Rheuma Facts

A Quarterly Magazine

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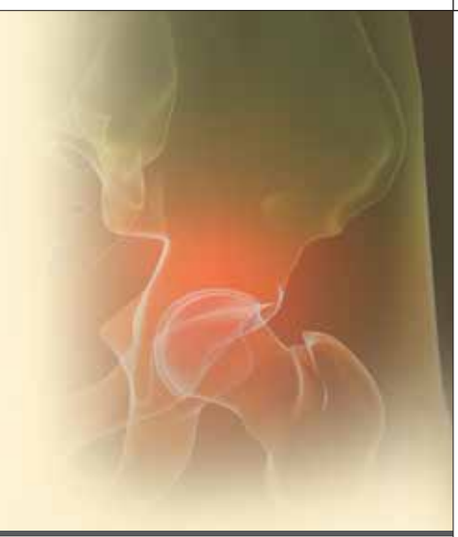

Current News

Heart Attack Risk Rises In First Month After Knee, Hip Arthroplasty

By: BIANCA NOGRADY, Rheumatology News Digital Network
AUGUST 31, 2015

FROM ARTHRITIS & RHEUMATOLOGY

Total knee and hip arthroplasty were associated with a significantly increased risk of myocardial infarction in the first month after surgery, but not at 6 months after surgery, a cohort study showed.

Analysis of data from 13,849 British patients who underwent a total knee arthroplasty, 6,063 patients who received total hip arthroplasty, and an equal number of matched controls showed a greater than eightfold increase in the risk of myocardial infarction in the first postoperative month in the knee arthroplasty group (hazard ratio, 8.75), and a fourfold increase in risk in the hip arthroplasty group (HR, 4.33), compared with controls.

The study observed 306 cases of myocardial infarction among individuals who underwent total knee arthroplasty and 128 cases in those who underwent total hip arthroplasty (Arthritis Rheumatol. 2015; August 31 [doi:10.1002/art.39246]).

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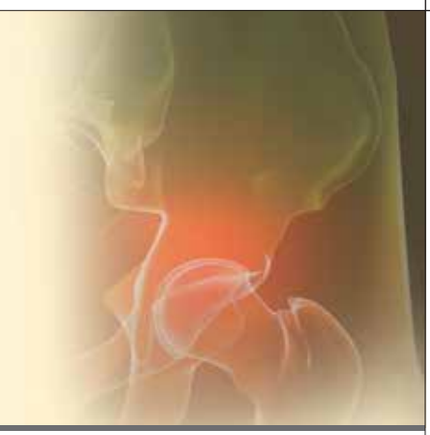

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Multiple Fractures in a 15-Year-Old Boy

Summarized by:
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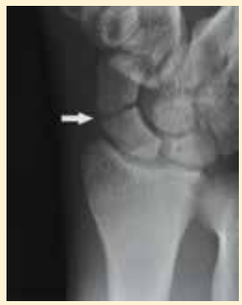
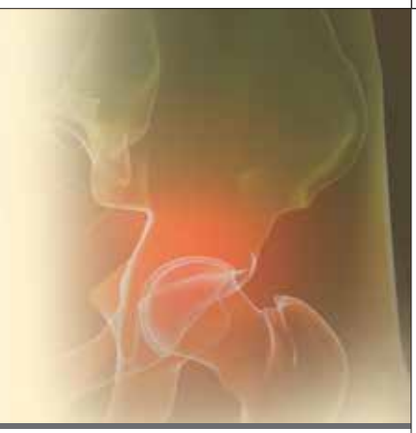




A 15-year-old athletic boy with type 1 diabetes and Hashimoto thyroiditis has had four fractures over the past 2 years. He had two sternum fractures from playing basketball and two separate falls that led to a thumb and radius fracture. He also has had a scaphoid injury. The injuries all had trauma associated with them, although his mother suggests that the injuries were more common in her son than among his team-mates, who also have similar activity levels. The mother also has a question about an outside radiology report that states "osteopenia is noted." He does not have a history of previous injuries or fractures and does not easily bruise.

