

#198 - BONE MINERAL DISEASE IN PATIENTS WITH CHRONIC ATROPHIC GASTRITIS: A CASE CONTROL STUDY

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Uribe J¹, Fuentes I¹, Martínez F¹, Silva F¹, Bustamante M¹, Lustig N¹, Maquilon S¹, Vargas J¹, Espino A¹, Riquelme A¹, Shah S², Florenzano P¹, Latorre G¹

¹Pontificia Universidad Católica de Chile, Santiago, Chile ²University of California, San Diego, California, Estados Unidos (EEUU)

Background: Chronic atrophic gastritis (CAG) is a progressive inflammatory condition of the gastric mucosa, caused by *Helicobacter pylori* (*Hp*) infection and autoimmune gastritis (AIG), which is characterized by atrophy due to the loss of gastric glandular cells. Parietal cell atrophy leads to hypochlorhydria or achlorhydria. Through this mechanism CAG may lead to impaired absorption of calcium and vitamin-D, potentially leading to decrease bone density (DBD).

Aim: Investigate and compare the frequency of DBD in patients with CAG (cases) of any origin, contrasted with healthy controls without CAG (Operative Link for Gastritis-Assessment (OLGA) stage 0).

Methods: Case-control study to compare DBD in patients with CAG (cases) to patients with OLGA 0 (controls). Cases included either *Hp*-related CAG or AIG. Adult patients with availability of dual-energy X-ray absorptiometry (DEXA) scans and esophagogastroduodenoscopy with gastric biopsies by Sydney protocol were included. The primary outcome was DBD as defined by osteopenia (T-score -2.5 to -1) and osteoporosis (T-score ≤ -2.5). We performed logistic regression adjusted for age and sex to quantify the association between DBD.

Results: 139 patients were included. The CAG group comprised 74 patients (97% female; mean age 66 years-old), 44 of whom (59%) had *Hp* infection and 41% (n=30) AIG. Sixty-five patients were OLGA 0 (control group) (95% female; mean age 66 years-old). Mineral density, T-score and Z-score by group are described in Table 1. Logistic-regression revealed that DBD was more commonly in patients over 50 years-old with CAG contrasted to OLGA 0 patients (Odds-Ratio 2.3; 95%CI: 1.02-4.9). No significant differences were observed among *Hp* related CAG and AIG.

Conclusion: DBD was more common in patients with CAG of any origin, particularly in patients over 50 years-old. These results emphasize the importance of early screening and management of bone health in patients with CAG to mitigate potential complications associated with impaired bone mass.

Table 1. Clinical characteristics and bone mineral disease among patients over 50 years with and without chronic atrophic gastritis.

	Cases		Controls
	<i>Hp</i> related CAG n = 44	AIG n = 30	OLGA 0 n = 65
Age, mean (95%CI)	69 (67-71)	66 (63-69)	66 (64-68)
Female, n (%)	42 (95)	30 (100)	62 (95)
OLGA			
OLGA 0	0 (0)	0 (0)	65 (100)
OLGA I-II	17 (39)	29 (97)	0 (0)
OLGA III-IV	27 (61)	1 (3)	0 (0)
Osteopenia, n (%)	28 (64)	20 (67)	32 (49)
Osteoporosis, n (%)	9 (20)	4 (13)	12 (18)
Decrease bone density *, n (%)	37 (84)	24 (80)	44 (67)
DEXA, median (IQR)			
Lumbar mineral density	1.012 (0.917-1.113)	1.061 (0.946-1.179)	1.049 (0.949-1.127)
Lumbar T score	-1.45 (-2.25 to -0.65)	-1.05 (-1.9 to 0)	-1.1 (-2 to -0.6)
Lumbar Z score	-0.2 (-0.85 to 0.6)	0.4 (-0.5 to 0.9)	-0.3 (-0.9 to 0.6)
Hip mineral density	0.831 (0.761-0.873)	0.849 (0.803-0.949)	0.877 (0.801-0.932)
Hip T score	-1.5 (-1.95 to -1.2)	-1.25 (-1.7 to -0.6)	-1.1 (-1.7 to -0.8)
Hip Z score	-0.15 (-0.6 to 0.3)	0.15 (-0.4 to 0.5)	0 (-0.4 to 0.5)

*Primary outcome defined as the combination of osteopenia or osteoporosis; *Hp*: *Helicobacter pylori*; CAG: Chronic atrophic gastritis; AIG: Autoimmune gastritis; OLGA: Operative Link for Gastritis Assessment; CI: Confidence interval; DEXA: dual-energy X-ray absorptiometry; IQR: Interquartile range.