Osteonecrosis of Hip

- Hip, groin pain; decreased range of motion
- 3rd-6th decades most common
- M:F = 4:1

Etiology

- Posttraumatic: Disrupted blood supply
  - Hip dislocation: If not reduced within 12 hours, 50% develop ON
  - Tear small vessels, compress larger vessels; ischemia initially reversible, but if prolonged, spasm and thrombosis lead to necrosis
  - Subcapital fracture: 30% of displaced femoral fractures develop ON
- Corticosteroid use: Enlargement of intramedullary fat cells and ↑ marrow pressure inhibits blood flow
  - Of all patients on steroids, 2% develop ON
  - Risk greater with short duration (6 weeks) and high doses (> 20 mg); 5-25% of patients develop ON
  - Risk ↑ in renal transplant patients on steroids, with osteodystrophy (40% develop ON)
  - 10% of long-term survivors of bone marrow transplantation who received high doses of steroids develop ON

Etiology (other)

- Sickle cell anemia: Sickled cells thrombose in microvasculature at low oxygen tension
- Gaucher disease: Marrow packing → ↑ pressure
- Systemic lupus erythematosus (SLE): Vasculitis + steroids; 5-40% develop ON
- Caisson disease: Nitrogen air embolization from dysbaric phenomena
- Radiation: Vasculitis results in ON
- HIV/AIDS: May relate to antiretroviral therapy or hyperlipidemia
- Alcohol abuse: Likely due to fat emboli from liver

Associated abnormalities

- Major morbidity from ON is not from infarct, but is result of healing process
  - Infarcted bone is as strong as normal bone
  - Healing weakens bone by resorbing dead bone
- Bones that are mostly covered with articular cartilage (such as femoral head) are particularly at risk for ON because of tenuous blood supply

MRI

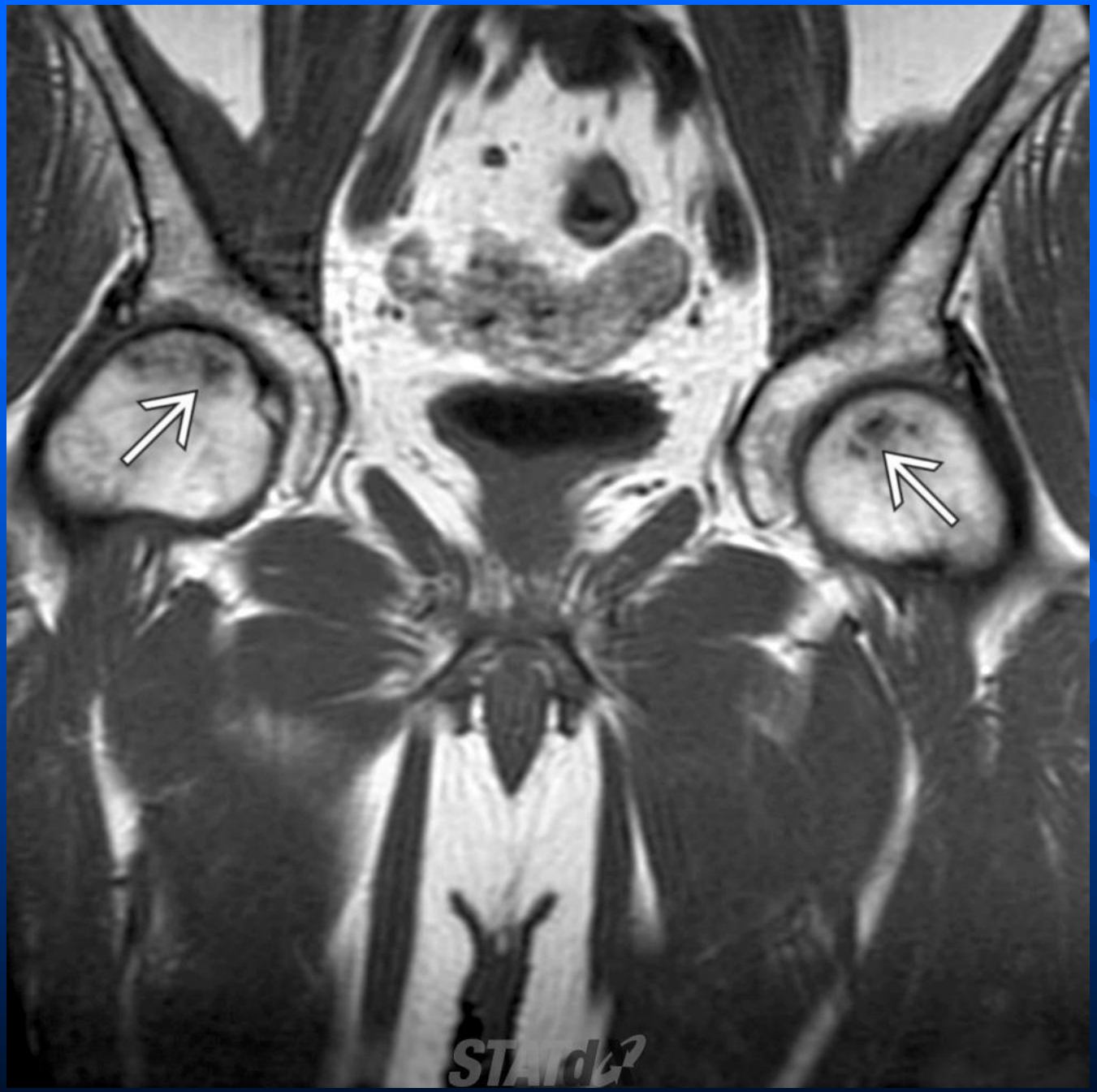
- Initial MR findings: Nonspecific bone marrow edema↓ T1W signal, ↑ signal on fluid-sensitive sequences
- Edema may extend from femoral head into femoral neck
  - Bone marrow edema pattern has extensive differential diagnosis
- During 1st few months following infarct, infarcted bone will appear normal;
- other than edema, MR changes do not occur until healing has begun
- Stages within infarcted bone progress from normal marrow → hemorrhage → edema → fibrosis
  - T1WI: Bright marrow → hypointense → dark
  - T2WI: Hypointense marrow → bright → dark

