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Case Report

On Musculo-Membranous Pyloric Bands

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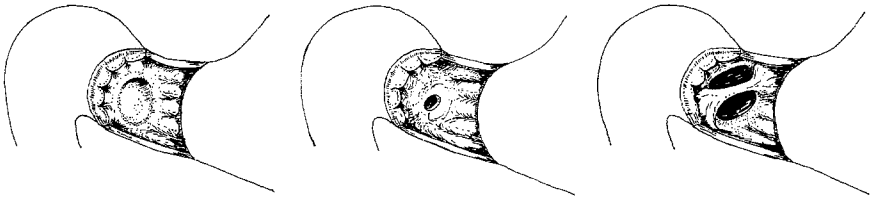
Summary. Three pyloric bands observed within the period from 1965 to 1972 are described. To date, two such findings have been described in the international literature. The aetiology and the mechanism of development are still largely unknown. Chronic recurrent ulcers, perforating cysts or diverticula, sequelae of epithelial occlusions occurring during the embryonic development of the gastro-intestinal tract and genuine duplication of the pylorus are discussed as possible aetiological factors. A pyloric band involving all the layers of the wall of the pylorus must clearly be considered a duplication. If such completeness of the wall does not present, one is possibly dealing with a diaphragm pierced by two apertures.

There are numerous investigations and experimental studies dealing with the function, anatomy and the pathological changes of the pylorus. If we neglect pyloric hypertrophy, it is only rarely that mention is made of malformations or congential obstructions in this section of the gastrointestinal tract. Predominantly, occlusal membranes in the region of the pyloric canal are described. The survey of the literature compiled by Gerber (1964) includes 14 phyloric diaphragms in children and 21 in adults and also 3 cases of atresia in children. By 1967 this list had been extended by a further 5 pyloric diaphragms and an anatomical peculiarity, namely, a pyloric band, this additional material being listed by Smith and Tuttle (1969).

The term diaphragm is applied to a wall, covered on both sides with mucosa and comprising the muscularis mucosae and loose connective tissue which, when located antrally separates the antrum from the remainder of the stomach, when located prepylorically separates the pylorus from the antrum and when sited pylorically, separates the duodenum from the pylorus (Fig. 1a). This diaphragm can be complete, that is without breaks, or incomplete, that is, pierced centrally or excentrically (Fig. 1b). The term gastroduodenal (pyloric) band (Smith and Tuttle, 1969) or pylore double (Christien *et al.*, 1971) is used to describe a strip-like "bridge" covered with mucosa and built up of lamina muscularis mucosae, connective tissue and/or muscle, which traverses the pylorus from the posterior to the anterior wall, thus building two channels (Fig. 1c). The literature available to us revealed only two descriptions of a pyloric band (Smith and Tuttle, 1969; Christien, Branthomme, Volny, Dechamps and Morice, 1971). In the present study, three findings similar to the pyloric band are reported on and several mechanisms of development indicated for discussion.



a b c

Fig. 1. a Complete pyloric diaphragm. b Incomplete pyloric diaphragm. c Pyloric band

Findings

Case 1. In September, 1966, a 58-year-old male patient was admitted to the municipal hospital of Karlsruhe[1] suffering from daily attacks of severe pain in the upper abdomen. Conservative treatment of the radiologically-demonstrated gastric ulcer, sited in the prepyloric region on the lesser curvature of the stomach, was unsuccessful so that surgery became necessary. The resection of the stomach revealed a covered perforated gastric ulcer that had given rise to a small abscess.

The resected preparation formed a large stomach "sleeve" of 8×7 cm in size. In the pyloric canal there was a shallow ulcer having the size of a small coin. From the margins of the ulcer and at right angles to the lesser curvature, a 0.5 cm wide band of mucosa crossed the ulcer like a bridge.

Case 2. On the 4th of September, 1970, a 65-year-old man was admitted to the Surgical Clinic of the University of Erlangen[2]. The patient's history up to this 57th year was uneventful. From then on, gastric ulcers appeared which, in the following period recurred again and again and gave rise to the usual pattern of complaints. Numerous X-ray examinations in the following years confirmed an initially moderate, but, in the further course of the disease, increasing scarred shrinking of the antrum.

A further pyloric ulcer on the side of the lesser curvature finally lead to a two third resection of the stomach after Billroth. The operation and the post-operative recovery were without complications.

During the examination of the resected "sleeve" of the stomach by the pathologist[3], a 7 mm thick and 2.5 cm long bridge of mucosa located in the region of the pylorus was immediately found. This "bridge" traversed the lumen of the pyloric canal from the anterior wall to the posterior wall. Under this bridge there was a 17×12 mm large and 6 mm deep ulcer extending to the posterior wall of the duodenum and covered with a greasy substance (Fig. 2).

