Post-traumatic Osteoarthritis

Once the hammer has hit…

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Post-traumatic osteo-arthritis

- Common sequela of high energy impact injury
- Progressively debilitating
- May require surgery: fusion / replacement
- Delayed onset possible
- Super-imposed on pre-existing early OA

Early prognosis / prediction

- Early prognosis / prediction of PTOA difficult
- Incomplete understanding of
  - injury pathways
  - repair mechanism

Early prognosis / prediction

- Early X-rays not reliably predictive:
  - early joint space preservation with compromised cartilage viability
- MRI can cause confusion:
  - bone bruise with intact cartilage surface
- Bone scan may become negative
  - resolution of inflammation, remodelling

Early prognosis / prediction

- Patient may initially do quite well
- Reasonable initial restoration of joint function
- Compromised long term function possible
- May still be serious problem in long-term

Early prognosis / prediction

- PTOA - medical perspective :
  - appropriate counceling
  - supportive therapy as required
  - treatment progressive over time
  - no need to be definitive in prognosis

Early prognosis / prediction

- PTOA - personal injury legal perspective:
  - can be major determinant of magnitude of injury
  - timely settlement vs full appreciation

Early prognosis / prediction

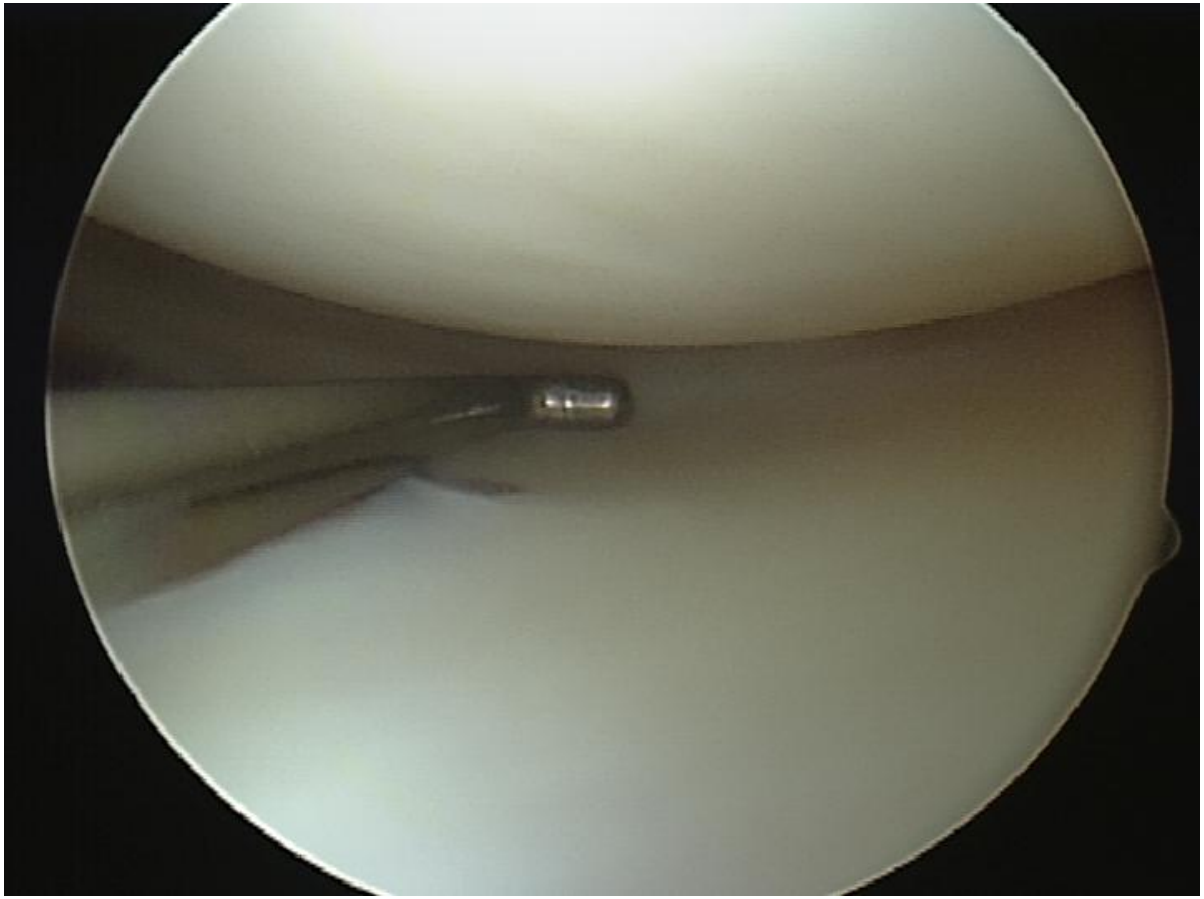
aggravation of pre-existing OA

- widely varying opinions common
  - risk of future OA readily downplayed
  - balanced assessment?

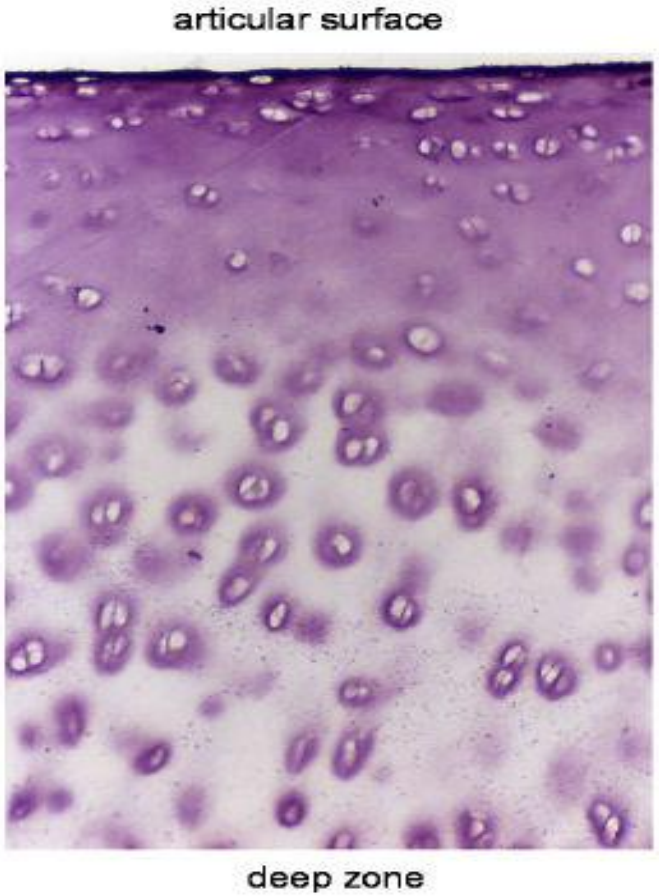
Articular cartilage - basics

- 2-3 mm layer of soft smooth gliding material
- ‘firm jello’
