Coexisting Gastric and Duodenal Ulcers

A Review

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WHETHER GASTRIC ULCER (GU) and duodenal ulcer (DU) are two manifestations of one disease or two different diseases has given rise to much conflicting opinion. In recent years considerable emphasis has been placed upon characteristic differences between the two lesions. Divergences in age and sex incidence, experimental production, gastric motility and acid secretory patterns, clinical manifestations, and blood group types[1, 2] suggest that the two lesions are etiologically dissimilar and tend to occur in mutually exclusive patient populations. On the other hand, several authors in the past have adduced evidence pointing either to a general ulcer "diathesis"[3, 4] or to a fundamental identity in the etiology of all peptic ulcers with differences in localization related only to local conditioning factors.[1, 2, 5]

The presence of coexisting† gastric and duodenal ulcers (CGDU) in the same patient is significant in this regard since it naturally raises a question as to whether GU and DU are in reality confined to mutually exclusive patient populations. Their coexistence, if sufficiently frequent, would further suggest that the fundamental pathological mechanism responsible for the production of peptic ulceration may affect either stomach or duodenum, or both organs simultaneously, depending upon the influence of secondary factors.

Although the finding of CGDU is in fact not unusual, its frequency has varied greatly in different series. In 1917 Carman[6] reported the existence of 16 CGDU (2.5%) in a surgical series of 629 peptic ulcer patients, while in 1926 Wilkie[7] observed 42 such cases (14%) in a similar series of 300 cases of peptic ulcer.

In 1953 Feldman[8] reviewed most of the data on CGDU available in the literature up to that time. He found that the incidence of CGDU varied

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†Also referred to as coincident,[7] concomitant,[10] associated,[50] combined,[9, 45, 46] and concurrent[6] gastric and duodenal ulcers.

strikingly with the source of these data; i.e., whether from postmortem, surgical, or radiological surveys. He concluded that the composite incidence was lowest in unselected radiological and postmortem series (0.15% and 0.32%) and highest in surgical series of benign and malignant GU's (17.4% and 24.6%). When analyzed in terms of the total number of peptic ulcer cases, the postmortem incidence of CGDU was 6.5% and the surgical incidence 7.0%.

In the past 10 years several large studies of peptic ulcer cases including CGDU have been added to the literature. It will be the purpose of this paper to review the information available on this type of lesion as it has been reported during this period with regard to comparative statistical incidence, age and sex of patients, acid secretory patterns, clinical course, pathogenesis, complications, and therapy. This information, based on a total of 1490 cases of CGDU, may increase our understanding of this type of lesion and the mechanism of peptic ulceration of the stomach and duodenum.

REVIEW OF FINDINGS

Incidence of CGDU

In Table 1 the general incidence of CGDU is expressed as the percentage incidence of CGDU in the total number of GU's and DU's in all series containing both lesions for the 10-year period reviewed. This varies from a low of 3.0% in Mangold's[9] combined radiological and surgical series of 5253 cases of peptic ulcer to a high of 29.5% in Aagaard's[10] surgical series. The composite average for all surveys during the past 10 years is 6.6%, which is not significantly different from the composite average of 6.5% in the postmortem group and 7.0% in the surgical group analyzed by Feldman[8] in 1953.

Table 1 also lists the incidence of CGDU in benign GU. This varies from 7.9% to 38.3% with a composite mean of 23.5%, approximately 3 times that seen in the general peptic ulcer population. This incidence is also somewhat higher than that of 17.4% reported by Feldman.[8] This increase over the past 10 years may be due either to a greater awareness of the disease and greater accuracy in the diagnosis of CGDU in patients with GU or to some change in the natural history of peptic ulcer.

The average incidence of CGDU in DU cases is 7.3%, as listed in Table 1. It is about 1/3 that noted for CGDU in benign GU's, and practically identical with that found for CGDU in the total number of peptic ulcer cases.

