

## Clinical Analysis and Correlation between Benign Gastric Polypoid Lesions and Dyslipidaemia

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### Abstract

Non-neoplastic polypoid lesion of the gastrointestinal tract especially in the stomach becomes increasingly common with time. In our study, we tried to evaluate the correlation between the benign gastric polypoid lesions namely Hyperplastic and Fundic gland polyps and hyperlipidaemia. To the best of knowledge, the study of this kind has not been reported till now.

A single center prospective cross-sectional case-control study over 215 (two hundred and fifteen) selected patients divided into three study groups were done. According to our study, Gastric polyp / polyps' shares, a significant correlation with hyperlipidaemia and gastric polyp of 9 or more than that in number is a potentially significant condition associated with Hypertriglyceridaemia. Or in other sense, there is a correlation between the etiological factors like lifestyle, food habit, geographic and / or other influences that might be responsible for the development of Gastric polyp / polyps as well as hyperlipidaemia.

**Keywords:** Gastric polyp; Multiple gastric polyps; Gastric non neoplastic lesion; Hyperlipidaemia; Hypertriglyceridaemia

### Introduction

Gastrointestinal disorders are a general universal concern. Along with the common pathologies, Gastric Polyps are demanding special attention due to their presentation that mimics with the common pathologies of the digestive tract. For this reason, gastric polyps become a serious concern. Along with the clinical presentations and suffering of the patients, this may be associated some other underlying metabolic abnormalities that have not been yet considered. Correlation between "Colonic polyps and Dyslipidaemia" has been described in different publications. But to the best of our concern, no study has been reported with the correlation of Gastric polypoid lesion and Dyslipidaemia. On this point of view, we approach our study to evaluate and establish a possible clinical correlation between the existence of "Gastric polyps" and Dyslipidaemia. The correlation carries a valuable statement of patient's physical and underlying metabolic risk status.

### Patients and Method

A single center, prospective cross-sectional case-control study over the period of twelve months from May 2014 to April 2015 was done. A number of 215 (two hundred and fifteen) selected patients of 21 to 71 years of age presented in the "Department of Digestive Disease-II, The 1st affiliated Hospital of Jiamusi University, Jiamusi, Heilongjiang, P. R. China" were included. Informed consent was taken from all the patients included in this study. The study was approved by the local

ethical committee. For the case group patients with primary gastric polyp / polyps diagnosed by esophagogastroduodenoscopy were included in the study. For the risk factor group patients with existing risk factor/factors of hyperlipidaemia without Statin therapy were included. For the control group patients with normal esophagogastroduodenoscopy findings or gastritis or gastric ulceration without gastric polyp and with at least one the existing risk factor of hyperlipidaemia like type-2 diabetes mellitus, hypertension, and high BMI were included.

Patients with major cardiovascular and cerebrovascular disorders, carcinoma, major metabolic conditions with end stage or advanced hepatic and renal disorders, previous history of carcinoma, recurrent gastric polyp, on STATIN therapy and those who were unwilling to participate in the study were not included in the study. Patients presented with esophageal, duodenal, colonic and rectal polyps along with gastric polyps or those who had a history of familial polyposis and familial hyperlipidaemia were excluded from the study.

### For the study purpose, we divided the patients into three groups

Group A contains patients with diagnosed case of primary gastric polyp / polyps namely Hyperplastic and Fundic gland polyps, with or without gastritis / benign peptic ulceration and with no existing risk factor or underlying condition of Hyperlipidaemia.

Group B patients with normal gastric endoscopic findings or with gastritis or benign peptic ulceration, with at least one of the existing risk factor / condition / disease associated with Hyperlipidaemia.

Group C contains a pool of patients with normal gastric endoscopy findings or with gastritis or benign peptic ulceration, with none of the existing risk factor associated with hyperlipidaemia.

All patients' fasting lipid profile (blood HDL, LDL, Triglyceride and Cholesterol), colonoscopy report, blood glucose level, HbA1C, renal function test (Serum Urea, Creatinine, albumin and Urine routine), Liver function test (ALT, AST, Serum Bilirubin, Prothrombin time), ECG and full blood count were monitored. As a routine procedure, all the patient went through screening for Hepatitis B and C virus before endoscopy. Patients height, Blood pressure, pulse, weight, and waist circumference were measured carefully. BMI was calculated. Patients' drug, family, and past disease history were taken carefully. In a patient with gastritis and gastric ulceration histopathological report was taken into consideration. Normal level of serum lipids were defined as follows: Triglyceride 0.28-1.70 mmol/L, Cholesterol 2.30-6.20 mmol/L, HDL (High Density Lipoprotein Cholesterol) 0.90-1.68 mmol/L, LDL (Low Density Lipoprotein Cholesterol) 1.8-4.0 mmol/L. The results were compared with a statistical analysis. A P value of <0.05 was considered statistically significant.

