X-Ray Diagnosis of Skeletal Fluorosis

In 1937, Kaj Roholm published his seminal study *Fluorine Intoxication* in which he described three phases of bone changes that occur in skeletal fluorosis. (See below). These three phases, which are detectable by x-ray, have been widely used as a diagnostic guide for detecting the disease. They describe an osteosclerotic bone disease that develops first in the axial skeleton (the spine, pelvis, and ribs), and ultimately results in extensive calcification of ligaments and cartilage, as well as bony outgrowths such as osteophytes and exostoses.

Subsequent research has found, however, that x-rays provide a very crude measure for diagnosing fluorosis since the disease can cause symptoms and effects (e.g., osteoarthritis) before, and in the absence of, radiologicaly detectable osteosclerosis in the spine. Moreover, German scientists have expanded on Roholm's description of the x-ray picture of the disease, by defining two additional phases ("subtle signs" & "stage O-I") that occur prior to Roholm's 3 phases. (See below). Research has found that individuals with these early skeletal phases can experience significant joint pain and stiffness. According to Franke (1975): "[W]e found patients with slight radiological changes (subtle signs or O-I) who complained of intense pains in the spine and in the large joints. On the other hand, some patients whose fluorosis was radiologically distinct were almost without complaints."

Making matters even more complicated is the fact that numerous studies have found that fluorosis can cause a diverse range of skeletal changes beyond the predominantly osteosclerotic form of the disease that Roholm described. Strict reliance on Roholm's description of classic fluorosis will thus frustrate efforts to detect, and prevent, skeletal fluoride poisoning.

Roholm (1937): The 3 Phases of Bone Changes in Skeletal fluorosis (Detectable by X-Ray):

"From the X-ray picture it is possible to differentiate between three phases of the same osteosclerotic process, each overlapping the next without any sharp boundary.

1st Phase.
"The changes are observed in pelvis and columna, but are doubtful or absent elsewhere. The density of bone is very little increased. The trabeculae are rough, blurred and give deep shadows; this is often distinct in corpora of the lower lumbar vertebrae. The bone has both a more prominent and a more blurred structure at the same time, which is very characteristic when the operator is familiar with the phenomenon, but otherwise is easily overlooked. The bone contour is sharp. In some few cases there is incipient osteophyte formation on the edge of corpora of the lumbar vertebrae. The boundary against the normal bone structure is not sharp, and in an isolated case it will be difficult to decide whether the change is a normal variation or a pathological finding. In serial examinations, however, the difference is distinct.

2nd Phase.

"The bone structure is blurred, the trabeculae merging together. Over often rather large areas the bone gives a diffuse, structureless shadow. At first glance the negative seems to have

been underexposed, but it is difficult or impossible to distinguish details even when the time of exposure or the tension is increased. The bone contours are uneven and somewhat blurred. The changes are most distinct in pelvis and columna, but also in the ribs and in the bones of the extremities, even if there they are less pronounced and often resemble the changes described as 1st phase. In the extremity bones the medullary cavity is usually moderately narrowed. In columna there are incipient or moderate ligament calcifications, especially caudally; they appear in the form of pointed, beaked osteophytes with an inclination to form bridges between vertebral bodies or as a diffuse blur lying posteriorly to corpora. In some cases (particularly among the younger individuals) the ligament calcifications are absent, though the bone structure is so changed that the case must be placed to the 2nd phase.

3rd Phase. On the negative the bone presents itself as a more or less diffuse marble-white shadow, in which the details cannot be distinguished. Changes are observable in all bones but are still greatest centrally, being most conspicuous in bones with cancellous structure, pelvis, columna, ribs and sternum. In the bones of the extremities there are changes in the structure that recall the 2nd phase, or fairly often only the 1st phsase. Among the worst affected individuals changes are to be seen in the cranium, usually rather moderate in intensity. Theca is denser and gives a deeper shadow than normally, sutures and vessel grooves are indistinct, and the same applies to impressiones digitatae. The air-sinuses in the cranial bones are diminished in size. The region around sella turcica gives a deep shadow but is normal as to contour. No distinct thickening of the processus clinoidei was observed.

