RHEUMATOLOGIC
CONSIDERATIONS IN THE
GERIATRIC PATIENT

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TYPICAL PRESENTATIONS OF RA

1. Insidious polyarthritis
2. Chronic polyarthritis (deforming)
3. Acute migratory polyarthritis
4. Palindromic rheumatism
5. JRA-Still’s Variant
6. Monarticular RA
7. Robust reaction type
8. Rheumatoid nodulosis
9. Elderly Onset

CLINICAL CHARACTERISTICS OF ELDERLY ONSET RA (EORA)

Onset after age 60
Acute onset common
F:M Ratio<3:1
Increased incidence of systemic symptoms
Predilection for large joints

EORA COMPARED TO YORA

Increased Incidence
Shoulder & Hip Synovitis
Acute Onset
PMR-like presentation
Elevated ESR

Decreased Incidence
Small Joint disease
Extra-articular disease
Nodules
Seropositivity
**REVISED CRITERIA FOR RA**
*(ACR 1987)*

- Morning Stiffness
- Swelling of ≥ 3 joints
- Swelling of wrist, MCP, or PIP joints
- Symmetrical joint swelling
- Hand x-ray changes
- Subcutaneous nodules
- Positive Rheumatoid factor

**DIFFERENTIAL DIAGNOSIS OF RA**

- CTD
- SBE
- Thyroid
- HPO
- Infection
  - Fibromyalgia
- Osteoarthritis
- Seronegative Spondylitis
- Gout (Tophi)
- PMR (Elderly)
- Rheumatic Fever
Anti-Cyclic Citrullinated Peptide Ab (Anti-CCP)

- Detected by Elisa technique
- As sensitive as (47-80%) but more specific (97%) than IgM rheumatoid factor
- Marker of erosive disease
- Undifferentiated CTD-may predict RA
- Detected in “Healthy” population years before clinical RA
- Found in 40% “Seronegative RA”

RHEUMATOID ARTHRITIS

- Treat aggressively, EARLY!
- The most significant damage to the joints occurs in the initial 1-2 years of disease.
CURRENT MANAGEMENT OF RA

RITUXIMAB
ABATACEPT

SINGLE AGENT:
SSZ-HCQ-TNF
LEF-GOLD (?)

COMB-TX:
1) MTX & SSZ
2) TRIPLE TX
3) MTX & TNF

MTX

NSAID

SINGLE AGENT:
SSZ-HCQ-TNF
LEF-GOLD (?)

COMB-TX:
1) MTX & SSZ
2) TRIPLE TX
3) MTX & TNF

MTX

NSAID

ADJUNCTIVE STEROIDS (ORAL OR IA)

Additional TNF Inhibitors

A) Certolizumab Pegol (Cimzia)
   - Prohibited + addition of TDM to reduce infertility and immunogenicity
   - Dose: 600 mg sub-q monthly, week 2 & week 4; continued dose depending on response

B) Golimumab (Simponi)
   50 mg sub-q every 4 weeks

Interleukin – 6 Inhibitor (IL-6)

* Tocilizumab (ACTEMRA) –
  * IL-6 Receptor Inhibitor indicated for RA after at least one (1) TNF Inhibitor has failed
  * Dose: 8 mg/kg IV, increased to 8 mg/kg based on clinical response (linear extension). IV dose q 4 weeks
  * Side Effects: Infections, headaches, HTN, ↑ LFTs, Amyloidosis, ↓ WBC or Platelets
Systemic Corticosteroids

**Advantages**
- Control of symptoms
- May facilitate disease control by DMARDs
- Control of life-threatening emergencies

**Disadvantages**
- No effect on disease progression
- Hypercorticism

Source: Hardin and Longenecker, 1992

**PROGNOSIS OF EORA IN COMPARISON TO YORA**

Shorter duration of the disease
Improved joint counts
Lower ESR
Better physician assessment
A 63-year-old female presents to the office with the complaint of difficulty getting out of a chair. She also has vague symptoms such as fatigue and lack of energy in association with morning stiffness and aching in the proximal portions of her arms and legs. Lab data reveals a mild anemia, normal biochemistry profile, and a westergren sedimentation rate of 75 mm/hr. PE is unremarkable.