## Results

Baseline characteristics of our study group. Case group: A Risk group: B, and Control Group: C (Table 1).

	Group A (N=71)	Group B (N=50)	Group C (N=94)
Gender			
Male	35 (49.3)	30 (60)	53 (56.3)
Female	36 (50.7)	20 (40)	41 (43.6)
Age (in years)	52.1 ± 9.9	57.1 ± 10.1	48.2 ± 12.1
LDL (mmol/L)	3.12 ± 1.2	3.7 ± 1.2	2.8 ± 0.7
HDL (mmol/L)	1.58 ± 0.41	1.5 ± 0.4	1.41 ± 0.3
TG (mmol/L)	1.7 ± 1.01	2.1 ± 1.6	1.13 ± 0.5
Cholesterol (mmol/L)	5.2 ± 2.1	5.02 ± 1.9	4.65 ± 1.0
BMI (kg/m <sup>2</sup> )	22.31 ± 1.82	24.17 ± 2.95	22.02 ± 1.85
Associated condition			
Gastritis			
Present	39 (54.9)	39 (78)	86 (91.4)
Absent	32 (45)	11 (22)	8 (8.5)
Ulceration			
Present	4 (5.6)	20 (40)	24 (25.5)
Absent	67 (94.4)	30 (60)	70 (74.5)
Hemorrhage			
Present	4 (5.6)	4 (8)	8 (8.5)
Absent	67 (94.4)	46 (92)	86 (91.5)
DM			
Present	0	20 (40)	0

Absent	100	30 (60)	100
HTN			
Present	0	25 (50)	0
Absent	100	25 (50)	100
Overweight			
Present	0	17 (34)	0
Absent	100	33 (66)	100

**Table 1:** Baseline characteristics of our study groups. Data are reported as number (%) or mean ± SD.

Our study includes 215 patients (Male 118 and female 97). In group A n=71, among them male represents 35(49.3%) and female 36(50.7%). In Group B n=50 male 30(60%) and female 20(40%). In Group C n=94 male 53(56.3% and female 41(43.6%). The mean age of the Group A, B, and C were correspondingly 52.1, 57.1 and 48.2 years. Mean value of Low-density lipoprotein (LDL), High-density lipoprotein (HDL), Triglyceride (TG) and Cholesterol in all the three groups are 3.12, 1.58, 1.7, 5.2 mmol/L in Group A; 3.7, 1.5, 2.1, 5.02 mmol/L in Group B and 2.8, 1.41, 1.13, 4.65, 22.02 mmol/L in Group C. Mean BMI in Group A, B and C is 22.31, 24.17 and 22.02. Associated Endoscopic findings like Gastritis, gastric ulceration and gastric hemorrhage was present 39(54.9%), 4(5.6%) and 4(5.6%) in group A; 39(78%), 11(22%) and 4(8%) in group B and 86(91.4%), 8(8.5%) and 8(8.5%) in group C. Diabetes mellitus, hypertension and overweight was present in 20(40%), 25(50%) and 17(34%) in group B (high risk group). As shown in Table 1. Comparison of the mean difference between our study groups regarding lipid profile and BMI values (Table 2).

	Group A (N=71)	Group B (N=50)	Group C (N=94)
LDL (mmol/L)	3.12 ± 1.2	3.7 ± 1.2*	2.8 ± 0.7
HDL (mmol/L)	1.58 ± 0.41*	1.5 ± 0.4	1.41 ± 0.3
TG (mmol/L)	1.7 ± 1.01	2.1 ± 1.6	1.13 ± 0.5*
Cholesterol (mmol/L)	5.2 ± 2.1	5.02 ± 1.9	4.65 ± 1.0
BMI (kg/m <sup>2</sup> )	22.31 ± 1.82	24.17 ± 2.9*	22.02 ± 1.85

**Table 2:** Comparison of the mean difference between our study groups regarding lipid profile and BMI values. Data are reported as number (%) or mean ± SD. \*P< 0.05: (Between B and A, B and C for LDL, Between A and C for HDL, Between C and A, C and B for TG, Between B and A, B and C regarding BMI).