Staging, Grading, & Classification

- Steinberg classification: Based on radiographic appearance and location of lesion.
  - Stage 0: Normal radiographs, MR, and bone scan of at-risk hip (often contralateral hip involved, or patient has risk factors and hip pain)
  - Stage I: Nl radiograph, abnormal bone scan/MR
  - Stage II: Cystic or sclerotic radiographic changes
  - Stage III: Subchondral lucency or crescent sign
  - Stage IV: Flattening of femoral head
  - Stage V: Joint space narrowing
  - Stage VI: Advanced degenerative disease
  - Extent of disease grouped into < 15%, 15-30%, or > 30% (extent of femoral head involvement for stage I and II, extent of crescent fracture in stage III, and amount of articular surface involved in stage IV)

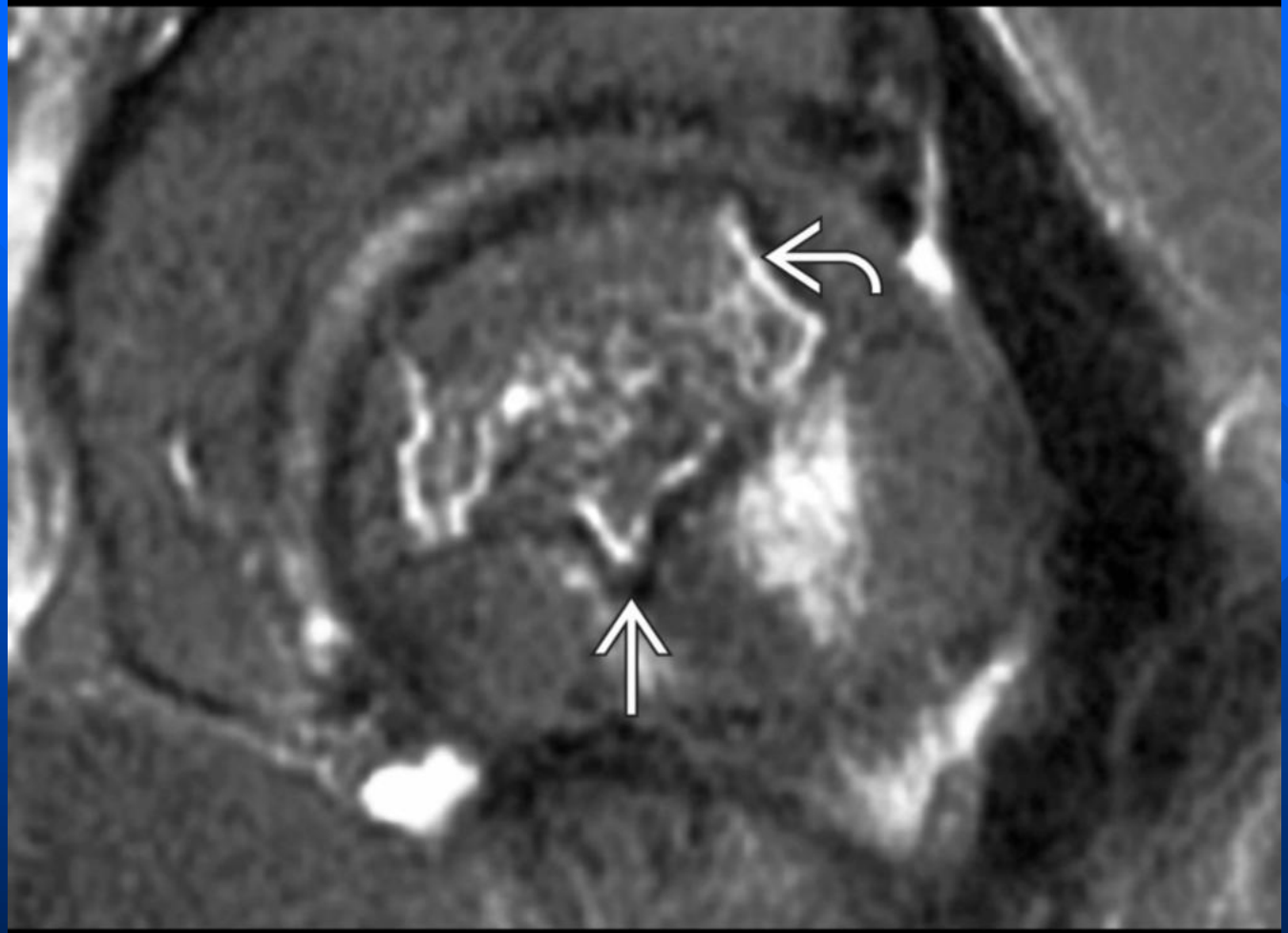
DDX:

- Bone Marrow Edema Pattern
  - Extensive differential diagnosis, including transient osteoporosis of hip, infection, neoplasm; may require time before definitive diagnosis becomes apparent
- Insufficiency Fracture of Femoral Head
  - Patient population: Elderly, osteoporotic women
  - ± significant articular surface collapse, fragmentation
  - Does not develop double line sign
  - Usually absent risk factors for ON