CHARACTERISTICS OF POLYMyalGIA RheumAtICA

- Older patient
- Normal physical examination
- Aching and stiffness

CLINICAL FEATURES OF PMR (SYMPTOMS AND SIGNS)

- Pain  
  - Disability
- Stiffness  
  - Tenderness
- Fatigue  
  - Limitation of Motion - areas of involved
- Depression  
  - Arthritis
- Carpal Tunnel Syndrome
DEFINITION OF PMR

1. Pain in neck, shoulders, and pelvic girdle for at least one month. Morning stiffness and gelling without muscle atrophy or weakness.
2. Age ≥ 50 years old
3. ESR ≥ 50 mm/hr
4. Relief of symptoms within 4 days with as low as 10-15 mg Prednisone per day

DIFFERENTIAL DIAGNOSIS OF PMR

RA and other CTD
Viral Myalgias
Polymyositis
Multiple Myeloma
Osteoarthritis
Fibromyalgia
Occult CA
Occult Infection

LAB IN PMR

Anemia
ESR (≥ 50 mm/hr)
RA (-)
ANA (-)
Muscle Enzymes – Normal
EMG – Normal
Liver Profile
PMR – THERAPY

A) NSAIDS – trial warranted?
   - will not prevent vascular complications

B) Corticosteroids - *Drug of choice (low dose)
   If sx free x 6-12 months, may D/C steroids
   50% may relapse
   ? Add MTX (steroid sparing)
   Prognosis
   ? Assoc. with ↑ CV mortality

MANAGEMENT OF PMR

ASA or NSAID’s
Corticosteroids
   - Dosage
   - Duration
Biopsy
   - Indications

** N.B. 1 – Sudden Blindness 7 Years After Dx.
N.B. 2 – PMR May Evolve into RA
GCA – THERAPY

Corticosteroids 0.7-1.0mg/kg/day
- maintain x one monthly before tapering

* Addition of 81mg ASA
  May prevent occlusive disease

* Add Imuran/CTX/MTX?
  Steroid sparing

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“Rheumatologic Considerations in the Geriatric Patient”
Richard A. Pascucci, D.O.
**RHEUMATIC DISEASES: ASSOCIATED CRYSTALS**

<table>
<thead>
<tr>
<th>Crystal</th>
<th>Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Monosodium urate monohydrate</td>
<td>Gout</td>
</tr>
<tr>
<td>Calcium pyrophosphate dihydrate</td>
<td>Pseudogout</td>
</tr>
<tr>
<td>Dicalcium phosphate dihydrate</td>
<td>?</td>
</tr>
<tr>
<td>Apatite</td>
<td>Osteoarthritis?</td>
</tr>
<tr>
<td>Adrenal corticosteroid esters</td>
<td>Tendon, muscle, and/or synovial calcification</td>
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<tr>
<td>Cholesterol</td>
<td>Iatrogenic postinjection flare</td>
</tr>
<tr>
<td></td>
<td>None (chronic effusion)</td>
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</tbody>
</table>
Patient Demographics of Osteoarthritis

- Affects more than 22 million Americans
- About 80% of patients show x-ray evidence of osteoarthritis by age 55
- Peak incidence reported in patients over age 65
- Women affected approximately twice as often as men

SYMPTOMS OF OSTEOARTHRITIS

- Pain
  - Localized to characteristic joints
  - Aggravated by activity
- Stiffness
  - Generally less than 15 minutes duration
  - With inactivity
- Onset gradual and additive
- Acute and intermittent flares

CLINICAL FEATURES OF OSTEOARTHRITIS

Age: > age 40 (usually)
- Morning Stiffness: Usually insignificant
- Joint Distribution: DIP, PIP, First CMC, Knee, Hip, First MTP, Spine

- Insidious Onset
- Rare Systemic Manifestation
- Osteophytes and Eburnation
**JOINTS USUALLY SPARED IN OSTEOARTHRITIS**

- MCPs
- Wrist
- Shoulders
- Elbows
- Ankles

**DIFFERENTIAL DIAGNOSIS OF OSTEOARTHRITIS**

RA – ESR, DISTRIBUTION, SYSTEMIC, ETC.
Other DIP Diseases – Psoriatic, Reiter’s
CPPD – Distribution, Flares, Crystals, etc.
Localized Joint Disorders (Early) – Aseptic necrosis, PVS Infections, etc.