With regarding the mean value of LDL, HDL, TG, Cholesterol and BMI of the three study groups, statistically, significant correlation was found in LDL level (P<0.05) in between Group B and A; Group B and C. For HDL level (P<0.05) between the case group A and control Group C. For TG level (P<0.05) between control Group C with case group A and risk Group B. Significant P value <0.05 is also seen between group B and A, Group B and C regarding BMI as shown in Table 2. Comparisons of lipid profile in Group A regarding gender, age and no. of gastric polyps (Table 3).

Total no=71	LDL (mmol/L)	HDL (mmol/L)	TG (mmol/L)	Cholesterol (mmol/L)
Gender				
Male (n=35)	3.22 ± 1.14	1.61 ± 0.51	1.69 ± 1.0	5.38 ± 2.0
Female (n=36)	2.99 ± 1.20	1.55 ± 0.27	1.72 ± 1.16	5.0 ± 2.22
Age group				
Less than 40 (n=12)	3.39 ± 1.54	1.61 ± 0.35	1.16 ± 0.82	4.69 ± 2.31
41-60 (n=45)	3.07 ± 1.03	1.62 ± 0.45	1.70 ± 0.96	5.08 ± 2.02
More than 60 (n=14)	2.97 ± 1.28	1.42 ± 0.23	2.18 ± 1.42*	5.96 ± 2.16
No. of gastric polyps				
0- 4 (n=21)	3.4 ± 1.28	1.68 ± 0.32	1.33 ± 0.59	5.22 ± 1.28
5-8 (n=45)	3.0 ± 1.16	1.52 ± 0.44	1.74 ± 1.10	4.97 ± 2.20
9 or more (n=5)	2.74 ± 0.26	1.7 ± 0.29	2.90 ± 1.65*	7.0 ± 2.03

**Table 3:** Comparison of lipid profile in group A regarding gender, age and no. of gastric polyps. Data are reported as number (%) or mean ± SD. \*P< 0.05: No. of gastric polyps group (9 or more) compared to (0-4) for TG; Age group (more than 60) compared to (0-40) for TG.

Data analysis of case Group “A” shows significant correlation for TG level p<0.05 regarding age group (>60years subgroup and <40 years sub-group) and no. of gastric polyps (9 or more gastric polyp subgroup and 0-4 gastric polyp sub-group), as shown in Table 3. Pearson correlation coefficients regarding- age, BMI and No. of gastric polyps with lipid profile in Group A (Table 4).

	LDL (mmol/L)	HDL (mmol/L)	TG (mmol/L)	Cholesterol (mmol/L)
Age (in years)	-0.185	-0.163	0.342**	0.187
BMI (kg/m <sup>2</sup> )	-0.315**	-0.098	-0.106	-0.111
No. of gastric polyps	0.014	-0.026	0.192	-0.197

**Table 4:** Pearson correlation coefficients regarding- age, BMI and No. of gastric polyps with lipid profile in Group A. \*\*P< 0.01 for TG with age and LDL with BMI.

Total No. n=50	LDL (mmol/L)	HDL (mmol/L)	TG (mmol/L)	Cholesterol (mmol/L)
Gender				
Male (n=30)	3.76 ± 1.12	1.43 ± 0.72	2.18 ± 1.68	4.76 ± 1.87
Female (n=20)	3.59 ± 1.37	1.56 ± 0.39	1.92 ± 1.45	5.42 ± 2.06
Age group				
Less than 50 (n=14)	3.34 ± 1.30	1.32 ± 0.26	2.06 ± 1.74	5.24 ± 1.63
More than 50 (n=36)	3.83 ± 1.17	1.56 ± 0.48	2.08 ± 1.54	4.94 ± 2.08
DM				
Present (n=20)	1.62 ± 0.46	4.20 ± 0.95*	2.27 ± 1.60	5.08 ± 2.06
Absent (n=30)	1.41 ± 0.41	3.36 ± 1.27	1.95 ± 1.58	4.99 ± 1.92

In our study group “A,” we found a significant positive correlation between age and TG level (r=0.342, P=0.004). Regarding BMI, we found a negative correlation between BMI and LDL (r=-0.315, P=0.008) as shown in Table 4. There is also a positive correlation between age and number of gastric polyps (r=0.274, P=0.021). So according to this study, there is an increase in a number of gastric polyps with an increase in age.