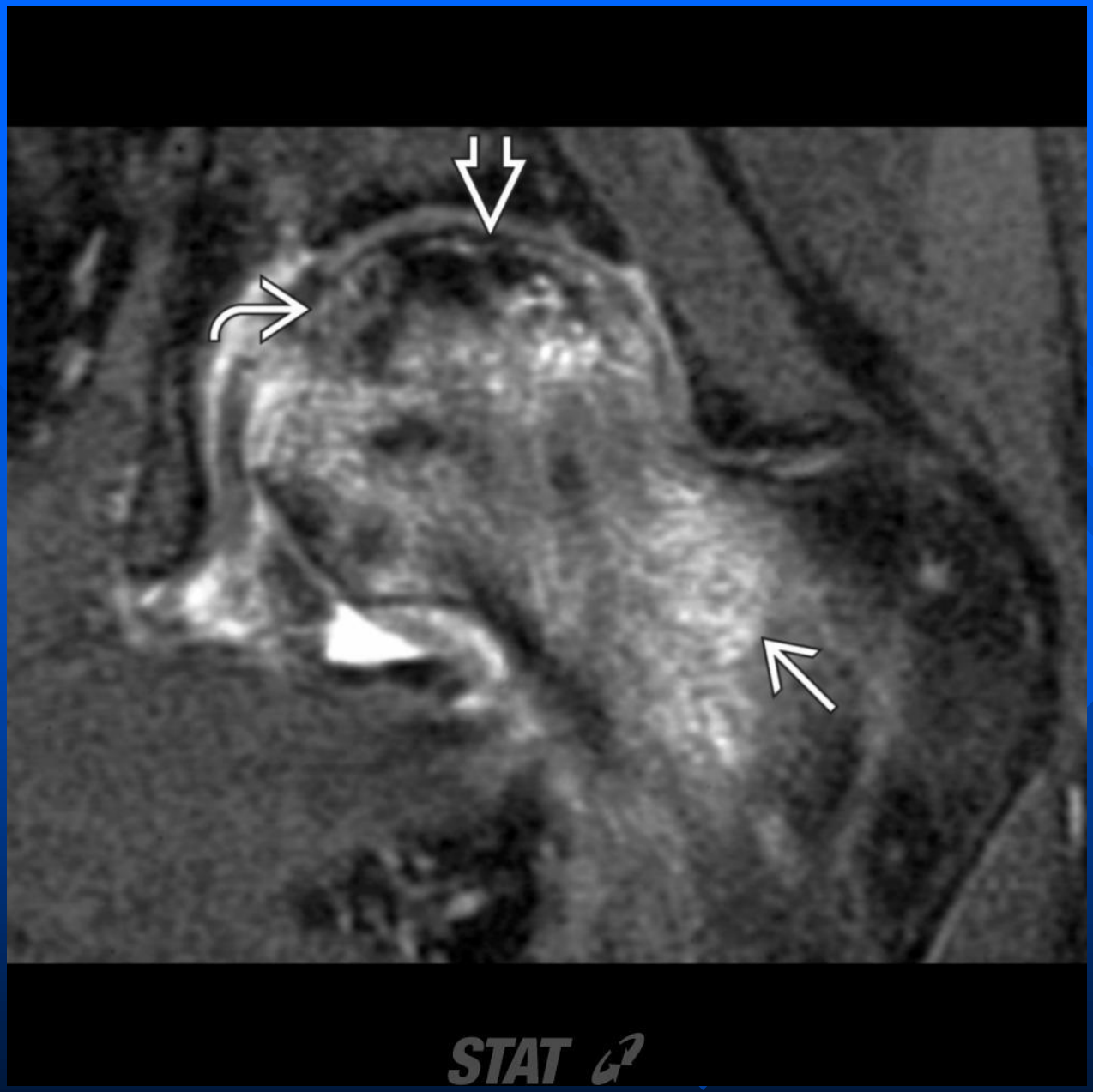


Coronal T1WI MR of the earliest changes indicative of ON is shown. Band-like foci (white solid arrow) of low T1W signal are present in the anterior aspect of each femoral head.