His type 1 diabetes, which was diagnosed at age 10 years, is poorly controlled. He has positive thyroid peroxidase antibodies but has not developed acquired hypothyroidism yet. Medications include insulin, antacids, and vitamin D at 1000 international units (IU) daily. His vision is checked as part of his annual examinations for diabetes and is unremarkable. His family history includes a mother with Hashimoto thyroiditis and a grandmother with osteoporosis diagnosed in her 60s. No family history of genetic disorders or other autoimmune diseases is noted.

A review of systems is negative for hearing abnormalities, diarrhea, or developmental delay. He denies increased joint flexibility or skin laxity.

Physical Examination and Workup

His weight is 49.7 kg. His body mass index (BMI) is 18.98 kg/m^2.

He is a thin, average-height male with an unremarkable examination. His sclera is white. His dentition is normal without caries. No scoliosis is present. His extremities are thin but with appropriate muscle tone. He has no bowing and no birthmarks.

His laboratory results are as follows:

- Intact parathyroid hormone (PTH) level: 29 pg/mL (reference range, 10-65 pg/mL)
- Calcium PTH level: 9.8 mg/dL (reference range, 8.6-10.3 mg/dL)
- Phosphorus level: 3 mg/dL (reference range, 2.7-4.6 mg/dL)
- Alkaline phosphatase level: 370 IU/L (reference range, 90-420 IU/L)
- Thyroid-stimulating hormone (TSH) level: 2.43 mIU/L (reference range, 0.3-5.5 mIU/L)
- 25-hydroxy vitamin D level: 20 ng/mL (reference range, 25-100 ng/mL)

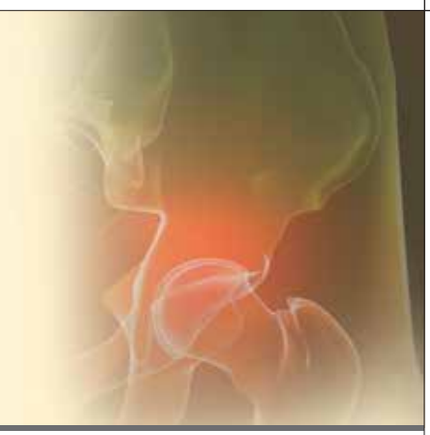
Dual-energy x-ray absorptiometry (DEXA) scanning reveals a left femoral neck Z-score of -0.8, a left hip Z-score of -1, and a lumbar spine Z-score of -1.8.

On the basis of the history, physical examination and work up differential diagnosis should be:

1.Paget's disease
2.Vitamin D deficiency
3.Osteogenesis imperfecta
4.Hypophosphatemic rickets

Discussion

This patient, who has uncontrolled type 1 diabetes, has had multiple fractures in the setting of vitamin D deficiency. DEXA scanning did not demonstrate low bone mineral density; his Z-scores were all above -2. His PTH, calcium, and TSH levels were within reference ranges. Although he could have a complication of an autoimmune disorder, his 25-hydroxy vitamin D levels are lower than the reference range and must be corrected before any further evaluation is undertaken.



Diabetic osteopathy can alter the structure of the bone over time, leading to weaker bones that may easily fracture. However, this patient has only had diabetes for the past 4 years and is not to be expected to have frank diabetic complications yet.

The patient does not have many of the physical findings associated with osteogenesis imperfecta. Individuals affected with type 1 osteogenesis imperfecta are usually of normal height and have few bone deformities. This disorder usually becomes evident around the time children start to walk and improves after puberty. However, they usually have blue sclera, and about 50% of individuals experience hearing loss. Varying degrees of dentinogenesis imperfecta and hearing loss can be present.