Comparison of lipid profile in group B regarding- gender, age, HTN, DM and overweight, gastritis and gastric ulcer (Table 5).

HTN				
Present (n=26)	1.37 ± 0.43	3.79 ± 1.32	2.22 ± 1.71	4.61 ± 1.86
Absent (n=24)	1.62 ± 0.43	3.59 ± 1.11	1.92 ± 1.44	5.47 ± 1.99
Overweight				
Present (n=17)	1.46 ± 0.40	3.55 ± 1.18	2.32 ± 1.58	4.53 ± 1.62
Absent (n=33)	1.51 ± 0.47	3.77 ± 1.25	1.95 ± 1.59	5.28 ± 2.09
Gastric ulcer				
Present (n=20)	1.58 ± 0.46	4.12 ± 1.15*	1.87 ± 1.43	5.24 ± 2.04
Absent (n=30)	1.43 ± 0.42	3.42 ± 1.20	2.21 ± 1.68	4.88 ± 1.92
Gastritis				
Present (n=39)	1.57 ± 0.47*	3.61 ± 1.15	1.96 ± 1.51	4.84 ± 1.93
Absent (n=11)	1.21 ± 0.13	3.99 ± 1.45	2.50 ± 1.81	5.68 ± 2.0

**Table 5:** Comparison of lipid profile in Group B regarding- gender, age, HTN, DM and overweight, gastritis and gastric ulcer. Data are reported as number (%) or mean ± SD. \*P< 0.05.

Data analysis of Group B shows a significant correlation between the high level of HDL with Diabetes mellitus and gastric ulcer (P<0.05). A significant correlation between Gastritis and LDL level is also noted (P<0.05) as shown in Table 5.

Regarding the status of associated conditions in our study Groups A, B and C gastritis and gastric ulcer differed significantly across

groups P<0.05 (Table 6). Table 7 showed a significant correlation (P<0.05) between case Group A, risk factor group B with the control Group C regarding hyperlipidaemia.

	Case (Group A)	Risk (Group B)	Control (Group C)	P value
Gastritis [n (%)]	39 (54.9)	39 (78)	86 (91.4)	0.01
Gastric ulcer [n (%)]	4 (5.6)	20 (40)	24 (25.5)	0.01
Gastric hemorrhage [n (%)]	4 (5.6)	4 (8)	8 (8.5)	0.773

**Table 6:** Correlations between the study groups regarding associated gastric pathology (Gastritis, Gastric ulcer and gastric hemorrhage).

	Group A	Group B	P value	Group A	Group C	P value	Group B	Group C	P value
Hyperlipidaemia [no (%)]	53 (74.6)	43 (86)	0.129	53 (74.6)	9 (9.57)	0.00*	43(86)	9 (9.57)	0.00*

**Table 7:** Correlations between the study groups (A and B, A and C, B and C) regarding the gross Hyperlipidaemia. \*P< 0.00 for the correlation between the study group A with control group C and risk factor Group B and control Group C.

In case group A out of 71 patient with multiple Gastric polyp 53 patient (74.6%), in Risk factor Group B 43 out of 53 patients (86%) and in control group 9 patients out of 94 patients (9.57%) showed a gross hyperlipidaemia status. Data analysis of Table 7, the percentage of patient presents with Hyperlipidaemia among the different study groups shows the following results. Correlation between case Group A (Gastric polyp Group) and Group B (Risk factor group) is not significant (P=0.129). Correlation between Gastric polyp Group A and control Group C, Risk factor Group B and Control group C is found statistically significant. P=0.00 in both cases. According to our study, this explains that the endoscopic evidence of gastric polyp is a physical

status that bears a risk of hyperlipidaemia. Or in other sense, there is a correlation between the etiological factors like lifestyle, food habit, geographic and / or other influences that might be responsible for the development of Gastric polyp / polyps as well as hyperlipidaemia. These findings do not establish hyperlipidaemia as a cause of gastric polypoid lesion or vice versa. A positive correlation is also seen in the number of the gastric polyps and LDL level. But serum Cholesterol and HDL level showed a negative correlation with the number of the polyps in this study. A significant negative correlation was found between BMI and LDL level.

## Discussion

Because of the consequence of obesity and sedentary lifestyle, Metabolic syndrome and Dyslipidaemia becomes an epidemic of this time. Cardiovascular disease, stroke and diabetes are potent clinical entities with significant mortality and morbidity. As because of such outcome the underlying pathogenesis and progression of the associated clinical pathologies have become increasingly important scientific point of interest.