The ulcer had infiltrated into the subserosa, which was thickened by connective tissue, and gave the impression of being a strongly progressive, hard, peptic duodenal ulcer. At the oral pole, the mucosa was predominantly of the antral type and at the aboral pole duodenal mucosa. A cross-section of the pyloric band showed a connective core which was covered on all sides with true pyloric and duodenal mucosa. Most of the mucosa was of the duodenal type, the antral mucosa being more poorly represented (Fig. 3).

Case 3. Mr. S. J. was admitted to the Medical Clinic of the University of Erlangen in November, 1971 at the age of 53 years. For the past 7 years, he had suffered from burning pains in the epigastrium, in particular in the fastin state. Barium examination of the gastrointestinal tract confirmed the presence of a gastric ulcer, an unusually wide and asymmetrical pylorus and an atypical peristaltic alteration of the prepyloric antrum. During oesophagogastro-bulboscopy, the prepyloric antrum proved to be deformed.

1 We should like to thank Prof. K. Spohn, M.D. for allowing us to make use of the patient's case history.

2 We should like to thank Prof. G. Hegemann, M.D. for allowing us to examine the case history.

3 Particular thanks are due to Prof. P. Hermanek, M.D. for providing us with the documents of case 2.

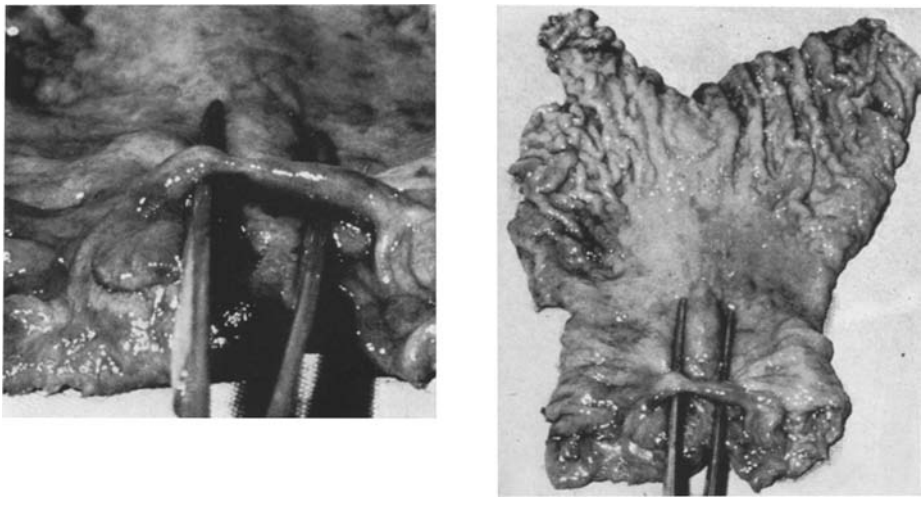


Fig. 2. Case 2, 65-year-old man: mucosal bridge in the region of the pyloric canal, surgical preparation

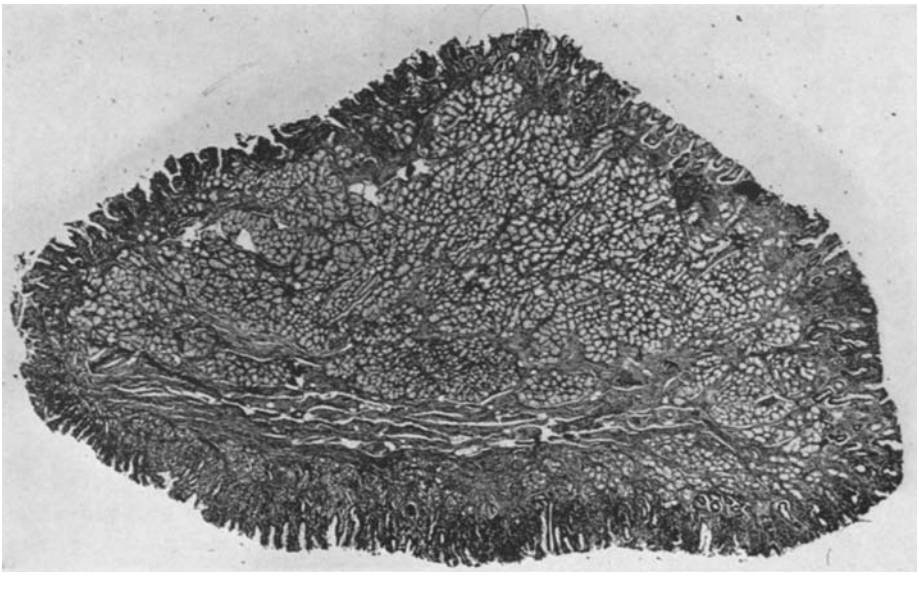


Fig. 3. Cross-section of the mucosal bridge (magnification 20:1). Two sides covered predominantly with duodenal mucosa, one side with antral mucosa

