.e. post-prandial distress syndrome and epigastric pain syndrome according to Rome III criteria(14). Participants were labeled to have uninvestigated dyspepsia when they: 1-report one or more of: Bothersome post-prandial fullness (feeling uncomfortably full after an ordinary-sized meal) more than one day/week, Early satiety (inability to finish a regular-sized meal) more than one day/week, Epigastric pain or burning one day/week or more. 2-Symptoms persistent on the last three months. 3- Symptom onset at least six months before. While the criteria of post-prandial distress syndrome were 1-The presence of all of: Bothersome post-prandial fullness (feeling uncomfortably full after an ordinary-sized meal) on more than one day/week, Early satiety (inability to finish regular-sized meal), more than one day/week. 2- Symptoms persistent for the last three months., 3-Symptom onset at least six months ago. The epigastric pain syndrome was considered to be present in participants who have: Presence of all of the following: 1- Epigastric pain or burning which should be: a-Intermittent (almost always disappear completely at the same day), b- in middle of abdomen (Not generalized or localized to other abdominal or chest regions), c- At least of moderate severity, d- Present on one day/week or more, e- Not relieved by defecation or passage of flatus (Never or rarely gets better after defecation), f- Not fulfilling criteria for biliary pain (gallbladder and sphincter of Oddi disorders). 2-Symptoms persistent for the last three months. 3-Symptom onset at least six months before. On each day a group of doctors selected according to the inclusion and exclusion criteria, the study was explained for them with detailed explanation of the questionnaire including the definition of the symptoms and about the demographic data needed Few days before the test They were asked to complete the questionnaire, the weight and the height were measured, and then the speed of eating was assessed for each participant by measuring the time he consumed for each of the three meals at the same day using a stop-watch.Finally the body mass index and the average meal-time were calculated for each participant. The statistical analysis was performed by using the IBM SPSS software version 19. Numeric variables with normal distribution were expressed with mean and standard deviation (SD). Categorical variables were expressed with numbers and percentages.Group comparisons were performed using the Pearson chi-square test and the t-test for continuous variables. A P-value <0.05 was considered statistically significant.

Results:

The mean age of the participants who completed the survey was 31.8 years ± 3.58. The mean body mass index was 26.04 ±

2.77 kg/m². 18 doctors (13.6%) were female and 114 (86.4%) were male; 18 doctors (13.6%) were smokers, 24 (18.2%) were ex-smokers, and 90 doctors (68.2%) were non-smokers. The mean meal duration of the participants were ranging from 4.8-35.2 minutes, (mean = 19.01 ± 4.47). Among the 132 participant, 50 subjects (37.88%) had met the Rome III criteria of functional dyspepsia (46 male and 4 female), 22 of them (16.67% of total) had met the criteria of post-prandial distress syndrome (18 male and 4 female), while 16 (12.12%of total) met the criteria of epigastric pain syndrome (all of them were male),6 participants (4%) had both EPS & PPD and 18 (14%) did not fulfill any subtype although they fulfilled the criteria of FD, as shown in figure 1.

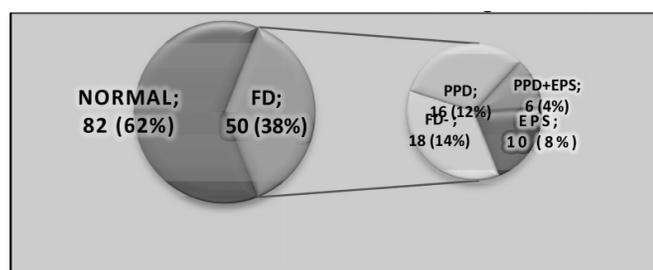


Figure 1:The proportions of healthy subjects, functional dyspepsia, and its subtypes among participants.

There was a statically significant correlation between the speed of eating and the prevalence of functional dyspepsia, i.e. quick eaters had a higher chance to have functional dyspepsia (P-value < 0.001), as shown in table 1, also there was a significant correlation between the quick eating and the prevalence of epigastric pain syndrome (EPS) as shown in table 2. While the post-prandial distress syndrome was not significantly related to fast eating in our study, table 3.

