Traumatic and Iatrogenic Osteonecrosis

Current Concepts

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**Definition**

**AVN** = massive necrosis of bone and bone marrow occurring as the only, or largely predominant abnormality (usually due to systemic factors)

**Local AVN (osteonecrosis)**

- trauma or microtrauma
- vertebral osteonecrosis
- idiopathic
- small bones
- iatrogenic
Blood supply of immature long bones

1) Metaphyseal vessels
2) Proper nutrient arteries
3) Periosteal vessels
4) Epiphyseal arteries
5) Perichondral arteries
Multiple vascular pathways contribute to an adaptive response to traumatic disruption of bone circulation.

The microcirculation is not merely a passive conduit for blood flow, but plays an active role in controlling bone processes.

The gold standard for experimental measurement of bone blood flow is the radioactive microsphere technique.

<table>
<thead>
<tr>
<th>TABLE I Typical Values of Blood Flow in Different Regions of Bone</th>
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<tbody>
<tr>
<td>Blood Flow (mL/min/100 g)</td>
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<tr>
<td>---------------------------</td>
</tr>
<tr>
<td>Cortical bone</td>
</tr>
<tr>
<td>Cancellous bone</td>
</tr>
<tr>
<td>Cortical marrow</td>
</tr>
<tr>
<td>Periosteum</td>
</tr>
</tbody>
</table>
Noninvasive methods of measuring bone blood perfusion

J.P. Dyke¹ and R.K. Aaron²

- Measurement of **perfusion** within the bone with dynamic contrast-enhanced MRI in models of osteoarthritis and the femoral head

- Bone **blood flow and remodeling** using ¹⁸F-Fluoride PET in fracture healing and osteoarthritis
Mechanisms for development of osteonecrosis

1. Blood vessel disruption
   - Trauma

2. Intraluminal obliteration
   - Fat microemboli
   - Thrombosis (sickle cell)
   - Bubbles (caisson)
   - Intravascular coagulation

3. Increased marrow pressure
   - Adipocyte hypertrophy
   - Bone edema

4. Cytotoxicity
   - Viability of osteoblasts, osteocytes

Intraosseous blood flow

Ischemia

- Favorable vascular anatomy
  - Favorable red/yellow marrow ratio
  - Reversible

Bone necrosis

- Genetic factors?
  - Vulnerable vascu larization
    - Fat-rich marrow
    - Epiphyses, metaphyses +++
  - Critical ischemia

Limited size
- Limited loads (<1/3 weight-bearing area, metaphysis)
  - Good outcome

Extensive
- Heavy loads (>1/3 weight-bearing area)
  - Poor outcome
2 types of osteonecrotic disorders

a) **Medullary** osteonecrosis is caused by interference in the blood supply to the medullary cavity, which results in trabecular bone and cells death (main lipocytes)

b) **Corticocancellous** osteonecrosis involving a joint. The trabeculae and often the subchondral bone both die, and the region does not calcify as occurs in medullary disease, since there are far fewer lipocytes in these regions
Causes of bone necrosis

Idiopathic - Secondary

Traumatic

Microtrauma or unclear

Iatrogenic - Postoperative
Traumatic osteonecrosis of the hip

- 16% nondisplaced subcapital fractures
- 27% displaced subcapital fractures
- 3% anterior hip dislocations
- 13% posterior hip dislocations

Transcervical fractures and compression fractures of the femoral head also may lead to osteonecrosis
deep branch of the MFCA during **hip dislocation**:

After complete capsulectomy and tenotomy of all external rotators, except for the tendon of **obturator externus**, the head of the femur is dislocated with external rotation.

There is no stretch or compression of the deep branch of the MFCA during dislocation and the normal course of the vessel remains unchanged. Obturator externus and its tendon protect the vessel.
Association of femoral head fractures with posterior hip dislocation ranges from 4% to 17%.

A first classification of these fracture dislocations was proposed by Pipkin in 1957.