A gastric protruding or polypoid lesions are commonly encountered in about 2% esophagogastroduodenoscopy [1]. They might be benign or malignant in character and can be classified as epithelial and subepithelial lesion. The malignant lesions include carcinoid, early gastric carcinoma and advanced gastric carcinoma. Benign gastric lesions include "Gastric polyps" and these are subdivided into Hyperplastic, Fundic gland polyp and gastric adenoma. Our interest in this study has been concentrated on the benign gastric polypoid lesion or gastric polyps namely Hyperplastic and Fundic gland polyps. To analyze the significance of the correlation between Gastric non-neoplastic lesion and Dyslipidaemia we designed our study in three groups. In our study, the case group A (Gastric polyp) contains total 71 patients where all the cases of gastric polyps included in this study were of less than 1 cm in size. Group B represents patients suffering from diseases or condition which is associated and a potent underlying cause of Dyslipidaemia / Hyperlipidaemia like type 2 Diabetes mellitus, Hypertension, and high BMI etc. The control group C contains a pool of patients devoid of any of the risk factors or disease that might be associated with Dyslipidaemia / hyperlipidaemia and no evidence of gastric polypoid lesions in endoscopy. Hyperlipidaemia is defined as the high level of any of the blood lipids, Low-density lipoprotein Cholesterol (LDL), Cholesterol (CHOL), Triglyceride (TG) and High-density lipoprotein Cholesterol (HDL). In our research we defined normal levels of different blood lipids as following: Triglyceride 0.28-1.70 mmol/L, Cholesterol 2.30-6.20 mmol/L, HDL 0.90-1.68 mmol/L, LDL 1.8-4.0 mmol/L.

Statistical correlation among our three study groups under the terminology of hyperlipidaemia evaluates a significant correlation ( $P<0.05$ ) between the Case group A, Risk factor group B with the control group C. We also found a significant correlation ( $P<0.05$ ) between the TG level and multiple gastric polyps of more than 9 in number. There was a positive correlation between age and gastric polyp, triglyceride level and a number of the gastric polyps. According to our current study, this represents evidence of gastric polyp / polyps as a significant underlying factor or condition that might share some common etiological phenomena of Hyperlipidaemia in this study group population. This might include lifestyle, food habit, regional and or other influences that share same common etiology with hyperlipidaemia. And gastric polyps of 9 or more than that in number are potentially important physical characteristics of underlying hypertriglyceridaemia. Beside that the increase in a number of polyps shows an increase in the risk of Hypertriglyceridaemia and an increase in Age increases the risk of gastric polyps. This clinically interesting finding suggests a guide to the physician for a curious look to evaluate hyperlipidaemia in the case of patients with gastric polypoid lesions. Significant positive correlation between age and triglyceride level represents age itself is a factor for increased level of Triglyceride (TG) (Tables 3 and 4). Our finding partly supports the meta-analysis done on Chinese population by Huang, Y and others where they observed a trends of higher prevalence of TC, TG, and LDL-C abnormalities were observed in studies of older participants ( $\geq 30$  years old) compared to

those studies that enrolled younger participants ( $\geq 18$  years old), the highest estimate of 49.3% [2]. Beside that the mean value of the three study groups Serum lipids (LDL, HDL, TG, Cholesterol) showed a statistically significant correlation in LDL level ( $P<0.05$ ) in between Group B and A; Group B and C. For HDL level ( $P<0.05$ ) between the case group A and control Group C. For TG level ( $P<0.05$ ) between control Group C with case Group A and risk Group B (Table 2).

One interesting associated finding of our study is the significant negative correlation between the LDL with BMI ( $P<0.05$ ) and a negative correlation between serum HDL, TG and Cholesterol level in Group B patients. This is probably due to increasing diet awareness and the particular food habit of the overweight people included in this study of this part of the northern China. Also a high HDL level was found significantly correlated ( $P<0.05$ ) with type 2 Diabetes mellitus in group B. Besides this Gastric ulceration also showed a significant correlation ( $P<0.05$ ) with high HDL level and Gastritis was found significantly correlated with serum LDL level as shown in Table 5.