The bone contours almost everywhere are wooly and blurred. Very often the bones or certain parts of them have a rough and slightly enlarged appearance, but otherwise the shape is not altered. On the extremity bones are irregular periosteal thicknesses, some flats, others more rough. The interosseous membrane in antibrachium and crus are calcified to a greater or smaller extent. The normal cristae corresponding to the muscle attachments are increased in size and resemble exostoses. On costae, especially vertebrally, there is calcification of the insertions of the intercostal muscles, which appear like “rime frost needles” or irregular shadows to both sides. There are considerable ligament calcifications, varying up to very severe, in columna, particularly in pars lumbalis and thoracalis. In columna cervicalis these changes are less pronounced, but distinct. The ligament calcifications appear partly in the form of bridge-like connections with fairly sharp borders between corpora, partly as a diffuse opacity and density round about the intervertebral and costovertebral articulations. Processus transversi and spinosi are rough and thickened; between the latter are considerable ligament calcifications with irregular borders. In the pelvis, ligamentum sacrotuberosum is sometimes calcified. The intensity of the calcification and the diffuse density of the bone usually are in conformity with each other; in some elderly workers, however, there is a density of the osseous tissue which does not attain to the extreme degree, side by side with very pronounced ligament calcification.

In the extremity bones, both short and long, the medullary cavity is diminished in width and the boundary against compacta is less sharp than normally. The width of compacta is correspondingly increased. In tibia and femur the width of the medullary cavity sometimes decreases to half the normal, in metacarpals and phalanges there is sometimes a partial occlusion of the cavity.

The interarticular spaces are of normal width everywhere and the contours are sharp. Limited calcifications of the capsule in hip and knee joints are seen. The intervertebral disks are not visibly changed and the calcification of the costal cartilage does not exceed the normal.

If the result of the Rontgen examination is to be summarized, the first thing to emphasize is the fact that the affection is a system-disease, for it attacks all bones, though it has a predilection for certain places. The pathological process may be characterized as a diffuse osteosclerosis, in which the pathological formation of bone starts both in periosteum and in endosteum. Compacta densifies and thickens; the spongiosa trabecula thicken and fuse together. The medullary cavity decreases in diameter. There is a considerable new-formation of bone from periosteum, and ligaments that normally do not calcify or only in advanced age undergo a considerable degree of calcification. All signs of bone destruction are absent from the picture."
SOURCE: Roholm K. (1937). Fluoride intoxication: a clinical-hygienic study with a review of the literature and some experimental investigations. London: H.K. Lewis Ltd. pp. 141-143

Fritz (1958): Two additional, Subtle Phases of fluorosis

"In addition to the well-known radiological stage classification, stage I to III according to Roholm, two prestages according to Fritz have proved to be important in our investigations: the so-called (subtle signs) and the stage O-I. Concerning the subtle signs… a condensation of the bone-structure and an enlargement of the bone trabeculae in the lumbar spine are evident. In addition, there are accompanying shadows along the tibia, fibula, radius, and ulna. At the stage O-I the structure of the thoracic spine has already increased in density, whereas in the lumbar region the normal structure of the bone begins to disappear. The periosteal apposition of new bone at the bones of the forearm and lower legs are more distinct. For better proof of the periosteal appositions on these bones, we used slightly underexposed similar to the kind of radiography employed for soft parts."
SOURCE: Franke J, et al. (1975). Industrial fluorosis. Fluoride 8: 61-83.

Diagnosing Skeletal Fluorosis: US Public Health Service Chart

US Public Health Service 1991	
OSTEOSCLEROTIC PHASE	**ASH CONCENTRATION (mg/kg)**
Normal Bone	**500 – 1,000**
Preclinical Phase asymptomatic; slight radiographically-detectable increases in bone mass	**3,500 – 5,500**
Clinical Phase I sporadic pain; stiffness of joints; osteosclerosis of pelvis & vertebral column	**6,000 – 7,000**
Clinical Phase II chronic joint pain; arthritic symptoms; slight calcification of ligaments; increased osteosclerosis/cancellous bones; with/without osteoporosis of long bones	**7,500 – 9,000**

Phase III: Crippling Fluorosis limitation of joint movement; calcification of ligaments/neck, vert. column; crippling deformities/spine & major joints; muscle wasting; neurological defects/compression of spinal cord	**> 8,400**

The Problem with Using Bone Fluoride Content as a Diagnostic Tool:

The U.S. Public Health Service's diagnostic guide for skeletal fluorosis is incorrect on two key premises. First, it states that the pre-clinical phase of fluorosis is "asymptomatic." Numerous published studies show that this is not so. Second, it relies on the fiction that there are clear, firm bone fluoride levels that do, or do not, cause the disease. Again, however, studies have repeatedly found that people with skeletal fluorosis can have levels far below this range. Indeed, individuals with crippling skeletal fluorosis in the United States have been found to have as little as 1,900 ppm in their bone — a concentration that, according to the PHS, should not even produce the earliest symptoms of the disease. (Bruns & Tyle 1988).