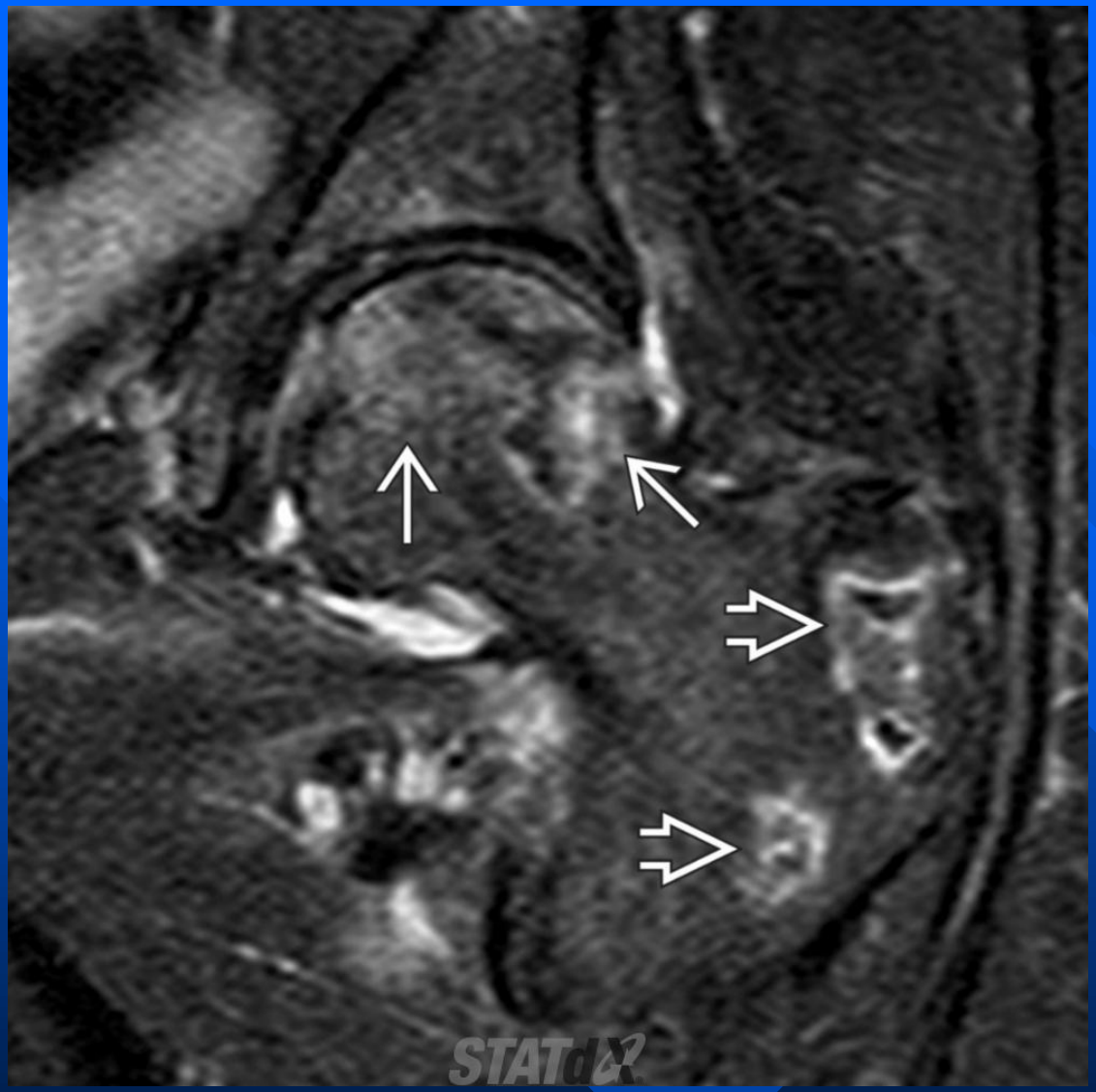
Double line sign



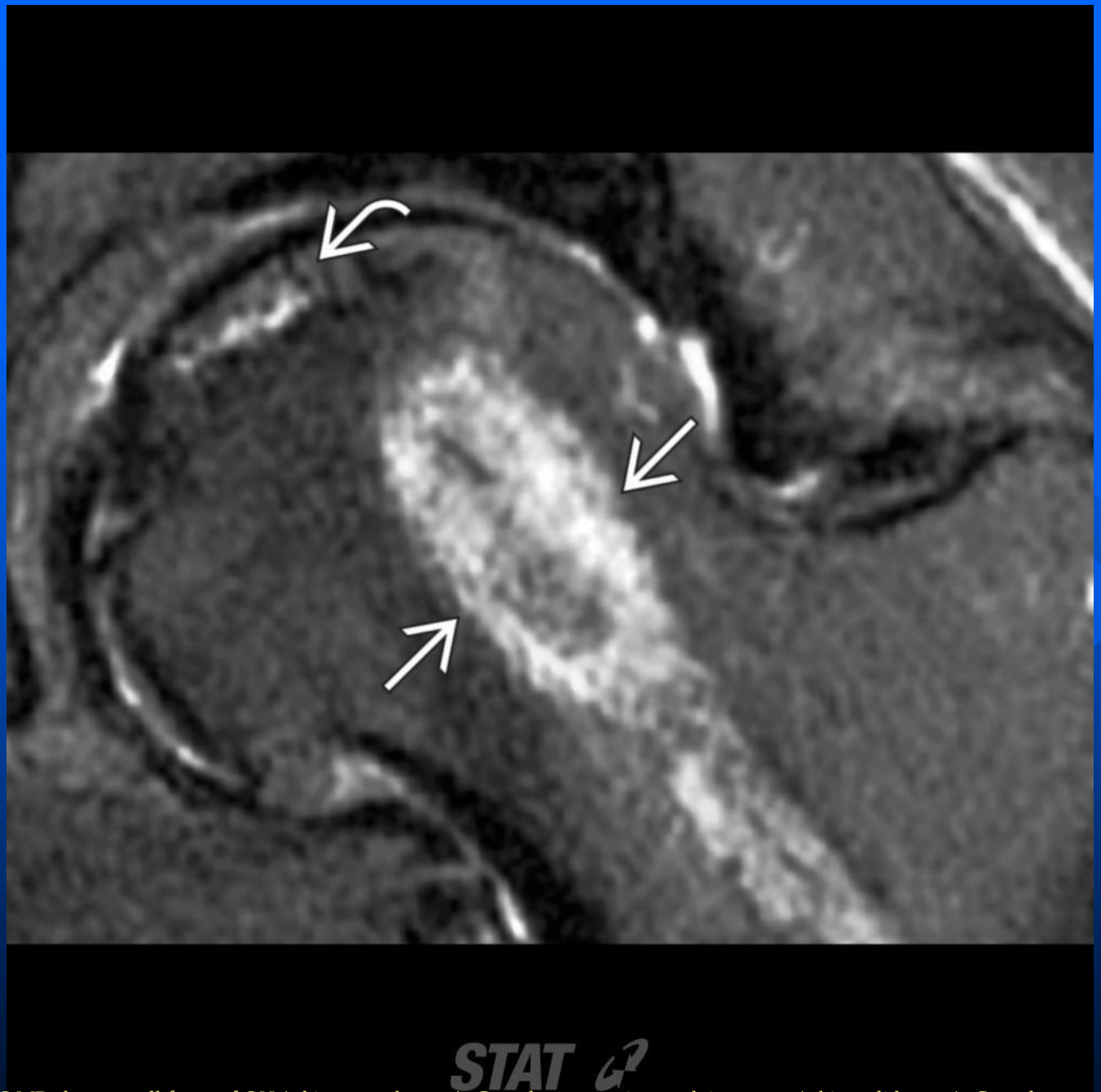
Coronal PD FS MR shows characteristic double line sign of ON. The hypointense outer dark line (white solid arrow) represents sclerosis at the border between the infarcted and normal bone. The bright line (white curved arrow) is created by the advancing granulation tissue/inflammatory response.



Coronal PD FSE FS MR shows subtle subchondral fracture (white curved arrow), articular surface irregularity (white open arrow), and extended hyperintense bone marrow edema pattern (white solid arrow) in the femoral head and neck. This edema pattern is most common in stage III disease and is highly associated with pain.



Coronal STIR MR with ON (white solid arrow) and bone infarcts (white open arrow) 2° to chemotherapy is shown. While the etiology is the same, terminology associated with these lesions is often confusing. ON and avascular necrosis refer to subchondral lesions, while bone infarct describes lesions distant to an articular surface.



Coronal PD FSE FS MR shows small focus of ON (white curved arrow). Core decompression track is present (white solid arrow). Core decompression is best used for stages I and II. Once fracture/collapse have occurred, the changes are irreversible.