Table (1)

Relation of FD, EPS, PPDS with speed of eating								
Variable	Speed of eating						p value	
	<10	10-14.9	15-19.9	20-24.9	25-29.9	>=30		
Disease status								
FD	No	0	4	34	26	12	6	<0.001
	Yes	4	20	16	6	4	0	
EPS	No	4	14	48	28	16	6	<0.001
	Yes	0	10	2	4	0	0	
PPDS	No	2	20	42	28	12	6	0.345
	Yes	2	4	8	4	4	0	
Body mass index								
Normal	0	10	22	6	6	2	<0.001	
Over weight	4	16	26	26	6	0		
Obese	0	6	0	0	0	0		
Very obese	2	0	0	0	0	0		

Table (2)

The relation of BMI with FD, PPDS, and EPS						
Variables		BMI				p value
		normal	overweight	obese	very obese	
FD	No	34	46	2	0	0.038
	Yes	12	32	4	2	
PPDS	No	40	64	6	0	0.008
	Yes	6	14	0	2	
EPS	No	40	70	4	2	0.376
	Yes	6	8	2	0	

Table (3)

Comparison of the prevalence of FD, PPDS, and EPS between two age group & gender				
Variables		Age group		p value
		≤35	>35	
FD	No	68	14	0.141
	Yes	20	2	
PPDS	No	94	16	0.496
	Yes	18	4	
EPS	No	102	14	0.158
	Yes	12	4	

Comparison of the prevalence of FD, PPDS, and EPS between gender				
Variables		Gender		p value
		Male	Female	
FD	No	68	14	0.141
	Yes	46	4	
PPDS	No	96	14	0.496
	Yes	18	4	
EPS	No	98	18	0.09
	Yes	16	0	

Table (4)

The relation between FD, PPDS, and EPS and smoking status				
Variables		smoking status		p value
		non smoker	Smoker	
FD	No	62	6	0.004
	Yes	28	12	
PPDS	No	77	11	0.015
	Yes	13	7	
EPS	No	82	12	0.005
	Yes	8	6	

Variables		smoking status		P value
		non smoker	Ex-smoker	
FD	No	62	14	0.33
	Yes	28	10	
PPDS	No	77	22	0.431
	Yes	13	2	
EPS	No	82	22	0.932
	Yes	8	2	

Table 5

The relation of speed of eating and FD, PPDS and EPS regarding smoking status								
non-smoker and ex-smoker								
Variables		Speed of eating						p value
		<10	10-14.9	15-19.9	20-24.9	25-29.9	≥30	
FD	No	0	8	32	26	8	2	<.001
	Yes	5	18	9	2	4	0	
EPS	No	5	16	41	28	12	2	<.001
	Yes	0	10	0	0	0	0	
PPDS	No	3	24	34	28	8	2	0.022
	Yes	2	2	7	0	4	0	

Smoker						
Variables		Speed of eating				p value
		<10	10-14.9	15-19.9	20-24.9	
FD	No	0	0	4	2	0.123
	Yes	1	6	3	2	
EPS	No	1	4	5	2	0.784
	Yes	0	2	2	2	
PPDS	No	1	4	4	2	0.809
	Yes	0	2	3	2	

The assessment of the relation between the body mass index (BMI) and the prevalence of FD, EPS, and PPD, revealed that there is a significant relationship between the high BMI and the prevalence of FD (P-value = 0.038), PPD (P-value = 0.008), but there was no statistically significant relation between the BMI and the EPS (tables 2).

The effect of age on the prevalence of each syndrome was assessed by dividing the participants into two groups according to their age (≤35 and >35 years) and compare the prevalence in each group by the chi-square test. There was no significant relation between age and the prevalence of functional dyspepsia, epigastric pain syndrome, or post-prandial distress syndrome (tables 3). There was also no statically significant effect of gender on the prevalence of FD, PPD, and EPS as shown in (table 3). FD, PPD, and EPS have significant correlation with current smoking, as shown in (table 4), past smoking was not associated with increased prevalence of each (tables 5).