Table 2 contains a supplementary list of cases of CGDU drawn from

TABLE 1. INCIDENCE OF CGDU IN PEPTIC ULCER, DU, AND BENIGN GU

Author	*Yr.*	*Type of survey*	*No. CGDU*	*Total No. PU cases*	*% CGDU in total PU cases*	*No. DU*	*No. CGDU*	*% CGDU in total DU*	*No. GU*	*No. CGDU*	*% CGDU in total GU*
Glenn & Harrison[51]	1951	Surgical	9	289	3.1	215	9	4.2	83	9	10.8
Rauch[52]	1952	Surgical	90	845	10.6	682	90	13.2	253	90	35.6
Smith *et al.*[53]	1953	Radiological & surgical	—	—	—	—	—	—	879	173	19.7
Tanner[14]	1954	Surgical	—	—	—	—	—	—	—	—	25.0
Johnson[50]	1956	Surgical	32	347	9.2	203	29	14.3	116	29	25.0
Comfort *et al.*[11]	1957	Surgical	—	—	—	—	—	—	779	184	23.6
Mangold[9]	1958	Radiological & surgical	157	5253	3.0	3901	157	4.0	—	—	—
Bernardo *et al.*[22]	1958	Surgical	—	—	—	—	—	—	469	52	11.1
Aagaard *et al.*[10]	1959	Radiological & surgical	122	413	29.5	100	2	2.0	313	120	38.3
Marks & Shay[15]	1959	Surgical	—	—	—	—	—	—	33	10	30.3
Watkinson[18]	1960	Postmortem	227	1848	12.3	1356	227	16.7	677	227	33.5
Gelb & Becker[45]	1962	Radiological & surgical	53	1435	3.7	1120	36	3.2	315	25	7.9
TOTAL	1951-1962	Mixed	690	10430	6.6	7577	550	7.3	3917	919	23.5

TABLE 2. INCIDENCE OF CGDU IN PEPTIC ULCER*

Author	Yr.	No. CGDU	Total No. PU cases	% incidence of CGDU in all PU cases	Total No. GU	% incidence CGDU in GU	Total No. DU	% incidence CGDU in DU
Aird *et al.*[36]	1954	113	3011	3.8	1015	11.1	1851	6.1
Clarke *et al.*[37]	1955	48	1545	3.1	438	11.0	1059	4.5
Køster *et al.*[40]	1955	30	1047	2.9	337	8.9	680	4.4
Peebles Brown *et al.*[38]	1956	38	1980	1.8	300	12.6	1642	2.3
Buckwalter *et al.*[41]	1956	69	1839	3.7	469	14.7	1301	5.3
Billington[39]	1958	83	1236	7.2	961	8.6	192	43.2
TOTAL		381	10,658	3.6	3520	10.8	6725	5.7

*Statistics drawn from blood group studies.

reviews of large peptic ulcer populations in which the primary purpose of the investigation was the correlation of blood groups with peptic ulcer disease. They are included in this review because of the large number of cases studied but are listed separately since in all these series the detection of CGDU was not the primary object of investigation and only those cases with available blood group information were included. As may be seen, the composite incidence of CGDU in all peptic ulcer cases and in cases of benign GU analyzed in this manner is considerably lower than that noted in the studies previously cited.

FREQUENCY OF GASTRIC MALIGNANCY IN CGDU

Table 3 shows the incidence of malignant gastric ulcer in cases of single GU and CGDU. While the incidence of malignancy in single GU ranged

TABLE 3. FREQUENCY OF GASTRIC MALIGNANCY IN GU AND CGDU

	Single Gastric Ulcer (GU)				*Coexisting gastric and duodenal ulcers (CGDU)*			
	No. cases			% incidence of malignancy	*No. cases*			% incidence of malignancy
Author	*Total*	*Malignant*	*Benign*		*Total*	*Malignant*	*Benign*	
Bernardo *et al.*[22]	464	47	417	10.12	55	3	52	5.45
Smith *et al.*[53]	810	71	739	8.76	190	17	173	8.94
Comfort *et al.*[11]	802	207	595	25.81	203	19	184	9.35
TOTAL	2076	325	1751	15.65	448	39	409	8.71

from 8.8% to 25.8% (mean 15.7%), it was considerably lower in CGDU —ranging from 5.5% to 9.4%, with a mean of only 8.7%. It may be concluded, therefore, that the gastric ulcer in CGDU is less frequently malignant than GU alone.

Sequence of Ulcers in CGDU

As shown by serial radiography, the DU was the first lesion observed in 59.8% of the cases (Table 4). In a small number of cases (mean, 8.0%) the reverse order was observed, with the GU appearing first. In the remaining cases both ulcers either appeared simultaneously or their sequence of appearance was undetermined.

