

PharmaCE™

a continuing education program for *JPT* readers

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ACCREDITATION

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LANTHANUM CARBONATE

(see page 99)

Goal

To provide a brief overview of the pathophysiology and consequences of chronic hyperphosphatemia and review of the efficacy and safety data for lanthanum carbonate in reducing phosphate levels in patients with chronic kidney disease.

Objectives

After reading this article, the reader should be able to:

1. list the currently available phosphate binders that treat hyperphosphatemia secondary to chronic renal disease;
2. define chronic renal disease;
3. identify laboratory parameters that define successful treatment of patients with chronic kidney disease;
4. discuss the advantages and disadvantages of lanthanum carbonate compared with other phosphate binders in the treatment of hyperphosphatemia related to chronic kidney disease.

Test Questions

1. According to the clinical practice guidelines established by the National Kidney Foundation's Kidney Disease Outcomes Quality Initiative, the definition of chronic kidney disease is:
 - (a) GFR <70 mL/min/1.73 m² for 3 months or longer.
 - (b) GFR <60 mL/min/1.73 m² for 3 months or longer.
 - (c) GFR <50 mL/min/1.73 m² for 3 months or longer.
 - (d) GFR <40 mL/min/1.73 m² for 3 months or longer.
 - (e) GFR <40 mL/min/1.73 m² for 6 months or longer.
2. According to the clinical practice guidelines established by the National Kidney Foundation's Kidney Disease Outcomes Quality Initiative, which of the following laboratory parameters meet(s) criteria for successful treatment of a patient with chronic kidney disease?

- (a) serum phosphate level 5.5–6.2 mg/dL
- (b) serum calcium–phosphate product >60 mg²/dL²
- (c) corrected serum calcium level 9.5–11.4 mg/dL
- (d) serum intact parathyroid hormone level 150–300 pg/mL
- (e) Both a and b are correct.

3. Which of the following regarding lanthanum carbonate is *true*?
 - (a) It must be taken with a full 8 ounce glass of water (no other beverage).
 - (b) Clinical studies have shown that it has more potent phosphate-lowering effect compared with sevelamer.
 - (c) It is chewable.
 - (d) It forms lanthanum acetate in the gut.
 - (e) It is a bivalent cation.
4. All of the following are phosphate binders used in the treatment of chronic hyperphosphatemia associated with kidney disease *except*:
 - (a) aluminum hydroxide.
 - (b) calcium carbonate.
 - (c) cinacalcet hydrochloride.
 - (d) lanthanum carbonate.
 - (e) sevelamer hydrochloride.
5. Which of the following phosphate binders is non-aluminum, non-calcium?
 - (a) Renagel
 - (b) Tums
 - (c) Fosrenol
 - (d) Amphojel
 - (e) Both a and c are correct.
6. Which of the following regarding clinical studies of lanthanum carbonate for hyperphosphatemia is *true*?
 - (a) Treatment periods ranged from 2 to 10 years.
 - (b) There were low numbers of patient withdrawals.
 - (c) Only open-label studies are available.
 - (d) The most common adverse effects were gastrointestinal.
 - (e) None of the above is true.
7. Which of the following regarding clinical studies of lanthanum carbonate for hyperphosphatemia is *true*?
 - (a) In some studies, doses of comparator standard therapies were not specified.
 - (b) Some studies were underpowered to determine significant treatment differences.
 - (c) Lanthanum carbonate was more effective than placebo in lowering serum phosphate to target levels.
 - (d) Lanthanum carbonate is as or less effective than standard therapies in lowering serum phosphate to target levels.
 - (e) All of the above are true.
8. Which of the following regarding bone disease related to lanthanum carbonate therapy is *true*?
 - (a) Lanthanum carbonate causes less adynamic bone disease compared with calcium acetate.
 - (b) Lanthanum carbonate causes more adynamic bone disease compared with calcium acetate.

Answer sheet appears on facing page.

LAF 237 FOR TYPE 2 DIABETES

(see page 105)

Goal

To review the role of incretin therapies, specifically LAF 237 (vildagliptin), as new treatment options for patients with diabetes mellitus, with an emphasis on the clinical benefits the incretin effect can provide in treatment, such as improved glycemic control and prevention of associated complications.

Objectives

After reviewing this article, the reader should be able to:

1. discuss the role of incretin hormones, specifically GLP-1, in the pathogenesis of type 2 diabetes mellitus;
2. identify the role of DPP-4 inhibitors in the enhancement of GLP-1;
3. discuss the impact of DPP-4 inhibitors on glycemic control.