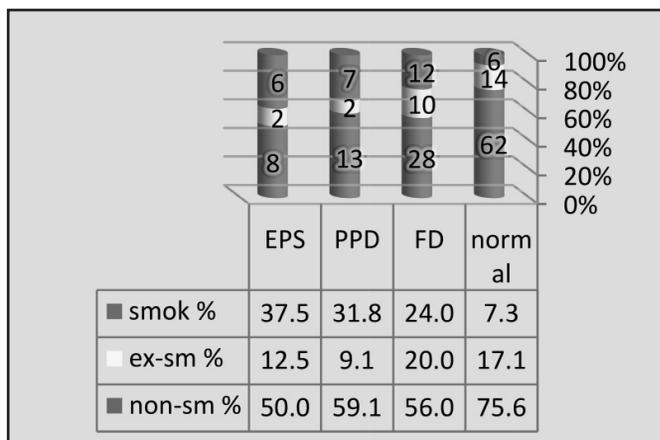


Figure 2: comparison of the proportions of smokers, ex-smokers, and non-smokers between the control group & those with uninvestigated dyspepsia.

After adjusting the results of the relationship between speed of eating to the functional dyspepsia with each of the other parameters, i.e. smoking, gender, and BMI, we got the following results:

The speed of eating in the group of non-smokers and ex-smokers still have a significant correlation with the prevalence of FD (P<0.001), PPD (P=0.022), and EPS (P<0.001). While in smokers, its effect was not significant on speed of eating (tables 4-5).

In female doctors, there was no significant relation of the speed of eating with FD or PPD and there was no female participant fulfilled the criteria of EPS, while in male doctors there was a relation between speed of eating and each of FD (P<0.001), EPS (P=0.009), but not with PPD (table 3).

We divided the participants according to their BMI into two groups, the group of normal BMI (18-25kg/m²) which included 46 participants, and the other 86 participants were of “above normal” BMI (including the overweight, obese and very obese participants, i.e. BMI>=25kg/m²), there was no underweight participant. In

normal BMI group, the speed of eating had a significant relation with the prevalence of FD (P<0.001), but not with PPD or EPS; while in those with “above normal” BMI, there was a significant correlation between eating speed and each of FD (P<0.001), PPD (P=0.008), and EPS (P=0.001) (table 6).

Table 6

Relation of speed of eating and FP, PPDS and EPS regarding BMI							
Variable		BMI >=25					p value
		<10	10-14.9	15-19.9	20-24.9	25-29.9	
FD	No	0	6	18	22	2	<0.001
	Yes	6	16	8	4	4	
PPDS	No	4	20	20	24	2	0.008
	Yes	2	2	6	2	4	
EPS	No	6	14	26	24	6	0.001
	Yes	0	8	0	2	0	
normal BMI							
Variables		10-14.9	15-19.9	20-24.9	25-29.9	>=30	p value
FD	No	2	18	6	6	2	<0.001
	Yes	8	4	0	0	0	
PPDS	No	8	18	6	6	2	0.551
	Yes	2	4	0	0	0	
EPS	No	6	20	6	6	2	0.066
	Yes	4	2	0	0	0	

Discussion:

There are few studies that focused on the speed of eating as an expected risk factor for dyspepsia. Actually, when we searched the PubMed Central website for such data, the only two studies that included the speed of eating were the study of Dong Hyun Sinn et al in Korea(17), and that of F Khademolhosseini et al in Iran(18). The prevalence of functional (uninvestigated) dyspepsia in our study was 37.88% which was higher than the rates reported in USA/2004 (15.8%)(19), in Korea/2010 (12%)(17), in New Zealand/2011 (34.2%)(20), in Japan/2011 (10.3%)(21), and in Iran/2010 (29.9%)(18), this difference may be caused by the difference in the definition of functional dyspepsia, or may denote population difference including racial, geographic, social, and also dietary factors. In our study there was a significant correlation between the fast eating and the prevalence of each of functional dyspepsia and the epigastric pain syndrome (P-value<0.001 for both), but no significant correlation with the post-prandial distress syndrome. Both other studies showed similarly a significant correlation between fast eating and functional dyspepsia, but none of them referred to the relation of fast eating with each of the subtypes of functional dyspepsia. This correlation means that the fast eating is a predictor rather than being a causative

