



No More Publications for Acute Pancreatitis

Letter to the Editor

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The recent publication by Alsohaibani et al. [1] in your esteemed journal provided us with an incentive to express our concerns about the unexpected rise of publications related to new cases of acute pancreatitis (AP) after intragastric balloon (IGB) placement in obese individuals. More detailed, from 2008 to 2015, only six cases were reported, while from 2016 till today, there have been a further 20, including the 10 cases of the recent study from Saudi Arabia. Based on these, we are tempted to think that this exponential increase is rather a matter of fashion—“let’s publish our case”—rather than a real need to inform the medical community of a serious complication.

The abovementioned argument is reinforced by the fact that, from a total of 26 cases, the 16 come from Saudi Arabia, while the remaining 10 are from all over the world, including one from Brazil. How many balloons have been placed in Saudi Arabia to account for 16 cases of AP? And why are there no published cases, apart from one, from Brazil where at least 41,863 IGB had been placed by June 2016?

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Were there no other cases in Brazil? One can safely assume there were, especially since the related consensus about IGB guidelines and adverse effects explicitly and in detail states that “IGB removal is recommended by consensus in cases of moderate or severe pancreatitis; when pancreatitis is classified as mild, removal is not mandatory” [2]. There is, apparently, significant experience of cases of AP of varying severity, and one could also assume that the appearance of post-IGB acute pancreatitis is an accidental event, whose incidence cannot be predicted. Likewise, such cases have been reported even after a diagnostic colonoscopy [3].

Furthermore, since the Brazilian consensus is that “mild” AP be regarded as a separate entity that does not require “mandatory” balloon removal [2], it should be stated here that, by analyzing 24 out of 26 cases of AP due to balloon pressure on the pancreas, only 7 were conservatively treated and one of them was eventually removed at a second time. On the other hand, from the data given by all authors about the severity of AP in their cases, 16 were classified as mild and 3 as moderate; for the remaining 5 (out of the 10 cases of the last publication) no such information is given. However, these authors reported that of the 10 cases, only one had a Bedside Index of Severity Acute Pancreatitis (BISAP) score 2, in which the IGB was removed, while in the remaining cases the score was 0 [1].

In regard to the etiology of AP induction, 24 were attributed by the authors to the direct pressure of the IGB on the soft pancreatic tissue. The other 2 cases were related to a Spatz adjustable balloon, the catheter of which was dislodged and impacted into the duodenum; these 2 cases will not be further discussed because the etiology of pancreatitis is different. This explanation of direct pressure on the pancreatic body or tail sounds reasonable at first reading, but the IGB is typically placed in the gastric fundus, which has no direct anatomic relation to the pancreas. So, it is difficult to see how a balloon mounted in the fundus exerts pressure on the pancreatic tail.

It has been reported that in some cases, even at the time of insertion, the IGB does not remain in the fundus and slips to the antrum. In a series of 668 IGB, 509 were placed and

remained in the fundus, while the other 159 moved to the antrum at the time of the placement; 6 months later, at removal, 354 only were found in the fundus, while 314 were in the antrum, which means 155 IGB moved at some time-point within the 6 months period [4]. Therefore, a possible scenario to verify the concept of extrinsic pressure on the pancreas would be either initial placement or a subsequent migration of the IGB in the antrum. The former possibility may provide an explanation as to why 8/24 cases developed AP within the first 15 days after IGB placement; the latter may explain the remaining 16 cases. However, the broad time-frame (1 to 8 months) in these cases remains without obvious explanation. Taking into account the weight loss referred to in these publications, one may possibly associate the relatively early onset of AP (within 1–4 months) with the rapid weight loss (16 kg or 5 BMI units in 10 weeks) in these individuals: a rapid reduction in the visceral fat might reasonably mean a drop of the intra-abdominal pressure, thus making the IGB prone to migration into the antrum [4].

Concerning the theory of direct balloon pressure on the pancreas as the cause for AP, it seems reasonable. However, it could also be argued that it is not absolutely necessary for the IGB to have migrated, in order to directly compress the pancreas. The combination of increased intra-abdominal pressure due to obesity and the permanently dilated stomach due to the IGB lead to an increase in the pancreatic interstitial pressure and thus to a reduction of tissue microcirculation, meaning tissue hypo-perfusion, which in turn may result in the manifestation of AP [5]. There is, however, one further option: when a rigid latex capsule was placed over the normal pancreas (as the latex balloon in our case), the secretory stimulation of the pancreas resulted in a reduction in pancreatic blood flow [6]. Although there is no knowledge about hormonal stimulation of the pancreas in cases of an IGB being placed in the stomach, it is well documented that gastric/antral

dilatation increases pancreatic exocrine secretion by antro-pancreatic reflux [7]. These secretions may play an important role in permissive or contributory initiation of AP.

In conclusion, whatever the causative factor of pancreatitis induction in obese individuals treated with an IGB, it would appear, from all the above, that, without special reason, further publications on this subject are unnecessary.

Compliance with Ethical Standards

Conflict of Interest All authors declare that they have no conflicts of interest.

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