- Superficial layer → smooth
- Deeper layer → soft, compliant
- Nutrition by joint fluid
- Scattered living cells in cartilage substance
- Anchored to bone

Articular cartilage - basics



Cartilage basics



thin gliding layer

scattered chondrocytes

Articular cartilage - basics

- Cartilage cells essential for maintenance
- Ongoing surface wear with use
- Ongoing surface repair as required
- Repair capability diminishes with age

Traumatic joint injury

- Force applied overwhelms tissue resilience
  - bone injury → bone bruise, fracture
  - ligament injury → sprain, rupture
  - cartilage injury → contusion, disruption
  - soft tissue injury → contusion, disruption

Traumatic joint injury: multiple mechanisms

- Bone fracture:
  non-anatomic fracture healing →
  altered force transmission through joint →
  cartilage overload

- Classic orthopaedic approach → restore anatomy after fracture with cast or surgery to optimize outcome

Traumatic joint injury: multiple mechanisms

- Bone bruise:

  indicator of contusive force through joint surface

  possibly altered force transmission

  associated with future PTOA

- Readily dismissed as ‘non-significant’ finding

Traumatic joint injury: multiple mechanisms

- Ligament injury
  may compromise joint stability →
  altered force transmission through joint →
  cartilage overload

- Classic orthopaedic approach → restore stability after ligament injury to optimize outcome

Traumatic joint injury: multiple mechanisms

- Cartilage injury

Sharp injury: may heal side-to-side

Blunt injury

→ grossly disrupted: poor healing potential

→ contusion only: may cause irreversible damage to cartilage, in absence of gross disruption (!)

