In 1985, Hoekman et al. (44) reported PMD treatment for a boy with juvenile osteoporosis. The first evaluation showed that phosphatase levels and mature diaphyseal patient's spine and radius BMD increased during PMD at an accelerated rate between ages 7 and 12 years compared to baseline. Additionally, biochemical findings showed a decrease in bone turnover, indicating that PMD exposure was associated with decreased need for cortical thickness and trabecular bone mass for protection against fractures.

In 2006, Land et al. reported partial reconstitution of vertebral shaping (59) during 2-4 years of PMD. While his bone ALP remained elevated, serum osteocalcin was slightly decreased, suggesting that his bone remodeling was maximally suppressed during PMD exposure 1-1/2 years before biopsy. Referral for reconstitution led to low bone turnover, but the patient's bone density was lower compared to baseline. Of interest, serum BB-CK and TRAP levels were not detectable biochemically. This suggests that perhaps when his bone remodeling was maximally suppressed during PMD exposure, low bone turnover may not have been detectable biochemically.

In 2007, Waterhouse et al. (40) examined BP cessation for an average of 26 months in 17 patients. Notably, however, Ward et al. (41) in 2007 reported in a young child with OI marked decrease in BMD, which persisted even after discontinuing bisphosphonates. In our patient, bone fragility seems largely from BP-induced deceleration of bone growth.

Our patient had been referred in 2002 at 12 years-of-age, 1-½ years after cessation of escalating intravenous doses of pamidronate (PMD)-induced osteopetrosis (OPT). More than 20 years earlier, rickets and osteomalacia were described, but the patient's bone density and bone turnover were normal at baseline. Assessment of both cortical bone thickness and trabecular bone porosity in such metaphyses requires investigation. It may be that this complication of BP toxicity during childhood is unique to our patient. From the age of 12 years-of-age, his bone ALP remained elevated, and his serum osteocalcin was slightly decreased, suggesting that his bone remodeling was maximally suppressed during PMD exposure 1-1/2 years before biopsy. Referral for reconstitution led to low bone turnover, but the patient's bone density was lower compared to baseline. Of interest, serum BB-CK and TRAP levels were not detectable biochemically. This suggests that perhaps when his bone remodeling was maximally suppressed during PMD exposure, low bone turnover may not have been detectable biochemically.