factor for functional dyspepsia, because many known causes for functional dyspepsia (like psychological stress, personality type) (26) which are not included in our study, may influence the speed of eating as well. Regarding other parameters included in the study as risk factors for the functional or uninvestigated dyspepsia, the age of the participants was not associated with a significant increase in overall functional dyspepsia, post-prandial distress syndrome, or the epigastric pain syndrome. Most Asian studies failed to show the occurrence of UD in any particular age group (16). However, in an Indian study in Mumbai city, UD was commoner in older subjects (35% in subjects above 40 years and 26.5% in subjects below 40 years of age) (22). A study from Japan reported that FD is more common in younger age group, prevalence of FD was 13% and 8% in age groups below and above 50 years, respectively. (23) and another study in the USA showed decrement in the prevalence of dyspepsia with age advancement. (14) The age range in our study (26-41 years, mean 31.8 ± 3.58) is comparatively narrow, and this was a presumed advantage in our target population to decrease the effect of risk factors other than the speed of eating, therefore, although our results showed no relation between the age and functional dyspepsia, we can't confirm that in the Iraqi people without further studies on more discrete age groups. The increase in body mass index (BMI) had a significant relation with the increase in UD and PPD according to our results, with no specific effect on the prevalence of EPS. A lot of previous studies about risk factors of dyspepsia reported the high BMI as an independent risk factor, although Safae A, et al in Iran/2010, reported that there was no significant relation between FD and BMI (24). Neither FD nor any of its subtypes were associated with gender in our study, N J Talley et al in USA/1994, reported a female predominance in FD (14), but several population-based studies from Asia showed that frequency of UD was not related to gender (16), except one Japanese study that showed a male preponderance. (23) this discrepancy may reflect a social, habitual, or psychological factor that is widely different between the American, Japanese, and Iraqi populations. Cigarette smoking was significantly associated with higher prevalence of each of functional dyspepsia and its subtypes; however, past smoking was not significantly associated with increased prevalence of FD. Most of previous studies about this subject also reported a relation of functional dyspepsia with smoking, (12,14,18) and this may be due to increased basal acid gastric secretion related to smoking (25).

We also tried to adjust the association to the factors of gender and smoking, the results showed significant association between quick eating and uninvestigated dyspepsia in the male group but not in the females, and in the non- and ex-smokers but not in smokers. The high BMI is documented to be a risk factor for FD by many previous studies may be due to increased maximum gastric acid secretion that is associated

with high BMI (25). There is an association between quick eating and high BMI (table 36); therefore, to assess the independence of the association of quick eating and FD on the high BMI, we measured this association in different BMI groups and it was significantly higher rates in both normal and high BMI groups; therefore, the speed of eating can be considered as a risk factor of FD independent on BMI. The number of female participants in our study is 18, which was a small for statistical calculation of association, and the failure to confirm an association between quick eating and UD in female may be due to that or may be an association between the male sex and quick eating as risk factors; therefore, although Dong Hyun Sinn et al in Korea/2010 studied the association of quick eating and FD in females only and proved it, we think that further studies are needed. The smoking is proved also by many studies as a risk factor of UD, the non-association between quick eating and FD in the smokers in our study may be because smoking has an independent association with FD and again may be the small sample size of smokers (18 persons).

Conclusions:

The quick eating is associated with a higher prevalence of uninvestigated dyspepsia and for the epigastric pain syndrome and its association is independent on age, dietary contents, or BMI. Smoking is associated with a higher prevalence of UD, PPD and EPS, while high BMI is associated with increasing UD and PPD. There was no effect of the age or sex on prevalence of UD or its subtypes.