This patient also did not demonstrate the phenotype of a child with hypophosphatemic rickets. Not only does he not have the bowing of legs, short stature, or abnormal dentition, his serum phosphorus level is high-normal and not low. He is also not a likely candidate for Paget's disease, as his alkaline phosphatase level was within the reference range and not elevated, as is typically the case in Paget's disease

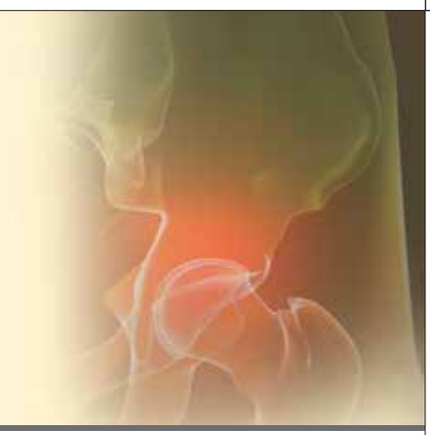
This patient's type 1 diabetes may contribute to low bone mineral density, which can increase his risk for fractures and also affect his ability to absorb vitamin D from his diet. However, his low vitamin D level is needed to be addressed first. Many factors lead to vitamin D deficiency, including a change in diet (consuming less vitamin D-fortified milk), reduced time outdoors and sun protection. Medications such as anti-seizure drugs and glucocorticoids can affect the metabolism of vitamin D, leading to increased requirements. Other disorders that can affect absorption of vitamin D, such as Cystic fibrosis and Crohn's disease, can also result in rickets due to vitamin D deficiency.

The exact value that determines whether a patient is vitamin D sufficient is highly debated; most experts believe that a 25-hydroxy vitamin D level greater than 30-32 ng/mL excludes clinically significant deficiency. PTH levels rise when 25-hydroxy vitamin D levels are below 20 ng/mL and stabilize when the level is more than 30 ng/mL. Secondary hyperparathyroidism contributes to decreased bone mineral density by releasing calcium from the skeleton in order to maintain normal serum calcium levels. Phosphorus is also released and excreted in urine, which may result in low or low-normal serum phosphorus levels. With the change in the calcium-phosphorus balance, mineralization is affected, which changes the architecture of the bones. This eventually may lead to small areas in the skeleton that are more fragile and at risk for fractures.

The Endocrine Society Clinical Practice Guidelines for evaluation, treatment, and prevention of vitamin D deficiency were released in 2011 and gave recommendations for the amount of vitamin D per age group.

Guidelines for Vitamin D Intake

Age (years)	Recommendations	For Those at Risk for Vitamin D Deficiency	Upper Limit Recommended
0-1	400 IU	-	1000 IU
1-18	600 IU	600 - 1,000 IU	4,000 - 10,000 IU
19-50	600 IU	1,500 - 2,000 IU	10,000 IU



Upper limits for the amount of vitamin D were also given by age to reduce the chance of achieving vitamin D intoxication, which is characterized by a 25-hydroxy vitamin D level of more than 150 ng/mL, which may lead to hypercalcemia and hyperphosphatemia.

Vitamin D levels should be checked in those at risk for vitamin D deficiency. Individuals who should be screened include:

- Children on chronic glucocorticoids, antiepileptic drugs, and antifungal medications.
- Those with chronic diseases that lead to malabsorption.
- Darker-skinned infants who live in higher-latitude areas with a lack of sunshine.
- Those with signs/symptoms of poor growth or irritability that could be due to an electrolyte abnormality.
- Those with frequent fractures.
- Women who are pregnant/lactating.
- Those with elevated alkaline phosphatase level for age.

The vitamin D levels for the patient in this case were corrected. He continues to follow up with his primary care physician and consulting endocrinologist to control his type 1 diabetes and Hashimoto thyroiditis.

The Endocrine Society Clinical Practice Guidelines recommend that individuals aged 1-18 years start with 2000 IU daily for at least 6 weeks or 50,000 IU weekly for at least 6 weeks. Once the goal 25-hydroxy vitamin D level is greater than 30 ng/mL, the individual can then be continued on 600-1000 IU daily as maintenance. In those with malabsorption, with obesity, or who are taking medications that interfere with vitamin D absorption, higher doses may be needed to accomplish this goal to replete and maintain the 25-hydroxy vitamin D level above 30 ng/mL. Calcium levels may increase with the improvement in vitamin D levels. Sun exposure alone is not enough to maintain appropriate vitamin D levels in teenaged individuals.

