Hyperphosphatemia, a nearly universal complication of kidney failure, is accompanied by hypocalcemia and low serum levels of vitamin D. Without treatment, these deficiencies usually lead to severe secondary hyperparathyroidism, which in turn leads to painful fractures, brown tumors, and generalized osteopenia. Dietary restriction of phosphate has long been the cornerstone of therapy, but this measure is usually not sufficient to control hyperphosphatemia. As a result, oral phosphate binders are used in over 90% of patients with kidney failure, at an annual cost of approximately $750 million (in U.S. dollars) worldwide.1

Historically, treatment with oral phosphate binders was intended to prevent symptomatic secondary hyperparathyroidism. More recently, achieving tighter control of markers associated with abnormal mineral metabolism (e.g., serum phosphate, calcium, and parathyroid hormone levels) has become a specific therapeutic objective.2 This therapeutic shift has been driven by several factors: observational data that link disordered mineral metabolism with adverse clinical outcomes; concern about vascular calcification, which is also associated with adverse outcomes and may correlate with exposure to calcium-based phosphate-binding agents; and, perhaps, the availability of new therapeutic agents.3

In this article we review the rationale for treatment with oral phosphate binders, discuss evidence that supports the use of available agents, and suggest an approach for clinical practice.