**Medical Management of OA Non-Pharmacologic Therapy**

- Patient Education – self-help, social support
- Weight loss
- Physical Therapy
  - ROM
  - Strengthening
  - Assistive Devices
- Occupational Therapy
- Aquatic Exercise Therapy
- Aerobics

Pharmacologic Therapy
- Analgesics – e.g. oral (acetaminophen) or Topical
- NSAID’s
- Opioid Analgesics (e.g. Propoxyphene, codeine)

Experimental Therapies
PHARMACOLOGIC THERAPY FOR PATIENTS WITH OA

**ORAL**
- Acetaminophen
- COX-2 Specific Inhibitor ??
- Nonselective NSAID plus Misoprostol or PPI
- Other Pure Analgesics
  - Tramadol
  - Opioids
- Intraarticular
  - Steroids
  - Hyaluronan
- Topical
  - Capsaicin
  - Methylsalicylate

*Choice of Agent(s) should be individualized


**Glucosamine Sulfate-Chondroitin Sulfate**

- Repair and Maintenance of Cartilage
- Several short-term controlled human studies show modest decrease OA symptoms
- May have Remittive Effect

**Hyaluronic Acid Treatment “Viscosupplementation”**

- Injected into knee joint for 3-5 consecutive weeks
- Equally as effective as Acetaminophen (pain relief) or Naprosyn
- No proof of altered joint Biology
- FDA Approved – side effects include local irritation or severe allergy (rare)
Future Directions in OA Therapy

- MMP inhibitors
- NO inhibitors
- COX-2 specific inhibitors
- Disease-modifying interventions

LATE-ONSET SLE
Occurrence after age 50
F>M
Frequent Misdiagnosis
Conservative Therapy

LATE-ONSET SLE CLINICAL MANIFESTATIONS
Arthritis
Rash
constitutional Sx.
Pleuritis/Pericarditis
Nephritis
Hematologic
LATE-ONSET SLE
LESS COMMON CLINICAL FEATURES

Lymphadenopathy
Raynaud’s Phenomenon
Neuropsychiatric Disease
Alopecia

DRUG – INDUCED SLE

1. Criteria
2. Female:Male Ratio
3. Black vs. White
4. Systems Spared
5. Serum Antibody
6. Clinical Symptoms
7. Predisposition –
   (a) HLA – type
   (b) Slow Acetylator
Lupus-like Syndrome: Drugs Implicated in Induction

**Anticonvulsants**
- Ethosuximide
- Mephénytoin
- Phenytoin
- Primidone
- Trimethadione

**Antimicrobial agents**
- Griseofulvin
- Isoniazid
- Nitrofurantoin
- Penicillin
- Streptomycin
- Sulfonamides
- Tetracycline

**Antihypertensives**
- Hydralazine
- Methyldopa
- Reserpine

**Antithyroid agents**
- Methylthiouracil
- Propylthiouracil

**Cardiovascular agents**
- β-Adrenergic blocking agents
- Procainamide
- Guanidine

**Psychotropic agents**
- Chlorpromazine
- Lithium carbonate

**Miscellaneous**
- Allopurinol
- Aminoglutethimide
- D-Penicillamine
- Gold Salts
- Methysergide
- Oral contraceptive
- Phenylbutazone
- Biologic Agents
Treatment of Postmenopausal Osteoporosis

FDA-Approved Indications

<table>
<thead>
<tr>
<th>Medicine</th>
<th>Prevention</th>
<th>Treatment</th>
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</thead>
<tbody>
<tr>
<td>Alendronate</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Risedronate</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Calcitonin</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Conjugated Estrogens</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Raloxifene</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>PTH</td>
<td>No</td>
<td>Yes</td>
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- Approved November 2002
- Anabolic Agent

Indications:
1. Postmenopausal ♀ @ High Risk for fx
   - Previous fx
   - Significantly Low Bone mass
   - Intolerant or unresponsive to other Tx.
2. ♂ - Primary or hypogonadal osteoporosis

CI: Paget's, ESRD, Pregnancy, METS, Osteomalacia, Stone Disease
TERIPARATIDE TX. FOR OSTEOPOROSIS CONT.