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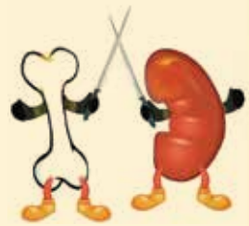
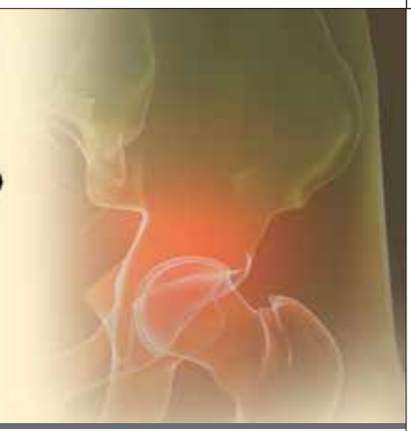
Bone Disease and Calcium Abnormalities in Elderly Patients With Chronic Kidney Disease





Summarized by:
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Chronic Kidney Disease (CKD)-related bone disease is known as renal or uremic osteodystrophy. It is associated with derangements in bone and mineral metabolism that leads to abnormal regulation of calcium, phosphorous, vitamin D, and PTH. It encompasses a spectrum of conditions that are classified based on bone biopsy findings including osteitis fibrosa (high turnover disease), mixed uremic osteodystrophy, osteomalacia (low turnover disease), and adynamic bone disease.

Osteoporosis and renal osteodystrophy may coexist in elderly patients with CKD, which makes the issue problematic to define. Osteoporosis in CKD is only a part of the constellation of metabolic bone problems. Therefore, its diagnosis and management may differ from general population.

Bones are more severely affected in CKD than that from normal aging. In a patient with renal osteodystrophy, there is the potential for low BMD to coexist with an enormous range of functional abnormalities. These range from high turnover bone lesions in patients with uncontrolled hyperparathyroidism to severely reduced bone remodeling activity in patients with adynamic bone disease. This is in contrast to the non-CKD patient with osteoporosis where bone remodeling is not severely affected.

IMPACT ON QUALITY OF LIFE

Patients with CKD-MBD and osteoporosis are associated with increased risk of fractures and are at a high risk of cardiovascular disease. The overall incidence of hip fractures among dialysis patients is about four-fold higher than that expected for general population. The risk is increased in both men and women. Fractures may limit ambulation, leading to loss of independence and chronic pain, thereby decreasing quality of life. Mortality risk in dialysis patients with hip fracture is twice that of patients without hip fracture. Women who are 65 years of age and older and have moderate renal dysfunction (eGFR 60 ml/min per 1.73 m^2) are also at an increased risk of hip fractures. In addition several risk factors for low BMD have been identified in the CKD population such as renal osteodystrophy, ethnicity, transplant status, and duration of dialysis.

EVALUATION

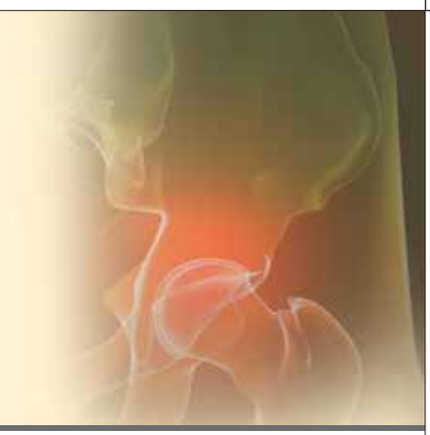
Assessment of bone strength in an elderly patient with CKD is complex. The initial evaluation of CKD-related bone and mineral disorders should include serum biomarkers and noninvasive imaging.

Bone histomorphometric analysis might be needed in some cases. Bone strength is represented by two main features:

Bone density and Bone quality.

Bone density can be measured using several different radiologic techniques, but bone quality is difficult to assess because it depends on architecture, turnover, and mineralization.