Bone Fluoride Levels in People with Skeletal Fluorosis vs. the *Alleged* Threshold Concentration

Study	Degree of Fluorosis	Bone F	% of Alleged Threshold (DHHS 1991)
Bruns (1988)	Stage III	1,900 ppm	23% (8,400 ppm)
Sandberg (1985)	Stage I	2,640 ppm	44% (6,000 ppm)
Wepp-Peploe (1966)	Stage III	2,530 ppm	30% (8,400 ppm)
Morris (1965)	Stage I	2,040 ppm	34% (6,000 ppm)
Singh (1961)	Stage III	600 ppm	7% (8,400 ppm)

Sources:

- Department of Health and Human Services (1991). Review of fluoride benefits and risks.
- Bruns BR, Tytle T. (1988). Skeletal fluorosis: a report of two cases. *Orthopedics* 11: 1083-1087.
- Sandberg D, Zichner L. (1985). A case of bone fluorosis of undetermined origin. *Arch Orthop Trauma Surg.* 104:191-95.
- Webb-Peploe MM, Bradley WG. (1966). Endemic fluorosis with neurological complications in a Hampshire man. *Journal of Neurology, Neurosurgery and Psychiatry* 29:577-583.
- Morris JW. (1965). Skeletal fluorosis among indians of the American Southwest. *American Journal of Roentgenology, Radium Therapy & Nuclear Medicine* 94: 608-615.
- Singh A, et al. (1961). Skeletal fluorosis and its neurological complications. *Lancet* 1: 197-200.

“Available data suggest that there is wide variability in individual tolerance to toxic effects of skeletal accumulation of fluoride.” “Fluoride concentrations of 200 to 6500 ppm have been reported in bones which were ‘normal’… But bones from patients with severe chronic fluorosis have been found to contain 700-7000 ppm, 905-13,580 ppm, 1120-6050 ppm, and 2040-11,500 ppm. This overlap with the ‘normal’ range is indicative of wide variability in individual sensitivity to harm.”
SOURCE: Groth, E. (1973), Two Issues of Science and Public Policy: Air Pollution Control in the San Francisco Bay Area, and Fluoridation of Community Water Supplies. Ph.D. Dissertation, Department of Biological Sciences, Stanford University, May 1973.

“Singh et al (1961) described skeletal fluorosis in individuals with F levels in bones in the 700 to 1600 ppm range. This F level in bones is far below that at which many claim fluorosis cannot occur. Data published by Call demonstrated that F content of bones does not parallel the F content in soft tissue organs. Therefore the presence or absence of ill-effect due to fluoride cannot be established on the basis of the F content of bones.”
SOURCE: Waldbott GL. (1968). Hydrofluorosis in the U.S.A. Fluoride 1: 94-102.

“From tabulations of the present study, it is apparent that the degree of bone change does not correlate well with the amount of fluoride present in the bone.”
SOURCE: Morris JW. (1965). Skeletal fluorosis among Indians of the American Southwest. American Journal of Roentgenology, Radium Therapy & Nuclear Medicine 94: 608-615.

“The fluoride content of bone does not appear to be the only factor contributing to the onset of fluorosis. Other metabolic factors must be considered. Concentrations of fluoride from 0.50% to 0.75% (dry, fat-free basis) have been found in ribs, sternum, and vertebrae during postmortem studies of persons with endemic fluorosis, whereas McClure et al reported fluoride values as high as 0.51% to 0.65% in various regions of the skeleton with no associated malfunction or microscopically detectable bone changes.”
SOURCE: Marier JR, et al. (1963). Accumulation of skeletal fluoride and its implications. Archives of Environmental Health 6: 664-671.