References:

- 1- *Therapeutics* 2006; Sep 1; 24(5):821-9.
- 2- Baron JH, Watson F, et al. Three centuries of stomach symptoms in Scotland. *Alimentary Pharmacology & Jones R. Dyspeptic symptoms in the community. Gut* 1989; 30: 893-8.
- 3- Talley NJ, Zinsmeister AR, et al. Dyspepsia and dyspepsia subgroups: a population-based study. *Gastroenterology* 1992; 102(4 pt 1):1259-68.
- 4- Brown C, Rees WD. *Dyspepsia in general practice. BMJ* 1990; 300:829-30.
- 5- Tebaldi M, Heading RC. *Clinical economics review: functional (non-ulcer) dyspepsia. Aliment Pharmacol Ther* 1998; 12:11-9.
- 6- Nicki R. Colledge, Brian R. Walker, Stuart H. Ralston; *Davidson's principles and practice of medicine, 21st ed. 2010. 851.*
- 7- Fisher RS, Parkman HP. *Management of nonulcer dyspepsia. N Engl J Med* 1998; 339:1376-81.
- 8- American Gastroenterological Association. *Medical position statement: evaluation of dyspepsia. Gastroenterology* 1998; 114:579-81.
- 9- S. L. Grainger, H. J. Klass, et al. *Prevalence of dyspepsia: the epidemiology of overlapping symptoms. Postgrad Med J.*

- 1994 March; 70(821): 154–161.
- 10-Tack J, Talley NJ, et al. Functional gastroduodenal disorders. *Gastroenterology* 2006; 130:1466-1479.
- 11-Lee Goldman, Andrew I. Schafer. *Goldman's Cecil medicine*, 24th edition 2012; pp:1002,1003.
- 12-Sanjay Nandurkar, Nicholas J. Talley, et al. Dyspepsia in the Community Is Linked to Smoking and Aspirin. *Arch Intern Med* 1998; 158:1427-1433.
- 13-N J Talley, A R Zinsmeister et al. Smoking, alcohol, and analgesics in dyspepsia and among dyspepsia subgroups. *Gut* 2004; 35:619-624.
- 14-Rome foundation. Rome III Disorders and Criteria. Official Website of Rome foundation (<http://www.romecriteria.org/>).
- 15-Talley N.J., Ruff K., et al; The Rome III classification of dyspepsia: will it help research? *Dig Dis* 2008;26: 203-209.
- 16-Uday C Ghoshal, Rajan Singh, et al. Epidemiology of Uninvestigated and Functional Dyspepsia in Asia. *J Neurogastroenterol Motil*, Vol. 17 No. 3 (July, 2011); 235-244.
- 17-Dong Hyun Sinn, Dong Hyuk Shin, et al. The Speed of Eating and Functional Dyspepsia in Young Women. *Gut and Liver*; Vol. 4, No. 2, June 2010, pp. 173-178.
- 18-F Khademolhosseini, D Mehrabani, et al. Prevalence of Dyspepsia and its Correlation with Demographic Factors and Lifestyle. *Middle East Journal of Digestive Diseases/ Vol.2 / No.1/ January 2010*, pp. 24-30.
- 19-Yasser Shaib, Hashem B. El-Serag. The Prevalence and Risk Factors of Functional Dyspepsia in a Multiethnic Population in the United States. *The American Journal of Gastroenterology*. 2004; 99(11)
- 20-Wyeth JW. et al; Functional gastrointestinal disorders in New Zealand; *J Gastroenterol Hepatol*. 2011 Apr;26 Suppl 3:15-8.
- 21-Ohara S, Kawano T, et al. Survey on the prevalence of GERD and FD based on the Montreal definition and the Rome III criteria. *J Gastroenterol*. 2011 May; 46(5):603-11.
- 22-Shah SS, Bhatia SJ, et al. Epidemiology of dyspepsia in the general population in Mumbai. *Indian J Gastroenterol* 2001;20:103-106.
- 23-Kawamura A, Adachi K, et al. Prevalence of functional dyspepsia and its relationship with *Helicobacter pylori* infection in a Japanese population. *J Gastroenterol Hepatol* 2001;16:384-388.
- 24-Solhpour A, Safae A, et al. Relationship between uninvestigated dyspepsia and body mass index. *East Afr J Public Health*. 2010 Dec; 7(4):318-22.
- 25-B. H. Novis, I. N. Marks, et al; The relation between gastric acid secretion and body habitus, *Gut*, 1973, 14, 107-112.
- 26-Hsu YC, Liou JM, et al. Psychopathology and personality trait in subgroups of functional dyspepsia based on Rome III criteria. *Am J Gastroenterol*. 2009 Oct;104(10):2534-42.