Risks: ↑ Osteosarcoma in Rats
(Use only for 2 years)
Side effects: Dizziness & Leg Cramps

Baseline lab: Ca++ Alk. Phos.
Po4 = 25-OH Vit D.
Creatinine
Dose: 20 μg Sub = Q daily
Cost: AWP = $7592/year

COMBINATION THERAPY

A) “The Effects of Parathyroid Hormone and Alendronate alone or in combination in postmenopausal osteoporosis”
Black DM, Greenspan SC, Ensrud KE, ET AL.
NEJM - September 25, 2003
Conclusion: No Evidence of synergistic effect

B) Raloxifene + PTH
Combination Better Than PTH alone.
Deal, C- Presented at ACR (October 2004)

DENOSUMAB

* Anti-Resorptive agent
- Inhibits RANKL

Trial Compared Denosumab to Alendronate (open case)
(412 pm females with low bone mass)
Denosumab
60 mg sub Q every 6 months
Results (24 months)

<table>
<thead>
<tr>
<th>Denosumab</th>
<th>Alendronate</th>
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</thead>
<tbody>
<tr>
<td>Bone Mass</td>
<td></td>
</tr>
<tr>
<td>Hip</td>
<td>↑ 5%</td>
</tr>
<tr>
<td>L. Spine</td>
<td>↑ 7%</td>
</tr>
<tr>
<td>Radius</td>
<td>↑ 1.75%</td>
</tr>
</tbody>
</table>
RANKL –

Receptor Activator of nuclear factor KAPPA B Ligand

- Mediates resorptive phase of bone remodeling
- Blocking the binding of RANK to its ligand inhibits the Osteoclast

BISPHOSPHONATE-ASSOCIATED AVN OF JAW (ONJ)

Review of 30 cases (1996-2006)

- 90% females
- 70% males
- New pain in 37%
- Old pain in 13%

56% cases after oral surgery (either in other 44% pts. w/o bisphosphonates)

41% pts. receiving bisphosphonates (mostly pamidronate)

Access, with frequent typically monthly injections of IV bisphosphonates and oral

BISPHOSPHONATE-ASSOCIATED AVN OF JAW (ONJ/cont.)

- CLINICAL PRESENTATION
  - Periosteal reaction or areas of radiolucency within bone – smooth or ragged borders?
  - Periodontal area – Pathological loss of bone
  - Ray view – white matter involvement

- Risk Factors
  - Frequency of bone involvement containing bisphosphonates
  - Recurrent fracture
  - DVT events of CA pts. w/o bisphosphonates
  - Cancer – Progressive Skeletal Malignancy

- Mechanisms?
Bisphosphonates and Fractures of the Subtrochanteric or Diaphyseal Femur

DM Block, MP Kelly, KH Connert, L Palermo et al
NEJM 362; 19: 1761-1771 May 13, 2010

*Secondary Analysis of 3 large, randomized Bisphosphonate Trials:
1) FIT (Fracture Intervention Trial)
2) FLEX (FIT Long-Term Extension)
3) HORIZON (Health Outcome and Reduced Incidence with Zoledronic Acid once yearly) Pivotal Fracture Trial (PFT).

Results: Review of 284 records for hip or femur fractures among 14,105 in those trials.

12 Fractures in 10 patients occurred in subtrochanteric or diaphyseal femur (2.3 per 10,000 patients years).

Bisphosphonates and Fractures of the Subtrochanteric or Diaphyseal Femur (con't)

*Relative Hazard: 1.03 in FIT
1.33 at FLEX
1.50 in HORIZON-PFT

Conclusions: 1) Occurrence of atypical fracture was rare, even among patients TX for 10 years
2) No significant increase in risk (but study underpowered for definitive conclusions)

* Not Significant
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Editorial

**Existing data about Subclinical Fractures and Bisphosphonates**
Elizabeth Shane, MD
Columbia Univ., New York
NEJM 362:19, 1625-1627 May 13, 2010

- Atypical fractures usually present 10-15 years before clinical occurrence, may be middle-aged men with osteopenia, association with secondary hyperparathyroidism.
- Concern for decisions: 1) 2-3% of all hip fractures (1.5 million worldwide in 2000 were osteoporotic)
- 2) Not all associated with Bisphosphonates
- 3) Such atypical fractures are seen in complex fractures of the hip.

