

Rapid Rx



Next Time...

Look for an update about Januvia (a new agent for Type 2 DM) !

Keep those questions coming...



Good Luck Cheryl!

Cheryl Walters, our PharmD student (U of T), has wrapped up her rotation with us. Thanks for all of your questions and consults. Our **next PharmD student** starts at **Stonechurch on March 24th**. **Why wait? Send your consults/questions today!**©

UPDATE: What's the deal with Duralith®?

As you'll recall, we mentioned a long-acting alternative that was supposed to be available by the end of 2007. **Oops!**...unfortunately, it is still unavailable so you'll need to **change patients** to the **regular release** lithium preparations by dividing the Duralith® dose into BID-TID without changing your total daily dose. A serum lithium level one week after the switch isn't a bad idea...remember, 12 hours post-dose for these ones. Your pharmacists would be happy to help discuss with patients – just sent us a consult! ☺ (LM)

Why are patients calling about their bisphosphonates?

A recent Canadian study has people talking about the role of oral bisphosphonates (BPs) in bone necrosis again. The nested case-control study found that patients with severe bone necrosis were 3 times more likely to have a previous or current history of BP therapy. However, the incidence of bone necrosis was still relatively rare - the overall incidence of 1:20,000 increased to 3:20,000 (J Rheum 15 Jan 2008).

Remember that this was an observational database study looking at all types of bone necrosis and did not establish causality. There were also other methodological limitations (which Cheryl has summarized in a nice 2-pager available under e-docs).

The concern has to be weighed against the morbidity and mortality associated with fragility fractures. Nevertheless, since BPs are widely used (estimated 190 million prescriptions worldwide) it is important for that we be aware of any bone pain experienced by patients receiving bisphosphonate treatment.

You can tell patients that...Bone necrosis is a rare disease and treatment with BPs may slightly increase the risk of developing this side effect. In most cases, the morbidity and mortality from a hip fracture far outweigh the low risk of developing bone necrosis on BP. (JJV/CW)

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A 2nd page for our Feb/Mar issue (with a new color scheme ☺)!

So talk to me...how does achlorhydria impact medication absorption?

Thanks to Liz Shaw for the question and Cheryl Walters for putting together the answer...

Achlorhydria results when gastric parietal cells do not function or are inhibited. Potential consequences of achlorhydria include gastric cancer, hip fracture, and bacterial overgrowth in the GI tract potentially leading to nutrient deficiency. Risk factors are increasing age and autoimmune conditions.

There are a number of conditions that cause or contribute to achlorhydria:

Cause/Condition	Mechanism	Potential impact on drug absorption from stomach
Medication: Proton pump inhibitors (PPI)	Direct inhibition of H ⁺ /K ⁺ ATPase (proton pump) → inhibits secretion of hydrochloric acid (HCl) in stomach	↓ absorption of medications soluble at acidic pH
Bariatric surgery	↓ in gastric surface area + ↓ HCl production	↓ time for drug absorption in stomach + drug bioavailability
Autosomal recessive lysosomal storage disease	Parietal cells only partially active → ↓ secretion of stomach HCl	↓ absorption of medications soluble at acidic pH

Little info is available re: specific recommendations for optimizing medication absorption in patients with achlorhydria. Some **general principles** can be drawn from bariatric surgery literature:

- **Enteric coated preps** are absorbed in the intestines → no concerns about alteration of absorption in achlorhydria/post bariatric surgery
- **Timing of meds:** For patients using PPIs, suggest taking medications requiring acidic pH with food → may have some acid secretion around meal times
- Assess whether **interference** with absorption is **well documented or theoretical**
 - If ↓ absorption is well documented: Is there evidence describing the clinical impact/outcomes related to ↓ absorption?

Example: Calcium (Ca) absorption in patients on PPI therapy

A retrospective, case control study of PPI users vs non-acid suppression users examined the risk of incident hip fracture (JAMA 2006; 296:2947-53). Long term, high dose PPI therapy was associated with a small increased risk of hip fracture, hypothesized to be related to a decrease in insoluble calcium absorption and possibly inhibition of the osteoclast H⁺/K⁺ ATPase pump (resulting in decreased bone resorption).

What does this mean for PPI users?

If patients consume the majority of their recommended Ca intake from dietary sources (soluble Ca), then it is unlikely that PPI therapy places them at increased risk of hip fracture. However, patients receiving most or all of the recommended Ca from supplements, intervention may be required, depending on the calcium supplement formulation.

The carbonate salt needs an acidic environment to dissolve and subsequently be absorbed.

Options to optimize Ca intake/absorption are: 1) take it with meals to maximize presence of gastric acid, 2) take it with acidic food/drink OR **BEST OPTION:** 3) switch to calcium citrate because it does not need an acidic environment for absorption. One study in achlorhydric patients showed significantly higher bioavailability with citrate vs carbonate (45 % vs 4 % respectively).

The drawback though is that Ca citrate supplements often require more frequent dosing/number of pills per day to achieve the recommended daily Ca intake.

These options should be presented to patients receiving the majority of Ca intake in supplement form to determine which is best for the individual.

For a table of other medications that may be impacted...check out edocs!