Test Questions

1. Which of the following contributes to hyperglycemia?
 - (a) increased hepatic glucose production
 - (b) β -cell secretory defects
 - (c) increased lipolysis
 - (d) peripheral insulin resistance
 - (e) All of the above are correct.
2. GLP-1 is released from:
 - (a) β -cells in the intestinal mucosa.
 - (b) β -cells in the pancreas.
 - (c) L-cells in the intestinal mucosa.
 - (d) L-cells in the pancreas.
 - (e) None of the above is correct.
3. Actions of GLP-1 include all of the following *except*:
 - (a) increase in gastric emptying.
 - (b) increase in satiety.
 - (c) regulation of insulin.
 - (d) regulation of glucagon.
 - (e) regulation of islet cell growth.
4. Vildagliptin delays the degradation of GLP-1, resulting in:
 - (a) decreased appetite.
 - (b) decreased postprandial glucose levels.
 - (c) decreased GLP-1 levels.
 - (d) Both a and b are correct.
 - (e) All of the above are correct.
5. The half-life of vildagliptin's inhibitory effect is estimated to be:
 - (a) 30 minutes.
 - (b) 65 minutes.
 - (c) 90 minutes.
 - (d) 130 minutes.
 - (e) 175 minutes.
6. Monotherapy trials with vildagliptin demonstrated all of the following *except*:
 - (a) decrease in glucose levels.
 - (b) decrease in glucagon levels.
 - (c) decrease in body weight.

- (c) Lanthanum carbonate causes more adynamic bone disease compared with placebo.
 - (d) Lanthanum carbonate causes similar rates of adynamic bone disease compared with placebo.
 - (e) None of the above is true.
9. The recommended initial dosing and administration instructions for lanthanum carbonate is:
 - (a) 750–1,500 mg/day, in divided doses, swallowed whole with meals.
 - (b) 750–1,500 mg/day, in divided doses, chewed with meals.
 - (c) 750–1,500 mg/day, in divided doses, swallowed whole without regard to meals.
 - (d) 750–1,500 mg/day, in divided doses, chewed without regard to meals.
 - (e) All of the above are reasonable regimens.
 10. Which of the following regarding drug interactions with lanthanum carbonate is *true*?
 - (a) Drugs known to interact with antacids should not be administered within 2 hours of lanthanum carbonate.
 - (b) Lanthanum carbonate may interfere with warfarin pharmacokinetics.
 - (c) No drug–drug interaction studies have been performed with lanthanum carbonate.
 - (d) Lanthanum carbonate is a substrate of the cytochrome P450 enzyme system.
 - (e) Both c and d are correct.
 11. How often can the dose of lanthanum carbonate be titrated to achieve target serum phosphate levels?
 - (a) every 2–3 days
 - (b) every 4–7 days
 - (c) every week
 - (d) every 2–3 weeks
 - (e) every month
 12. Which of the following are pathophysiologic changes that occur with chronic hyperphosphatemia in patients with kidney disease?
 - (a) lowered serum levels of ionized calcium
 - (b) elevated PTH levels
 - (c) diminished renal 1α -hydroxylase activity
 - (d) reduced vitamin D production
 - (e) All of the above are true.
 13. Which of the following regarding lanthanum carbonate and calcium carbonate is *true*?
 - (a) Both are approved for treatment of hyperphosphatemia secondary to chronic renal disease.
 - (b) Lanthanum carbonate has a lower risk of hypercalcemia compared with calcium carbonate.
 - (c) Lanthanum carbonate may have a higher risk of bone disease after long-term use compared with calcium carbonate.
 - (d) Lanthanum carbonate has a lower risk of gastrointestinal adverse effects compared with calcium carbonate.
 - (e) Both b and c are true.
 14. According to the author, what patient population might benefit most from lanthanum carbonate therapy?
 - (a) patients who are fluid restricted
 - (b) patients who can tolerate other phosphate binders
 - (c) patients who like cherry-flavored tablets
 - (d) patients who do not wish to take a phosphate binder
 - (e) patients with acute renal failure

- (d) increase in GLP-1 levels.
 - (e) None of the above is correct.
7. Normal glucagon ranges are:
- (a) 0–25 pg/mL.
 - (b) 25–50 pg/mL.
 - (c) 25–75 pg/mL.
 - (d) 50–75 pg/mL.
 - (e) 50–100 pg/mL.
8. Adverse effects reported with vildagliptin monotherapy included:
- (a) nasopharyngitis.
 - (b) headache.
 - (c) hypoglycemia.
 - (d) Both a and b are correct.
 - (e) All of the above are correct.
9. Current published combination therapy trials include vildagliptin and:
- (a) α glucosidase inhibitors.
 - (b) biguanides.
 - (c) insulin.
 - (d) sulfonylureas.
 - (e) thiazolidinediones.
10. Studies have shown that vildagliptin:
- (a) prolongs endogenous GLP-1 activity.
 - (b) improves postprandial glucose levels.
 - (c) reduces A1C levels.
 - (d) All of the above are correct.
 - (e) Both a and b are correct.
11. In current published trials, vildagliptin has not been studied in:
- (a) patients under 25 years of age.
 - (b) patients with type 1 diabetes.
 - (c) patients with hyperlipidemia.
 - (d) All of the above are correct.
 - (e) Both a and b are correct.
12. Current data support the administration of vildagliptin as:
- (a) once daily prior to breakfast.
 - (b) once daily after breakfast.
 - (c) once daily prior to supper.
 - (d) once daily after supper.
 - (e) once daily prior to bedtime.
13. Advantages of vildagliptin include:
- (a) route of administration.
 - (b) dosing regimen.
 - (c) low risk of weight gain.
 - (d) low risk of hypoglycemia.
 - (e) All of the above are correct.