Anatomical Location of the GU in CGDU

Table 5 lists the location of the gastric lesion in CGDU. The region of the pyloric antrum and angulus, as well as the middle 1/3 of the lesser curvature, were the sites most often involved. These are also the most common sites of involvement of single GU.

TABLE 4. SEQUENCE OF ULCERS IN CGDU

		DU first		*GU first*		*Simultaneous or undetermined*	
Author	*No. cases*	*No.*	*%*	*No.*	*%*	*No.*	*%*
Johnson[16, 50]	135	70	52.0	7	5.2	58	42.9
Mangold[9]	157	91	58.0	8	5.0	58	37.0
Aagaard *et al.*[30]	120	94	78.3	18	15.0	8	6.7
Gelb & Becker[45]	53	23	43.4	4	7.5	26	49.0
Total	465	278	59.8	37	8.0	150	32.3

TABLE 5. LOCALIZATION OF GU IN CGDU

		Angulus pyloric & Antral region		*Middle 1/3 of lesser curvature*		*Upper 1/3 of lesser curvature*		*Fundus & body of stomach*	
Author	*No. cases*	*No.*	*%*	*No.*	*%*	*No.*	*%*	*No.*	*%*
Mangold[9]	157	45	28.6	82	52.2	30	19.1	—	—
Comfort *et al.*[31]	184	107	57.8	76	41.4	—	—	—	—
Gelb & Becker[45]	53	22	42.0	—	—	—	—	26	50.0
Billington[39]	114	23	20.2	—	—	—	—	91	79.8
Total	508	197	38.8	158	31.1	30	5.9	117	23.0

It should be noted, however, that in the series of Comfort *et al.*[11] some differences in the distribution of GU in CGDU and single GU were found. In the CGDU group the GU was located in the area of the pylorus and pyloric antrum in 57.8% of the cases and along the middle ⅓ of the lesser curvature in 41.4%. In the group of single benign GU, however, only 48.9% were found in the pyloric and antral areas and 48.7% along the middle ⅓ of the lesser curvature.

In one series[11] multiple gastric ulcers were found to occur almost twice as often in patients with coexisting DU's as in patients with gastric localization of the lesion only, and in other series[9, 45] multiple GU's were found in 9% and 14% of the cases of CGDU.

Distribution of CGDU According to Sex and Age

Figure 1 shows the distribution of CGDU according to sex and age. The male:female ratio varied from 2.5:1 to 4.9:1 (mean 3.9:1) in various series. This mean figure is identical with the ratio found for CGDU (3.9:1) in Central Middlesex Hospital[9] but closer to the figure for GU found there for 271 GU's (2.4:1) and 691 DU's (6.8:1).[12]

Age distribution for CGDU shows uniformity in different series. The peak incidence occurs between 40 and 70 years, which parallels the age distribution of GU. Statistical data at the Central Middlesex Hospital[12] showed that the age of peak incidence for GU and CGDU was between 45 and 55, while that for DU was between 35 and 45. The postmortem survey at Leeds General Infirmary[13] shows a similar distribution. The study by Comfort[11] also reveals a similar age distribution for CGDU and GU patients (50.3 and 53.4 years, respectively). Tanner[14] noted in his

Author	No. of Cases	M	F	Ratio M:F	Age: >25	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	>74
Mangold[9]	157	125	32	3.9:1		5		27		52		43		28		
Watkinson[13]	227	174	53	3.3:1	3		13		22		52		72		49	15
Johnson[16]	119	96	23	4.2:1		2		13		34		43		19		7
Gelb et al[45]	53	38	15	2.5:1		2		4		7		16		15		9
Comfort et al[11]	184	153	31	4.9:1	Mean Age (Male) = 49.9						Mean Age (Female) = 50.8					
Rauch[52]	90	74	16	4.6:1												
Totals	830	660	170	3.9:1												

Fig. 1. Distribution of CGDU according to sex and age.

group of surgical cases that the age at operation of the CGDU patients was close to that of GU patients.

Thus, it would appear that both the sex ratio and the age of peak incidence for CGDU tend to approximate those for GU.

Gastric Acid Secretion

Acid secretion in patients with CGDU has been shown to range between that of patients with GU and DU or to approximate that of DU patients.