Correlation of BMD with bone histology is poor. Serum Biomarkers Surrogate markers of bone metabolism such as serum intact PTH, calcium (preferably ionized), phosphorus, alkaline phosphatases, and bicarbonate levels should be obtained initially. High intact PTH levels may correlate with high turnover bone disease. The optimal target level for intact PTH in CKD is not known. Noninvasive Imaging several imaging tools are available to assess bone health including DEXA scan,



quantitative computed tomography (qCT), and heel ultrasound. The value of BMD in evaluation of CKD related bone disease is not well established. DEXA scan is most commonly used to assess bone mass. It can only detect overall density but not quality of the bone. According to WHO definition, a T-score of less than -2.5 is defined as osteoporosis. The distal radius may be the preferred site in CKD patients.

QCT is more expensive than DEXA and results in greater exposure to radiation. Bone Biopsy remains the gold standard for diagnosis of renal osteodystrophy and assessment of bone architecture. The most accurate diagnostic test for determining the type of bone disease associated with CKD is iliac crest bone biopsy with double tetracycline labeling and bone histomorphometric analysis.

Bone biopsy is not performed routinely in clinical practice because it is invasive, available in limited centers, and requires special expertise and an experienced pathologist for interpretation. However, it should be considered in patients with Stage 5 CKD who have:

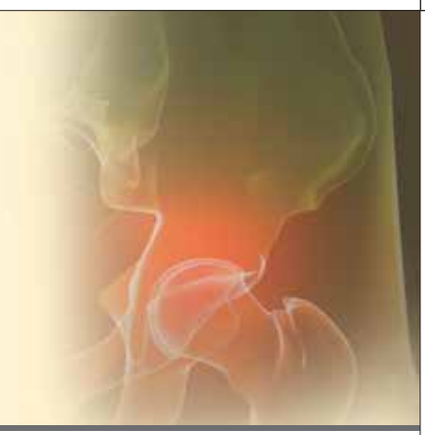
1. Fractures with minimal or no trauma (pathologic fractures).
2. Intact plasma PTH levels between 100 and 500 pg/ml (11.0 to 55.0 pmol/L; in CKD Stage 5) with coexisting conditions such as unexplained hypercalcemia, severe bone pain, or unexplained increases in bone alkaline phosphatase activity.
3. Suspected aluminum bone disease, based on clinical symptoms or history of aluminum exposure.

Bone biopsy should be strongly considered before starting bisphosphonate therapy in patients with Stage 5 CKD.

TREATMENT

Whether the standard agents used for osteoporosis in general population can be applied to patients with CKD is unclear. The management of osteoporosis includes addressing modifiable risk factors and using pharmacologic agents.

1. Smoking cessation is strongly recommended. Excess alcohol consumption should be avoided.
2. Exercise: Moderate weight-bearing physical activity has been associated with improvement in bone density and reduction in risk of hip fractures. However, in older women, the benefit is modest.
3. Calcium and vitamin D requirements: Vitamin D deficiency is common in older adults especially during winter. It may be related to reduced synthesis, inadequate intake, dietary restrictions, and inactive lifestyle in dialysis patients. 25(OH) Vitamin D deficiency is associated with increased iPTH levels, reduced BMD, and increased rate of hip fractures. The extra renal effects of vitamin D are also receiving increasing attention. Low 25(OH) vitamin D levels may be associated with increased risk of cardiovascular events in patients with peritoneal dialysis. Calcium and vitamin D supplementation has been reported to result in small improvement in hip bone density. It did not significantly reduce hip fractures but increased the risk of kidney stones. In CKD Stages 3 and 4, KDOQI recommends maintaining 25(OH) vitamin D



levels above 30 ng/ml by supplementation with ergocalciferol. In CKD Stages 3 to 5, total elemental calcium intake including dietary and calcium based binders should not exceed 2000 mg/d. The National Osteoporosis Foundation recommends a daily calcium intake of 1200 mg and vitamin D intake of 800 to 1000 IU/d for adults 50 and older.