It is classified according to the location of the head fracture in relation to the fovea.
New surgical approach for better access and visualisation for the treatment of femoral head fractures, using the “trochanteric flip” osteotomy

Thus inspection of the entire hip joint and accurate fragment reduction under direct visual control are possible.
Hip dislocation and femoral neck fracture: Decision-making for head preservation

Moritz Tannast\(^a\), Philip W. Mack\(^b\), Bernd Klaeser\(^c\), Klaus A. Siebenrock\(^a\)
Type I open fracture of the R femur in a polytrauma patient. Hypovolemic shock was probably the cause of AVN
ANFH develops in 0.5–1% of all pertrochanteric fractures

- mostly within 4 years after the injury

- patients older than 50 years of age
Results from disruption of the blood supply caused by fracture and/or dislocation

34% of 3-part fractures

90% of 4-part fractures

Almost 100% in splitting head fractures
Traumatic osteonecrosis of the humeral head

Arcuate a. Vessels from GTB Posterior circumflex a (x3 > anterior)

Important anastomoses postero-medial hinge
Ischemia predisposition factors

- a) length of medial metaphyseal head extension (< 8 mm in ischemic heads)

- b) integrity of the medial hinge (43 / 55 ischemic heads > 2 mm)

- c) splitting head component
Valgus impacted proximal humeral fractures and their blood supply after transosseous suturing

University hospital Patras 2000-2002

16 patients with 4-part VI fractures

11 women, 5 men (AA 44.6 Y)

Preop and postop (8-10/52) angiography

Transosseous suturing

Mean fup 40 months

Impaction angle (mean 42.4°)

Medial hinge integrity (mean 1.4 mm)
Image processing

Segmentation technique

Area of interest (AOI)

Thresholding

Sobel filters

↓ artifacts

Sharpening

Masks

“big vessels” = diameter > 0.5
- Malunion was present in nine patients (5%) at the time of the last follow-up......

- Humeral head osteonecrosis was seen in eleven (7%) of the 165 patients; four demonstrated total and seven, partial collapse.
Kienbock’s disease

Kienbock's disease is caused either by repeated minimal trauma or a single acute episode.

↑ incidence involves frequent impact to the wrist, such as hammering

74% negative ulnar variance

70% of lunates, multiple vessels in a or Y pattern
30% (I pattern), these lunates at increased risk
Kienböck’s Disease

Pedro K. Beredjiklian, MD

**TABLE 1. Lichtman Classification of Kienböck’s Disease**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>Stage 1</td>
<td>Normal x-ray, signal intensity changes on MRI</td>
</tr>
<tr>
<td>Stage 2</td>
<td>Lunate sclerosis on plain x-ray; fracture lines may be present</td>
</tr>
<tr>
<td>Stage 3</td>
<td>Collapse of the lunate articular surface</td>
</tr>
<tr>
<td>Stage 3A</td>
<td>Normal carpal alignment and height</td>
</tr>
<tr>
<td>Stage 3B</td>
<td>Fixed scaphoid rotation, proximal capitate migration, loss of carpal height</td>
</tr>
<tr>
<td>Stage 4</td>
<td>Lunate collapse along with radiocarpal or midcarpal arthritis</td>
</tr>
</tbody>
</table>


**TABLE 2. Treatment of Kienböck’s Disease According to Stage**

| Stage 1 | Cast immobilization for 3 months                                           |
| Stage 2 to 3A, ulnar negative variance | Radial shortening; ulnar lengthening; capitate shortening                  |
| Stage 2 to 3A, ulnar positive variance | Vascularized bone graft and external fixation; radial wedge or dome osteotomy; capitate shortening |
| Stage 3B | Intercarpal arthrodesis (scaphotrapezio-trapezoidal, scaphocapitate); lunate excision; radial shortening; proximal row carpectomy |
| Stage 4 | Proximal row carpectomy; wrist arthrodesis; wrist desenervation            |
Preiser’s disease

Or post-traumatic AVN of the scaphoid?

Repeated microtrauma maybe a causative factor
Idiopathic osteonecrosis of the scaphoid (Preiser’s disease) – MRI gives new insights into etiology and pathology

R. Schmitt\textsuperscript{a,*}, S. Fröhner\textsuperscript{a}, J. van Schoonhoven\textsuperscript{b}, U. Lanz\textsuperscript{c}, A. Gölles\textsuperscript{a}

European Journal of Radiology 77 (2011) 228–234

3 pathoanatomical zones are differentiated

1. the proximal scaphoid pole is the “zone of osteonecrosis” in Preiser’s disease

2. the middle scaphoid section in Preiser’s disease is the “zone of repair”

3. the distal scaphoid section is affected late and bone marrow viability is maintained over a long period representing the “zone of viability”.

\[\text{Image}\]
Kümmell’s disease

The intravertebral vacuum sign was first described by Kümmell in 1895.