Marks and Shay,[15] using the augmented histamine stimulation technique of Kay (MHR and MAO), and Comfort *et al.*[11] using the Ewald meal, reported acid values for CGDU intermediate between those observed in GU and DU. In two other studies, one using the measurement of nocturnal secretion[16] and the other a 24-hour test meal,[17] acid output was found to approach the DU pattern of hypersecretion. In only one study, using submaximal doses of histamine, were secretory values reported to be in the normal range.[45]

Diagnosis and Clinical Course

The diagnosis of CGDU has been made chiefly by X-rays or at operation (Table 6).

While gastrointestinal X-rays are relied upon most frequently, the difficulties inherent in their interpretation in cases of CGDU have been underscored.[11, 16] In 184 cases of CGDU[11] the GU was missed by the radiologist in 37 instances (20%) and the DU in 41 cases (22%). These errors were attributed (1) to the focusing of the radiologist on either the gastric or duodenal lesion, to the exclusion of a second one, and (2) to the difficulty encountered in visualizing small gastric (prepyloric) ulcers in the presence of varying degrees of pyloric obstruction.

Surgical exploration yields greater diagnostic accuracy. The difficulties inherent in this method, similar to those confronting the radiologist, have

TABLE 6. MEANS OF DIAGNOSIS OF CGDU

Author	*No. cases*	*Surgery*		*X-ray*	
		No.	*%*	*No.*	*%*
Aagaard *et al.*[10]	118	108	91.5	10	8.5
Morrison & Feldman[18]	21	—	—	21	100.0
Mangold[9]	145	6	4.1	139	96.0
Smith *et al.*[53]	190	—	—	190	100.0
Total	474	114	24.0	360	76.0

also been emphasized.[11, 16] This is especially true for the surgeon operating in an emergency such as perforation or hemorrhage.

The diagnosis of CGDU on purely clinical grounds is difficult. With the exception of a tendency toward a longer duration of symptoms, the main characteristic, pain, was generally indistinguishable from that due to single GU or DU.[9, 11]

In evaluating the recurrence of ulcer symptoms, one has to consider that in a number of such cases the recurrence of symptoms may well be due to the development of a new ulcer rather than to the activation of an old one. In 41 of 104 patients with CGDU (39%) in whom the DU was documented as the earlier lesion to appear, a symptom-free interval of at least one year was noted between the onset of symptoms and their recurrence, at which time a new gastric lesion was demonstrated.[9] Similarly, in another series of 21 DU patients, 6 experienced a recurrence of symptoms from 1 to 27 years later, at which time the earlier DU was radiologically shown to have completely healed and symptoms were due to the development of a new GU.[18]

The activity of the lesions at the time of diagnosis or postmortem is listed in Table 7. At the time of clinical diagnosis the most frequent findings were active lesions at both sites (41.7%) or a healed DU coexisting with an active GU (39.5%). Tanner[14] estimated from his own surgical material that the combination of a healed DU and active GU accounted for 90% of the cases of CGDU coming to surgery. In those cases in which the DU was fully active the GU was often in an early or subacute stage. The most frequent postmortem finding was a healed lesion at both sites (38.3%) and somewhat less frequently a healed DU and active GU (33.9%).

Thus, the natural history of CGDU is probably as follows: the DU is

TABLE 7. ACTIVITY OF ULCERS IN CGDU AT TIME OF DIAGNOSIS OR AT AUTOPSY*

Author	*No. cases*	*Both ulcers active*		*Healed DU, active GU*		*Active DU, healed GU*		*Healed DU, healed GU*	
		No.	%	*No.*	%	*No.*	%	*No.*	%
Johnson[16]	110	26	23.6	75	68.2	6	5.5	3	2.7
Morrison & Feldman[18]	21	15	71.5	6	28.5	—	—	—	—
Aagaard *et al.*[10]	79	14	17.7	64	81.0	—	—	1	1.3
Mangold[9]	157	98	62.4						
Tanner[14]					90.0				
TOTAL	367	153	41.7	145	39.5	6	1.6	4	1.1
*Watkinson[13]	227	49	21.6	77	33.9	14	6.2	87	38.3

the earlier lesion (Table 4), and as it progresses to chronicity or healing, the GU develops. This is shown by the fact that the two largest clinical groups (81.2%) are formed of patients in whom the DU and GU are both active or in whom the DU has progressed to complete healing, leaving an active GU.

