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Chronic myeloid leukemia and ankylosing spondylitis

Hemopathies could be associated with different autoimmune diseases especially rheumatoid arthritis and Sjögren syndrome. However, the association with ankylosing spondylitis (AS) had been rarely reported. The mechanism of this association remains unknown. It may reflect a mere coincidence or a common pathogenesis.
We report the original observation of concurrence of chronic myeloid leukemia (CML) and primitive AS.

Case report
Mr HM, 47 years old, was followed in our department for AS. This diagnosis was selected because of rocking, inflammatory sacroiliac joint pain, chronic inflammatory neck pain evolving since the age of 20. It was associated to bilateral sacroiliitis stage II on plain radiography. HLA typing showed a phenotype B27. The blood count (CBC) was unremarkable. The diagnosis of secondary spondylarthropathy was excluded since there was nor cutaneous, neither urethral signs and all digestive explorations were negative (colonoscopy with repeated biopsies). His rheumatic disease remained active despite non steroidal anti-inflammatory (NSAID) drug, which lead as to prescribe TNF alpha antagonist therapy.
Unfortunately, the skin test (TST) to tuberculin was strongly positive. An exhaustive search of active tuberculosis was conducted. The search for Mycobacterium tuberculosis in sputum and urine and QuantiFERON test were negative. The thoraco-abdominal, pelvic and cervical computed tomography (CT) scan had not identified any lymphadenopathy or other lesions. Since all these exams were negative, the patient had received anti-tuberculosis chemoprophylaxis and anti-TNF therapy would be started within three weeks. Unfortunately, before starting this treatment, a new CBC revealed leukocytosis at 39,000 cells/mm3 predominantly neutrophil count, which had increased 15 days later at 51,000 cells / mm 3.
An abdominal ultrasound had highlighted splenomegaly that was not seen on the abdominal CT scan. The myelogram showed a smear with a lot of megakaryocytes. The formula was as follows: myeloblasts: 2%, promyelocytes 16%, myelocytes 25%, metamyelocytes 33%, neutrophils 4%, neutrophils 8%, lymphocytes 1%, monocytes 1%, erythrocytes: 10%.
Genetic studies by molecular biology had revealed a pathologic clone with a Philadelphia chromosome resulting from the classic translocation between chromosome 9 and chromosome 22, in favor of CML. Thus, TNF alpha inhibitors were formally prohibited. NSAIDs were continued in combination with physical rehabilitation. For his blood disorders, the patient received imatinib (Glivec ®). The assessment of the response is actually ongoing.

Conclusion
This original case report illustrates that CML may be associated with authentic AS. The entanglement of these two entities suggests common genetic mechanisms to these conditions and in particular the major complex of histocompatibility. The occurrence of hemopathy in the course of AS limits the therapeutic options for AS and especially the anti-TNF ones.

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Une cause rare d'infection urinaire chez le nourrisson

Le kyste de la vésicule séminale est une entité rare rencontrée dans 0,005% de la population. La plupart des kystes des vésicules séminales sont congénitaux associés à des malformations de l'appareil urinaire à type de dysplasie rénale multi kystique et d'uretère ectopique.
L'association fréquente entre dysplasie rénale et anomalies urogénitales s'explique par l'interdépendance embryonnaire entre l'ébauche du rein, des uretères et de l'appareil génital.

Observation
Il s'agit d'un nourrisson de sexe masculin âgé de 5 mois né à terme d'une grossesse bien suivie, aux antécédents d'un épisode d'infection urinaire à l'âge de 20 jours pour lequel il n'a pas été exploré. Il a été hospitalisé pour une fièvre mal tolérée associée à un syndrome inflammatoire biologique et à une hyperleucocytose à la numération formule sanguine. L'ECBU était en faveur d'une infection urinaire. Une échographie abdomino-pelvienne (figure 1) a été réalisée objectivant une masse kystique bien limitée à paroi propre inter vésico-rectale à contenu finement échogène associée à un aspect hyper échogène de la graisse et à de multiples adénopathies mésentériques. Par ailleurs, elle a mis en évidence un rein gauche dysplasique de petite taille. Le diagnostic de duplication digestive rectale a été d'abord évoqué sur les constatations échographiques. Un scanner abdomino-pelvien (figures 2a, b, c) a donc été réalisé montrant un rein gauche dysplasique diminué de taille avec un uretère homolatéral dilaté tortueux communiquant avec une formation globalement arrondie inter-