Several terms have been used to describe this phenomenon, including intravertebral vacuum, delayed vertebral collapse, vertebral osteonecrosis and vertebral pseudarthrosis.

A fracture of the osteoporotic bone could lead to necrosis, Alternatively, ischemia/necrosis of the weakened osteoporotic bone could cause a fracture.
Kummell’s disease: delayed post-traumatic osteonecrosis of the vertebral body

Richard Ma • Robert Chow • Francis H. Shen
Post-traumatic osteonecrosis of the talus

It has been suggested that initial fracture displacement and delayed fracture fixation are associated with an increased incidence of osteonecrosis.

HAWKINS classification

Type I (0-20%)

Type II (20-50%)

Type III (~100%)

Type IV (100%)
Osteonecrosis with collapse of the dome occurred in twelve (31%) of thirty-nine patients. Osteonecrosis was seen in association with nine (39%) of twenty-three Hawkins group-II fractures and nine (64%) of fourteen Hawkins group-III fractures.

The mean time to fixation was 3.4 days for patients who had development of osteonecrosis, compared with 5.0 days for patients who did not have development of osteonecrosis.
Mean delay in fracture fixation 12.9 days

Hawkins sign in 59% of fractures at a mean of 7 weeks.

AVN developed in 7 of 17 fractures, with mean fixation time of 12.5 days; Hawkins sign was present in 20%.
Osteonecrosis after arthroscopic surgery
13 year-old patient
Avulsion of GH ligaments
Diagnostic arthroscopy/Open repair
6 weeks after surgery
Resolved spontaneously in 12 months
Humeral head osteonecrosis following arthroscopic rotator cuff repair

V. Beauthier · S. Sanghavi · E. Roulot · P. Hardy

…The presented shoulder necrosis may have occurred due to the use of multiple anchors which may have interrupted blood supply of the anteroinferior part of the head…….
First report by Brahme et al. in 1991 (after routine arthroscopic meniscectomy)

Current terminology **ONPK** (..SPONK)

ONPK after arthroscopic meniscectomy has been reported in 9 clinical studies with a total of 47 patients (2007)

Both genders were equally affected with a mean age of 58 years

87% of pt medial meniscus tear
OPNK - pathophysiology

The etiology of ONPK remains unclear.

The meniscal tear itself seems to have an association with osteonecrosis even before surgery has been performed.

**Increased tibiofemoral contact pressure** after meniscectomy may predispose to fracture of the cartilage and subchondral bone with an intraosseous leak of synovial fluid and subsequent osteonecrosis.

The **pathologic cartilage** has increased permeability for the arthroscopy fluid, which might lead to subchondral edema and consequent...
OPNK – differential diagnosis

In patients with persistent or worsening symptoms after knee arthroscopy:

(1) a missed diagnosis of early-stage osteonecrosis of the knee (SPONK),

(2) ONPK,

(3) a transient lesion that shares the BME pattern on MRI with SPONK and ONPK, and

(4) a recurrent meniscal tear
OPNK – differential diagnosis

Difficult to distinguish OPNK from SPONK in preop MRI

<table>
<thead>
<tr>
<th>Table 5. Five Radiographic Stages of SPONK</th>
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<tbody>
<tr>
<td>Stage</td>
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<tr>
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</tr>
<tr>
<td>1</td>
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<td>4</td>
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<td>5</td>
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In elderly patients delay MRI exam up to 6 weeks after onset
Case Report

Osteonecrosis of the Femoral Head after Hip Arthroscopy

Danielle L. Scher MD, Philip J. Belmont Jr MD, Brett D. Owens MD

...the cause of femoral head osteonecrosis in our patient may have been secondary to disruption of the primary blood supply to the femoral head during portal placement or we believe more likely as a result of a traction injury.