Editorial (con't)

**What are the implications of the Black et al Study for Clinical Practice?**

1) Subclinical fractures are extremely rare
2) Many more devastating hip fractures are prevented by Bisphosphonates, then potentially caused by them
3) Detailed recommendations beyond scope of this editorial – consider Drug Holiday, use bone turnover markers
4) Use of alendronate for 10 years compared to 5 years was associated with significantly fewer vertebral and non-vertebral fractures in PPS with BMD –2.5 or below

E. Shane, MD

Summary

- Recognize that Osteoporosis is a preventable and often a clinically silent disease
- Understand the NCCS Guidelines and incorporate them into your daily practice
- Utilize the PC DA to maximize the management of your patients
- Be familiar with the osteocrine and therapeutic options available
“The Effects of Strontium Ranelate on the Risk of Vetricbral fracture in women with post menopausal osteoporosis”

NEJM 350:5, 459-468, Jan-29, 2004

STRONTIUM RANELATE

“Re-Launched” as a new compound

Mode of Action
- Stimulates Bone Formation
- Decreases Bone Resorption
- May Suppress PTH
- No Mineralization Defects

Dosage: 2 Grams/Day

VERTEBROPLASTY

Utilizes cement injection into bone for stabilization of compression fracture(s)

Patient Selection
- (1) Severe Back pain < 12 months
  - (Refactory to analgesics)
- (2) Vertebral body compression fracture(s)
  - (Pain elicited with palpation at specific level(s))
- (3) MRI/Bone scan-no other explanation
  *Osteoporotic or pathologic Fx treated
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Osteoporosis Therapy Options Postmenopausal Women

AGE
STAGE
- AT RISK/Osteopenia → Osteoporosis → Severe Osteoporosis

Risk Factors
- Increasing risk of fracture with age

Staging
- Higher BMD
- Lower BMD

Treatment Options
- Bisphosphonates
- Raloxifene
- Teriparatide
- Calcitonin

Cyclooxygenase Isoenzymes

Physiologic Stimulus
- COX-1
- Constitutive
- Platelets
- Endothelium
- Stomach
- Kidney

Inflammatory Stimulus
- COX-2
- Inducible
- Macrophages
- Leukocytes
- Fibroblasts
- Endothelial cells

Nonsteroidal Anti-Inflammatory Drugs (NSAIDs)

- Largest class of pharmaceutical agents used worldwide
- Commonest agents in the pharmacologic therapy of arthritis
- Effective in relieving pain, inflammation, and stiffness in arthritic patients
- Enhance function and improve quality of life in arthritis patients
- Good safety profile when prescribed and monitored appropriately
**UTILIZATION OF NSAIDs**

- **2000** - 111, 400, 000 Rx for NSAIDs in US
  - $5 Billion Cost
  - $2 Billion OTC NSAIDs
- **Today** - 17, 000, 000 Americans utilize on daily basis
  - $10 Billion Market Worldwide
  - Marketed directly to consumers

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**USE OF NSAIDs**

- Osteoarthritis
- Rheumatoid Arthritis
- Other arthritic conditions
- Pain syndromes
  - Musculoskeletal pain syndrome
  - Soft tissue pain syndrome

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**MECHANISM OF ACTION OF TRADITIONAL NSAIDs AND COXIBs**

- COX-1 "constitutive"
- COX-2 "inducible"
- Arachidonic acid
  - Nonsteroidal anti-inflammatory drugs (NSAIDs)
  - Cyclooxygenase (COX)
- Prostaglandins
  - Platelet function and homeostasis
- Thromboxane A2
  - Mediate pain, inflammation, and fever
  - Protection of gastric mucosa
TOXIC EFFECTS OF NSAIDs

- GI disorders (dysegestis, PUD, occult bleed)
- CNS
- Transient
- Rash
- Hypersensitivity Reactions (ASA Allergy)
- Hypotension
- Nephrotoxicity (edema, K+, ARF, papillary necrosis, interstitial nephritis)

Protective Effect of Chronic NSAID Use on Cognitive Decline in Older Persons

- Interview Study of 7671 Patients
  - personal-3 and Telephone-4
- Use SPPMOS
  - Short, Portable Mental Status Questionnaire
- Chronic NSAID Usage
  - 3 years at 2 Interviews
- Role of Amyloid (?) in Alzheimer's

SUMMARY: BENEFITS OF COX-2 SPECIFIC INHIBITION

- COX-2 Specificity
  - Inflammation and pain reduced, similar to non-selective NSAIDs
- COX-1 sparing
  - GI toxicity reduced, in contrast to non-selective NSAIDs
  - Lack of effect on platelets

Cox-2 Cardiovascular Effects Hypothesis

Inhibition of vascular PGI₂ (Prostacyclin) synthesis
And
Lack of Effect on Platelet Thromboxane Synthesis
↓ Imbalance
↓ Prothrombotic State
↓ Increased Thromboembolic CV Events