In addition, two opening into the bulb were to be seen which were clearly separated from each other by a bridge of mucosa. Transpylorically, a duodenal ulcer could be seen on the lesser curvature. The patient died in 1972 in consequence of another illness after readmission to hospital.

At autopsy, the endoscopic findings were confirmed (Fig. 4). The musculature was lacking in the connective tissue core of the bridge but an almost luxuriant vascularity was observed in both ends.

Fig. 4. Case 3, 53-year-old man: mucosal bridge in the region of the pylorus. True-to-life plasticine model

Discussion

If we attempt to explain the development of these three pyloric bridges as described, there seem to be two possibilities. Firstly, on the basis of the long ulcer anamnesis and the fact that the ulcers varied in their spatial relationship to the pyloric canal, it would seem, in all three cases, that the changes must have been caused by inflammatory and mechanical factors which appeared incidentally only in advanced years. The anamnesis of the three patients at first do not always reveal the typical complaints of a gastric or duodenal ulcer. The course of the disease and the tendency to recurrence, proved to be uniform. Variable was the localization of the ulcers observed. The first case was a gastric ulcer located in the prepyloric region on the lesser curvature, the second patient suffered for 8 years from antral ulcers situated in the pyloric canal and which gave rise to a radiologically-recognizable scarred shrinking. Mr. S. J. on the other hand, suffered from a duodenal ulcer. Since the radiologically determined findings may be compared with a "snapshot" it would appear appropriate to emphasize the duration of the course of the disease. From this point of view, these mucosal bridges might be explained as the scarred residual condition of many years of suffering from ulcers.

On the other hand, we must consider the developmental aspects. Even though the rarity of these pyloric mucosal bridges seems to indicate that the mechanism of development is determined purely by chance, the pathological processes involved in this, still appeared to us to give rise to too many question marks. Tandler (1902), Kreuter (1905, 1913), Forssner (1913) and Anders (1925) have all been closely concerned with the development of the human intestinal tract. The basic studies of Max Ernst (1926) on the physiological cellular decay of

organs which do not succeed in reaching maturity are also closely associated. Summarizing, the following points might be mentioned as being adequately confirmed: Between the 30th and 60th day of the development in humans, an epithelial occlusion of the intestinal tract occurs. This occlusion is re-cannalized by vacuole formation and the subsequent growth of mesenchyma. Bremer (1944, 1957) explained this re-cannalization, in addition, in terms of a secretory activity of the endothelial cells which, via an intracellular formation of droplets and subsequent confluence effects a slow reformation of the lumen.

Whether this process also takes place in the segment which corresponds to the stomach, is variously assessed. The persistance of epithelial colonies and subsequent mesenchymal infiltration—discussed as possible aetiology in congenital small-bowel atresia—thus seems to be doubtful, at least for antral diaphragms. Either an epithelial occlusion, which occurs anyway, or a spreading of this to the region of the subsequent pylorus from the direction of the duodenum, with the persistence of an epithelial bridge is, in our opinion, the most probable explanation. A further possible explanation that might be considered is a later fusion of opposing walls in the pyloric canal a gap which, during embryogenesis remains almost capillary-fine for a considerable length of time (Anders, 1926).

Admittedly, further factors must also be involved, such as aberrant vessels, inflammation or local disturbances in nutrition, which can stimulate a fusion or at least support it.

If one considers the lack of muscles in the bridges described here, a certain parallelism with antral diaphragms becomes clear. The connective tissue bridges covered with mucosa might represent a diaphragm which has been reduced to this shape as a result of mechanical alterations. This point is now impossible to clarify. The genesis via embryonically-developed diverticula or perforations of wall-proximal cysts is, despite the unusual localization, perhaps, a possibility. It is not difficult to speak of a true duplication of the pylorus, in common with Christien *et al.* (1971), when, in the histological section of the band, all the wall layers of the pylorus can be recognized and the tunica muscularis clearly forms the core of the band. Since our own cases 2 and 3 do not comply with this condition, we have given preference to the purely descriptive designation, pyloric band.

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