❹ Role of Bisphosphonates: Bisphosphonates are effective in treating osteoporosis, but their use in CKD Stages 4 and 5 is controversial.
No data are available that proves that bisphosphonates reduce risk of fractures in dialysis patients with osteoporosis. Use of bisphosphonates in CKD has been linked to nephrotic syndrome, acute renal failure, and progressive renal disease. It is imperative to make a correct diagnosis before treatment is initiated because bisphosphonates are not indicated in adynamic or osteomalacic bone disease. Bone biopsy is recommended before using bisphosphonates. Alterations in calcium, phosphorous, vitamin D deficiency, and hyperparathyroidism should be addressed before starting bisphosphonates.
Bisphosphonates have been used in the setting of renal transplant to prevent bone loss in the post-transplant period.
In small studies, alendronate, risedronate, clodronate, and ibandronate have been shown to be safe to use in CKD. Some authors recommend using bisphosphonates for short period of time (2 years to 3 years) in the CKD population, although there is no evidence that it will result in reduction of fractures.

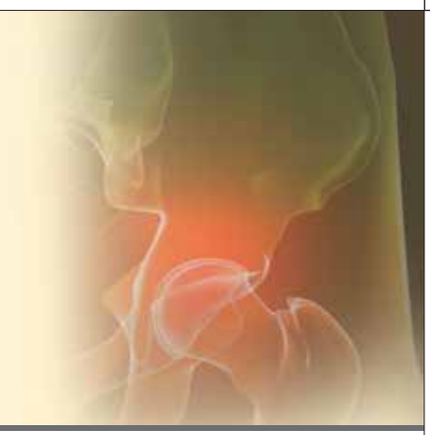
❺ Calcitonin: Calcitonin binds to osteoclasts and inhibits bone resorption. It has a low side effect profile and can be given intranasally. It could help protect bone mass when used along with calcium and vitamin D supplementation, especially in the post-transplant population.

❻ Estrogen/progestin therapy has fallen out of favor because of increased risk of breast cancer, stroke, and thromboembolism. It may be an option in women who are not able to tolerate other forms of treatment.

❼ Selective Estrogen Receptor Modulators (SERM): In a subgroup analysis, raloxifene was associated with a greater increase in spine BMD, a reduction in vertebral fractures, and no effect on nonvertebral fractures compared with placebo. It was safe to use in women with osteoporosis and mild to moderate CKD over the 2 years to 3 years observation period. Some SERMs may increase the risk of deep venous thrombosis (DVT) and pulmonary embolism. Therefore, these agents should be avoided in women with active or history of DVT.

❽ Cinacalcet: It is a calcimimetic that increases the sensitivity of calcium-sensing receptors in the parathyroid gland to calcium, thereby playing a role in regulation of PTH levels. It helps in improving bone histology, reducing bone turnover, and reducing fibrosis in patients with secondary hyperparathyroidism.

❾ Anabolic agents: A new class of anti-osteoporosis drugs are now available that stimulate bone formation. A subgroup analysis of patients with mild to moderate CKD included in the fracture prevention trial showed that teriparatide significantly increased lumbar spine and femoral neck BMD. Adverse effects included hypercalcemia and increased uric acid levels.



CONCLUSION

Age-related bone loss is an important part of assessment in the growing aging dialysis and CKD population. Osteoporosis and renal osteodystrophy may coexist in the elderly CKD population, making diagnosis and management complicated. The DEXA scan is most commonly used to diagnose osteoporosis. Management of osteoporosis includes addressing modifiable risk factors and pharmacologic approaches. Patients with CKD are at greater risk for osteoporosis than general population. Osteoporotic fractures have significant morbidity and negative impact on quality of life. More data is needed to address the optimal management of mineral and bone disorders in the elderly.

TAKE HOME POINTS

- Bone disease in elderly persons with CKD is complicated by co-existence of renal osteodystrophy and osteoporosis.
- Patients with CKD-MBD and osteoporosis are at increased risk of fractures and higher risk of cardiovascular disease.
- Hip fractures in dialysis patients is associated with doubling of mortality.
- Bone biopsy is the gold standard for diagnosis of renal osteodystrophy and assessment of bone architecture.
- Although bisphosphonates are increasingly being used in the post renal transplant setting for prevention of bone loss, there is no evidence that it results in reduction of incidence of fractures.