Since the coexistence of healed lesions at both sites was the most frequent postmortem finding (38.3%), it appears that in a number of these patients the course of the disease is favorable. Either spontaneously or as a result of therapy, complete healing of both lesions takes place. That the group with active lesions at both sites also consists of patients in whom the clinical course is not so favorable is suggested by the fact that they form a substantial proportion (21.6%) of CGDU at autopsy.

PATHOGENESIS

Both the primacy in time of the DU and the frequent finding of acid hypersecretion have strongly influenced the concepts advanced to explain the pathogenesis of CGDU.

It has been suggested that patients with CGDU have a primary DU, the course of which is complicated by the development of pyloric obstruction.[16, 19] The latter may be due to spasm, scarring, and deformity of the pyloric region following DU, leading to impaired gastric emptying and gastric retention. While in patients with pyloric stenosis and normal acid secretion intragastric buffering mechanisms may be adequate in neutralizing acid retained in the stomach, in patients with acid hypersecretion normal buffering may be inadequate and result in the formation of a secondary GU.[16]

An additional mechanism operating under these circumstances has been suggested by Dragstedt.[19] This was based upon his investigations into the different phases of gastric acid secretion in DU patients. According to Dragstedt, the DU in the majority of these patients is usually the result of the vagally mediated, high basal acid secretion. When the DU is followed by pyloric obstruction, antral stasis will develop, stimulating in turn the release of gastrin,[20] thus further increasing gastric acid secretion. Once again, the combination of pyloric obstruction and acid hypersecretion will favor the formation of a secondary GU.

In support of this view, Johnson[16] has reported that of 119 patients with CGDU, 76 (64%) had clinical, surgical, or radiological evidence of gastric retention, including 39 (33%) with well-marked pyloric stenosis at surgery. He also noted that of 331 patients admitted for pyloric stenosis, 52 (16%) had GU. Of 16 cases with pyloric channel ulcer disease reported by Burge *et al.*,[21] 9 (56%) had, in addition, a benign GU along

the lesser curvature. Although details regarding gastric emptying time were not given for all cases, the authors state, "There can be little doubt that the associated simple lesser curve gastric ulcer is caused by gastric stasis in the pyloric channel syndrome."

The presence of pyloric obstruction in patients with CGDU has not been a constant finding, however. Using a retention meal, Aagaard *et al.*[10] found delayed gastric emptying in only 13 (16.0%) of 81 patients with CGDU, and of 86 patients subjected to surgery, only 10 (11.6%) showed evidence of pyloric stenosis. Mangold[9] found either clinical or radiological evidence of gastric retention in only 25 (19%) of 131 patients with CGDU, and at surgery pyloric stenosis was present in only 12 (9.2%). Of 51 cases of CGDU reported by Bernardo,[22] only 9 (17.6%) had evidence of pyloric obstruction on admission, and of 53 cases reported by Gelb and Becker,[45] gastric retention was present in only 5 (9.4%).

The fact that the GU is the first lesion to appear in a small number of cases also suggests that the development of pyloric obstruction is not the only pathogenetic mechanism responsible for the development of CGDU.

Decreased gastric mucosal resistance to acid hypersecretion has also been incriminated in the pathogenesis of CGDU. Several authors have in the past suggested that the healing of DU coincides with the development of an associated gastritis in the hypersecreting stomach.[14, 23, 24] Healing under these circumstances may actually be due to the fall in acid secretion following inflammatory involvement of the stomach. It has been suggested further that persistent gastritis, together with the development of atrophic mucosal changes, lowers mucosal resistance as well and predisposes to gastric ulceration.[14] This would apply especially to cases of long-standing acid hypersecretion.[15]

Thus, the pathogenesis of CGDU may be no different from that of GU or DU occurring alone. The DU is the first lesion to appear in the majority of cases since gastric mucosal resistance to acid hypersecretion is normally greater than that of the duodenum. The development of a coexisting GU would then depend upon the degree and duration of the gastritis and acid hypersecretion associated with the DU. The fact that, generally, free acid values are lower in patients with CGDU than in those with DU may be a consequence either of the associated gastritis or of the "zonal" gastritis surrounding the GU.[25]