Due to the variation of clinical presentation gastric polyps remains a mystery in the clinical practice. Such cases may be clinically silent or present as features of gastrointestinal symptoms like dyspepsia, reflux, ulceration and even hemorrhage. Etiology of the gastric polyp depends on the type. Benign gastric polypoid lesions especially "Hyperplastic polyps" are mostly concern either as idiopathic in origin [3] or related with *Helicobacter Pylori*-related gastritis [4]. Fundic gland polyps are considered to be associated with familial adenomatous polyposis or Peutz-Jeghers syndrome [5,6]. Though a relationship with Fundic gland polyps and long time PPI use has been reported in many kinds of literature [7,8]. Besides polypoid lesions other focal gastric pathologies like Xanthomatosis has been reported to for carcinomatous appearance [9-11] and correlation with dyslipidaemia. A study conducted in Korea by Sun Young Yi on 771 patients has concluded that dyslipidemia and atrophic gastritis are found to be related to Gastric Xanthomatosis [12]. Other than the stomach, polypoid lesions in the intestine especially in the colon bears a high clinical significance. This is a universally accepted fact that most of the colorectal carcinomas arise from adenomatous polyps [13]. And a high dietary fat intake has considered as an influential factor that tends to promote colon carcinogenesis [14-17].

Beside to their carcinogenic character Intestinal polyps namely the colonic polyps have been identified for a significant relationship with various metabolic and systemic concerns. Inflammation, insulin resistance, and oxidative stress are the most potent identified factors described for the formation of colorectal neoplasms associated with metabolic syndrome and an increased tendency towards the male gender [18-20]. The association of colorectal adenoma and the inflammatory cytokines has also been reported in the literature [21-23]. Several studies has been reported that metabolic syndrome especially diabetes shows evidence with associated with colorectal adenoma and cancer [24-28]. Besides that a study on Chinese population by Liu, Chiu-Shong et al. revealed that increased likelihood of colorectal adenoma, a premalignant lesion of colorectal cancer, was associated with Metabolic syndrome. Of them, central obesity and Dyslipidaemia were independently increased the risk of colorectal adenoma [29]. The similarity of common lifestyle risk factors of insulin resistance with colorectal cancer suggests that hypertriglyceridemia, as well as hyperinsulinemia, is associated the development of colon cancer [30-33]. A 14-year multicenter prospective cohort study by Ahmed et al. [33] concludes with a positive association between metabolic syndrome and colorectal cancer, especially in men but not in



women [34]. Another cross-sectional study of Korean population showed that an increased risk of colorectal adenoma was associated with metabolic syndrome and, particularly with an increased risk of incurring proximal lesions, multiple adenomas, and advanced adenoma [35]. However literature published by Kim, Yeong-Ju et al. conclude with the finding that for the prevalence of colon polyps Dyslipidaemia was not a significant factor but it had a significant association with the prevalence of adenomatous colon polyps in men [36].

Obesity was found to be associated with colorectal cancer in several studies [37]. On the contrary multiple studies identified "Abdominal obesity" as a higher risk for colon carcinoma than body mass index [38-42]. Multiple animal studies showed evidence that suggest an effect of triglyceride on the development of adenomatous polyps [43,44]. A study on Japanese population evaluated that, serum levels of cholesterol and triglycerides are increased in patients with adenoma compared to those without adenoma while serum levels in patients with cancer are not elevated compared to those with adenoma. This indicates that a high fat intake that increases serum lipid levels may play a role in the development of adenoma but not in the development of carcinoma from adenoma.

## Conclusion

The gastric polypoid lesion is a concern that is taken with serious attention by the clinicians due to their usual presentations; those mimic the diagnosis and their carcinomatous character. But the concern for the underlying correlated conditions such as Hyperlipidaemia can increase the awareness of the physicians as well as the patients to prevent future circumstances. Our study has several limitations, which include a number of the patients and regional influences. A gastric polyp is not a common presentation and most of the cases of gastric polypoid lesions present with other major metabolic and systemic conditions. Despite that with the best of our effort we selected our study group solely presenting with Gastric polyp. According to our current study, we suggest evaluating the lipid profile of the patients representing with a gastric polyp. We also suggest a high priority for an endoscopic evaluation for gastric polypoid lesions of relatively older patients presenting with the various gastrointestinal symptoms with or without previous or family history of the gastric polyp. As because our findings conclude that, Gastric polyp shares a significant correlation with hyperlipidaemia and gastric polyp of 9 or more than that in number is a potentially significant condition associated with Hypertriglyceridaemia. Besides that, an increase in Age increases the risk gastric polyp along with the number of polyps. Our research findings do not establish hyperlipidaemia as a cause of gastric polyp or vice versa.

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