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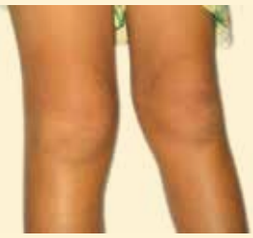
Reactive Arthritis



Summarized by:
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Reactive Arthritis is a form of arthritis that can cause inflammation and pain in the joints, the skin, the eyes, the bladder, the genitals and the mucus membranes. Reactive arthritis is thought to occur as a "reaction" to an infection that started elsewhere in the body, generally in the genitourinary or gastrointestinal tract.

Potential Causes of Reactive Arthritis

Reactive arthritis occurs after exposure / infection caused by certain types of bacteria. These include:

- Chlamydia, a bacterium contracted during sexually activity, which causes either burning urination or watery discharge from the penis or vagina.
- Bacteria such as Salmonella, Shigella, Yersinia or Campylobacter, which cause dysentery (diarrhea, abdominal pain, vomiting, fever). Exposure to these bacteria occurs after eating spoiled or contaminated food.

However, not everyone exposed to these bacteria will contract ReA. Those who go on to develop ReA tend to test positive for the HLA-B27 genetic marker, although other genetic factors may be involved. Thus, it is an interaction between an individual's genetic make-up and the initial infection that causes Reactive Arthritis.

Disease Course / Prognosis

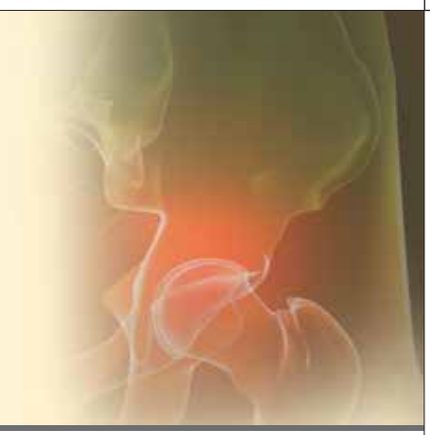
ReA usually develops 2-4 weeks after the infection. A tendency exists for more severe and long-term disease in patients who do test positive for HLA-B27 as well as those who have a family history of the disease.
Reactive Arthritis typically follows a limited course, where symptoms subsiding in 6 to 8 weeks. However, the condition has a tendency to recur. About 15-20% of people with ReA develop a chronic, and sometimes severe, arthritis or spondylitis.

Symptoms List

- Pain, swelling and stiffness in the joints including the knees, ankles and feet. Most commonly one or two large joints are involved.
- Fever and chills.
- Inflammation of the eye (conjunctivitis, uveitis or iritis) that can cause redness, pain, sensitivity to light and skewed vision.
- Enthesopathy - inflammation where the tendon attaches to the bone.
- Cystitis, which is an inflammation of the bladder or urinary tract, causing frequent urination and a burning sensation when urinating.
- Genital sores appearing on the shaft of the penis or scrotum, or in women, on the external areas of the genitals. These are usually blisters that break open and crust over. Although they heal without scarring, these blisters can be a source of great anxiety in those with ReA.

Examination

A patient's complete medical history, noting current symptoms as well as any previous diseases, problems and infections should be noted.



Tests may be done like CBS, ESR, Urine D/R and testing for the presence of Chlamydia, cells may be taken from the throat as well as urethra or cervix, a stool sample may be taken, and synovial fluid, may be removed from the affected joints for study.

Medication

NSAIDs (Nonsteroidal Anti-Inflammatory Drugs) are still the cornerstone of treatment and the first stage of medication in treating the pain and stiffness associated with ReA. When NSAIDs are not enough to control inflammation and pain, a short course of steroids can be given.

In short ReA is very easy to diagnose after taking thorough medical history and doing careful joint examination. A short history (acute) of mono or oligoarthritis with preceding history of gram negative infection in GIT or Urino genital system confirms diagnosis of Reactive arthritis. As the condition is self-remitting only 20% chance to develop into chronic Arthritis.

