Osteopathology can include conditions that impair the normal functioning of bone and bone marrow. For example, age-related declines in bone mass often coincide with increased fracture risk, reduced hematopoiesis, augmented bone marrow ischemia and adiposity, and immnosenescence. Even though normally ascribed to dysfunction of bone and bone marrow, osteopathology may derive a portion of its etiology from dysfunction of the bone vascular system. Declines in bone volume are associated with 1) diminished vasodilator capacity of bone arteries, 2) reduced bone blood flow, 3) impaired bone angiogenesis and blood vessel density, and 4) an increased distance between bone marrow blood vessels and bone surfaces. Thus, bone vascular dysfunction may rest internal to the skeleton (i.e., the bone marrow blood vessels) or external (i.e., nutrient arteries and veins that originate outside and penetrate the skeleton). We recently discovered severe calcification of bone marrow blood vessels, whereby this pathology extended beyond calcium deposition. In fact, the bone marrow blood vessels appeared ossified and bone-like in morphology, as evidenced by the presence of osteocyte lacunae on their abluminal surfaces. The ossification of bone marrow blood vessels progressively worsened with advancing age in rats and was associated with reduced bone volume, augmented bone marrow adiposity and a reduced number of patent bone marrow blood vessels. Additionally, there may be a link between ossification and an increased inflammatory bone marrow microenvironment. This presentation will present quantitative and characteristic data on bone marrow blood vessel ossification as a function of advancing age in male Fischer-344 rats. Additionally, the influence of bone marrow, via enhanced inflammatory cytokine production, on bone vascular function will be highlighted. Lastly, data will bear out that ossification of bone marrow blood vessels translate to the human model.