- Classic orthopaedic approach: cannot be fixed

Traumatic joint injury: multiple mechanisms

- Soft tissue injury (capsule, synovium etc)
  - bleeding, inflammation-related swelling
  - multiple ‘cartilage unfriendly’ mediators released
  - exact role unknown

- Classic orthopaedic approach: early aspiration vs ‘letting it settle’ → dealer’s choice

Osteo-arthritis after intra-articular fracture

- not fixed ‘properly’ → mechanical overload
- cartilage surface damaged
- cartilage substance damaged
- role of mediators

Clinical Evidence

- no readily available ‘actuarial’ data
- ‘bits and pieces’ only
- Multiple variables:

- Patient characteristics (age, gender, general health etc, etc)
- Injury severity
- ‘Adequacy’ of treatment
- Natural history of pre-existing OA etc

Classical laboratory evidence

- Animal model

  → difficult to create relevant model (species, legged-ness, insult, treatment etc)

  → both instability and contusive injury implicated in OA

Classical laboratory evidence

- Cell / tissue culture
  - → more controllable environment
  - → both contusive force and mediator toxicity implicated in cartilage injury
  - → ‘leap of faith’ remains

A bolt of lightning

- Yuki Togichi, Thomas Brown, U of Iowa
- '*Distribution and progression of chondrocyte damage in a whole-organ model of human ankle intra-articular fracture*'
- Presented March 2011, VOS meeting
- Published March 2011, JBJS

Dr. Brown paraphrased

- Research focus: ankle pilon (hammer) fracture
- High energy fracture of the ankle plafond
- Sudden axial load by the talus, acting as a hammer
- Severe injury, high rate of PTOA

Dr. Brown paraphrased
1/ intra-operative fragment collection

- Collected non-usable bone/cartilage fragments from operating room at time of surgical treatment of pilon fractures
- Noted decreased cartilage viability in culture

Dr. Brown paraphrased
1/ intra-operative fragment collection

- Multiple explanations:
- blunt impact trauma
- mediator exposure (hours/day after injury)
- interruption of nutrition
- processing / handling
- other?

Dr. Brown paraphrased
2/ development of mechanical fracture model

- Pilon fractures have ‘typical’ fracture pattern
- Cadaveric ankles were used to re-create this
- Custom drop tower
- Ankle potted upside down
- Controlled hit of talus by drop weight

→ Reproducible fracture pattern

→ Similar to observed clinically

Dr. Brown paraphrased
3/ fracture creation and cartilage culture

- 7 freshly amputated lower legs
- Directly from OR to drop tower
- Fracture created (all successful)
- Cartilage cultured
- Cartilage assayed up to 48 hours

Dr. Brown paraphrased
3/ fracture creation and cartilage culture

- Cartilage close to fracture line (<1 mm)
- Cartilage away from fracture line (>3mm)

→ Decreased early viability close to fracture line (8% vs 1% dead chondrocytes)

→ More rapid further decline in viability (26% vs 9% dead chondrocytes)

Note: no statistical difference between t=0 and t=48h in viability away from fracture line (i.e. 1% ~ 9%)

Dr. Brown paraphrased
Conclusion

- Blunt impact can immediately lead to cartilage cell death in macroscopically normal cartilage
- Further cell death occurs over 48 h, in absence of bleeding, inflammation etc
- Possibly, local mediators play a role

In-vivo assessment of cartilage viability

- Promising area of research

- MRI → multiple sequences and protocols being evaluated
  → most common: dGEMRIC
  lack of validation

In-vivo assessment of cartilage viability

- X-ray assessment of preservation of joint surface

→ Specificity acceptable

→ Not sufficiently sensitive to conclude:

‘the risk of developing arthritis is low’

In-vivo assessment of cartilage viability

- Arthroscopy:

→ Assessment of macroscopic integrity of joint surface

→Adjunct to assess water content has been on brink of introduction for several years.

Lack of validation

What is next?

- The understanding of PTOA is far from complete
- Inferences can be made about the various proposed mechanisms of injury
- It is not necessary to accept without contest a '*looks good, will do fine*' opinion
- Significant conjecture will remain for the foreseeable future

Thank you