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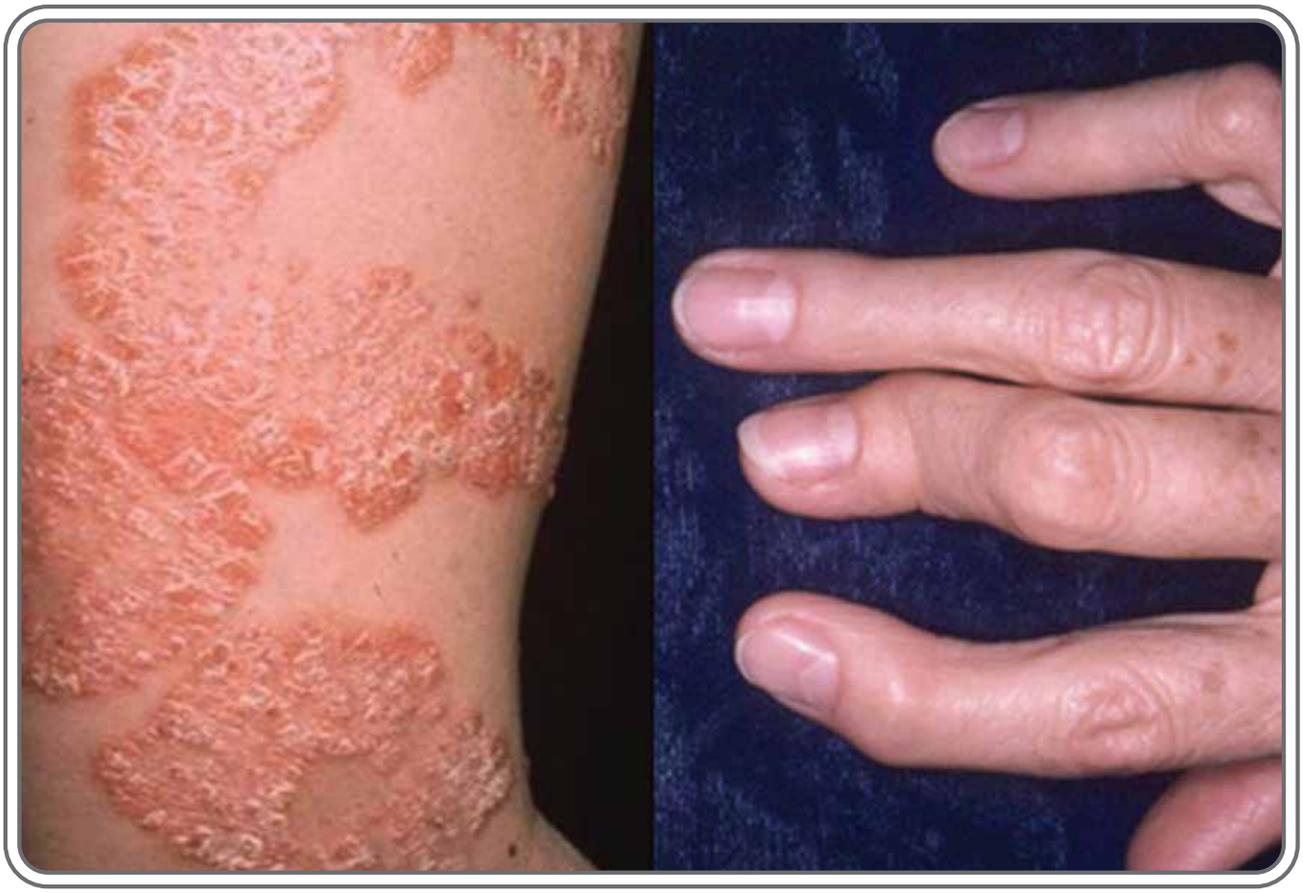
Photo Quiz



Question

What is the single diagnosis for the above given pictures?

Answer of last quiz

- Ankylosing spondylitis