COXIBs/NSAIDs
(Traditional or Non-Selective)
Questions to be Answered

CV
- Role of COXibs and CV/Thrombotic Events
- Effect of NSAID + ASA on MI Prevention
- Do Non-Selective NSAIDs cause MI? (Traditional)

HISTORY OF CV EVENTS WITH COXIBs

- VIGOR Trial – Bussel, et al, 2000: Divergence of data after 6 weeks of therapy
- CLASS Trial – Silberstein et al, 2000: No statistical significance
- APPROVe Trial – Colombo: (Dyspnea, Pulmonary Edema, Heart Failure, etc.)
  - Divergence after 3 months (Twice as frequent adverse events)
  - 1890 patients: Suspension of Authorization for valdecoxib in April 2004

NEJM 2000;303: 1081-1091.
COX-2 PROVEN TO BE DOMINANT SOURCE OF PGI₂ (PROSTACYCLIN)

- Vasodilation
- Inhibits Platelet Aggregation
- Prevents Proliferation of vascular smooth muscle cells (in vivo)


COXIBs/NSAIDs & HEART DISEASE

A) Blood Pressure
B) Fluid Retention
C) Oxidative Modification of Biologic Lipids (possible) leading to atherosclerosis
D) Exacerbation of CHF (esp. with Renal Fx.)


Cox-2 Cardiovascular Effect

HYPOTHESIS
Inhibition of vascular PGI₂ (Prostacyclin) synthesis
And
Lack of Effect on Platelet Thromboxane Synthesis
↓ Imbalance
↓ Prothrombotic State
↓ Increased Thromboembolic CV Events
Is CV Disease a COX-2 Class Effect?

* Events more likely with higher dosage and longer duration of therapy.
* Inconclusive evidence of class effect.

**Recommendation:** Utilize a Risk/Benefit analysis of each patient.
N.B. Patient with Inflammatory disease at higher risk for MI

Inhibition Of Clinical Benefits Of Aspirin On First Myocardial Infarction By NSAIDs


**Findings:**
- ASA 325 mg q other day; rate of 1st MI by ASA
  - NSAID use for 1-5y = no change
  - NSAID use for >60 days/y = ↑ risk MI

**Conclusions:**
Possible that differences in inhibition of ASA’s effect on platelets may lead to differences in clinical outcomes (not yet proven)

* Discrepancy & Redefine had no effect

**Recommendation:** Take ASA at least 2 Hours prior to the NSAID

LONG TERM USE OF NSAIDS And The Risk of Myocardial Infarction In The General Population

Garcia Rodriguez, JA and González-Pere, A. JNC Medicine 2008, 3:17

- Nested case-control analysis of 697 cases of acute MI and 796 control subjects (N=48 V.A.)
- NSAIDs use (ibuprofen, diclofenac, Naproxen) for >1 year did not ↑ risk for MI
- NSAID use for >1 year ↓ risk for nonfatal MI
- Discrepancy reached statistical significance for small risk
  - Ibuprofen may have reduced risk
    - Ibuprofen revealed unremarkable risk in statistical analysis
  - The use of ASA counteracted the risk for MI
RISK OF MI WITH PROLONGED USE OF TRADITIONAL NSAIDs

- Numerous trials with variable results
- ASA appears to obviate risk with NSAIDs
- Interaction between Ibuprofen (and others) and ASA not substantiated
- NSAIDs alone do not appear to offer Cardioprotection

Summary & Recommendations:
1) Take ASA 2 hours prior to NSAID
2) Must take ASA + NSAID if the risk for CAD

Recommendations (Considerations) for the use of NSAID/Cox-2 in 2006

- Careful consideration to the patient needs (indication) for an NSAID or Cox-2 (Avoid with CAD)
- Prescribe NSAID/Cox-2 in those with the lowest cardiovascular (CV) and/or GI risk if possible
  (Monitor patients in discretion for Decision Making)
- Consider alternative therapies (biodosage, injections, topical) if the risk is determined to be too great
- Utilize the lowest effective dose for the shortest period of time
- Starting therapy, Monitor patients for signs of developing toxicity

Andrew K. Bevan in International Journal of Advances in Rheum. Vol. 11 No. 1, 2006
MONITORING NSAID THERAPY

Initially:  CBC  UA  K+  SGOT  Creatinine
        { Q 1-3 Months
Stable:    Same Lab Q 3-12 months

From: Guidelines for Reviewers of Rheumatic Disease Care ACR (CORC)