While there is indeed evidence that chronic, and even atrophic, gastritis occurs to a greater or lesser degree in all patients with DU, this incidence varies greatly with the diagnostic method used. It has been described as occurring consistently,[26–28] in slightly less than ½ of the cases,[29, 30] occasionally,[23, 25, 31, 32] and infrequently.[33] Where the antral region was ex-

amined histologically in surgical and postmortem specimens, however, this was almost invariably the site of active inflammation.[26–28]

Why the associated antral gastritis should progress to frank ulceration in only a relatively small number of DU patients is not clear. Pyloric obstruction is not likely to be a critical factor in view of its absence in many cases of CGDU. The small number of cases in which the GU is the earlier lesion to appear is also unexplained by this sequence of events.

It is possible that the role of inheritance may be significant in this regard. Doll and Kellock[34] found that 11 patients with CGDU had 18 relatives with peptic ulcer, of whom 4 were siblings who had CGDU. In several of Mangold's[4] cases of CGDU near relatives were shown to be similarly affected.

According to Johnson,[35] a preponderance of his patients with CGDU was of the blood group O, most characteristic for British patients affected with peptic ulcer and particularly DU.[36–38] Similar findings were reported by Billington[39] in a large group of Australian patients. In some series,[37, 38, 40, 41] while the majority of patients with CGDU were of the blood group O, the statistical significance of the quantitative difference between each of the blood groups in cases of CGDU was not determined. In 2 of these,[36, 37] although the blood type O was found more frequently among peptic ulcer patients than in a reference group and the association between the blood group and peptic ulcer was statistically significant, no significant difference was found between DU and GU.

Thus, the proportion of relatives with ulcers at 2 sites appears to be greatly increased when propositi are similarly affected, and there is a tendency toward the predominance of blood group O in patients with CGDU.

While the significance of these findings is not certain, it may be that a critical factor in the pathogenesis of CGDU is wholly or partly genetically conditioned.

COMPLICATIONS

Pyloric Obstruction

Pyloric obstruction with gastric retention is a more or less prominent complication of CGDU and has already been discussed in the section on pathogenesis.

Hemorrhage

In one series of 157 patients,[9] 59 (37.6%) had a total of 89 hemorrhages. In 91 patients of this series in whom the sequence of development of the lesions was known (the DU preceding the GU), there were 36 instances of bleeding in 27 patients, all occurring within a year follow-

ing the diagnosis of the combined lesion. The GU proved to be the source of the bleeding in 16 (60%) of these patients and in the remainder the bleeding site was not determined. In at least 45 patients (28.5%) hemorrhage took place while both the GU and DU were coexistent for a little more than 1.5 years. Thus, at least every third patient with CGDU bled at least once, and most of them within a short time following the diagnosis. This incidence was higher than that in the period preceding the CGDU and higher than that observed in a comparable time period in patients with GU or DU alone.[42–44]

In another series of 119 patients with CGDU,[16] 47% experienced at least one episode of hemorrhage and, of these, 20% had several episodes. The incidence of hemorrhage in the general GU population from which these patients were drawn was only 8% for the same time period. Thirteen patients with CGDU underwent emergency gastrectomy for active bleeding and GU was the source of the bleeding in all of them. In 2 patients, bleeding was from several acute gastric ulcerations.

Of 53 cases of CGDU reported by Gelb and Becker[45], 28 experienced at least one hemorrhage and 9 of these experienced more than one.

Hemorrhage is therefore a prominent complication of CGDU, occurring in from 37.6% to 53% of the cases and often within a short time following the development of the two coexisting lesions. The most frequent source of bleeding was the GU, accounting for 60% to 100% of those cases in which the bleeding site was identified.

Perforation

The incidence of perforation was 24 times in 22 patients (14%) in one series of 157 cases of CGDU,[9] and 5 times in 3 patients (5.6%) in another series of 53 cases.[45] With two exceptions, all perforations occurred in the duodenum.

MORTALITY IN CGDU

Johnson[16] placed the mortality as high as 10% in his surgical series in contrast with an over-all mortality of 4% noted in a series of 3000 gastrectomies. Mangold[9] reported 5 deaths (3.2%) directly attributable to ulcer disease in his group of medical and surgical patients. In a postmortem survey, Watkinson[13] noted 85 deaths (37%) due to ulcer disease in 227 patients with CGDU, of which 73 were in men and 12 in women. This compared with 531 deaths (32.8%) due to ulcer disease in 1621 patients with either GU or DU alone. In the group of patients with CGDU, death was most often caused by active lesions at both sites or to a chronic gastric ulcer in the presence of a duodenal scar.

It appears, therefore, that CGDU tends to run a more severe course with a higher mortality rate than that due to either GU or DU alone.

TREATMENT

Because of the more severe course and higher incidence of complications in patients with CGDU, greater emphasis has been placed upon surgical treatment. Partial gastric resection has emerged as the procedure of choice with no apparent difference in the results obtained with either the Billroth I or Billroth II operations.

Walters[46] observed 73 patients with CGDU for a period of 5 years, 54 (74%) of whom had been subjected to Billroth II gastrectomy and 19 (26%) to Billroth I. There were no recurrences in either group during the period covered by the study. Hickenbotham[47] performed Billroth I operations on 19 patients with CGDU with one operative and 2 postdischarge deaths. There was one relapse during a 2-yr. follow-up period. Johnson[50] strongly urges that all cases of CGDU be treated by surgical procedures designed to control hypersecretion of acid. This should consist either of extensive Billroth II resection or a more limited resection combined with vagotomy.

Following the hypothesis that in CGDU the GU is caused by an augmented gastrin phase of gastric secretion in patients with a primary DU complicated by pyloric obstruction and gastric retention, several authors described the treatment of 4 patients with CGDU by means of vagotomy alone or vagotomy combined with gastroenterostomy or pyloroplasty. In all cases, the ulcers healed promptly. For similar reasons, Burge *et al.*[21] treated 9 patients with pyloric channel ulcers and associated lesser curvature ulcers with vagotomy and pyloroplasty. Immediate results were described as encouraging.

In one series of 157 patients,[9] 82 (52%) were subjected to one or more operations, of which 68 were partial gastrectomies. In another series,[45] 30 (57%) of 53 patients with CGDU underwent partial gastrectomy. In the latter series the indication for surgery in most of the cases was hemorrhage. In the remaining 23 of these 53 cases, medical therapy was applied with good results including healing of the gastric ulcers as confirmed by repeated GI series.

No detailed, controlled study of the results of medical versus surgical therapy is available and therefore no adequate comparison of these two modalities is possible at this time. However, the autopsy finding of a high proportion of healed lesions at both sites (38%) suggests that in a large group of these patients healing does occur and that such a comparative study is warranted.

SUMMARY AND CONCLUSIONS

The literature on coexisting gastric and duodenal ulcers (CGDU) covering the past 10 years, encompassing a total of 1490 cases, is reviewed. This form of peptic ulceration comprises 6.6% of all peptic ulcers, 23.5% of all benign gastric ulcers (GU), and 7.3% of all duodenal ulcers (DU).

When cases of CGDU are compared with those of single GU or DU, the following features of CGDU are apparent:

1. The DU is the earlier lesion to appear in the majority of cases.
2. The acid secretory pattern in the majority of cases is either intermediate between that of DU and GU or tends to approach that of DU.
3. The most common clinical feature is an active lesion at both sites, the next most common finding being a healed DU and an active GU. In contrast, the most frequent autopsy finding in these cases has been a healed lesion at both sites.
4. The incidence of complications in CGDU, especially hemorrhage, is higher than that for GU or DU alone. In hemorrhage, the GU is the most frequent site of bleeding; in cases of perforation, the duodenum is the main site.
5. The incidence of malignancy is lower in the GU of CGDU than in GU occurring alone.
6. As in cases of GU occurring alone, the most common sites of GU in CGDU are the pyloric antrum and middle $\frac{1}{3}$ of the lesser curvature.
7. The sex ratio and age distribution for CGDU are intermediate between those for DU and GU occurring alone, with a tendency for the sex ratio (males predominating) and the age distribution to parallel that for GU.
8. The duration of pain in CGDU is somewhat longer than in GU or DU alone. The characteristics of the pain itself, however, are indistinguishable from those due to either GU or DU alone.
9. Because of the more severe course of CGDU, the higher incidence of complications, and the higher mortality, emphasis in recent years has been on surgical treatment. There have been no controlled studies, however, of the usefulness of medical therapy in this condition, although there is evidence to suggest that it may be of value in selected cases.

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