Hyperuricemic Syndromes: Pathophysiology and Therapy
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It is our pleasure to introduce the volume ‘Hyperuricemic Syndromes: Pathophysiology and Therapy’ as a joined editorial effort between the Department of Nephrology and that of Onco-hematology of the San Bortolo Hospital, Vicenza, Italy.

We consider the uric acid disorders as a common field between nephrological and hematological diseases and for this reason we feel that a multidisciplinary approach can probably offer new insights into these metabolic syndromes.

Gout has been recognized since antiquity, and descriptions of the disease date back to the Babylonian empire. Hippocrates described gout well as podagra (literally ‘foot into a trap’), and some of his classical aphorism withstood the test of time: ‘A woman does not take the gout until her menses be stopped’ and ‘A young man does not take the gout until the first sexual intercourse’.

Gout has been the sign of distinction and was called the ‘patrician malady’, ‘king of diseases’ and the ‘disease of kings’. In fact gout afflicted kings, including Alexander the Great, Charlemagne, and Henry VIII, and famous personalities like Voltaire, Isaac Newton, Charles Darwin, and Leonardo da Vinci (fig. 1: an old disease depicted in an old painting).

Anton Van Leeuwenhoek, the Dutch inventor of the microscope, observed crystals from a gouty tophus in 1679. A classic description of gouty acute attack is reported in 1683 by Thomas Sydenham, from his own gouty suffering. Sir Alfred Garrod stated the concept that gouty arthritis follows a deposition of urate from supersaturated body in 1859.

The modern history of crystal-associated disease dates from more than 50 years, when McCarthy and Hollander identified urate crystals in joint effusion from patients with gout.
Fig. 1. William Hogarth: The marriage settlement. (National Gallery, London)
The first enzymatic defect responsible for one rare subtype of hereditary gout, hypoxanthine-guanine phosphoribosyltransferase deficiency, was discovered by Seegmiller, Rosenbloom, and Kelley in 1967. Allopurinol, in 1963, opened the way to the successful clinical management of gout.

In the more recent history, the hyperuricemic syndromes affecting patients with cancer, especially in the phase of cellular destruction after chemotherapy have been identified as Tumor Lysis Syndromes. These pathological conditions, together with the improved understanding of urate handling by the kidney have spurred new interest in the pathophysiology of hyperuricemic states, their clinical consequences and their management.

The recent development of a recombinant form of urate oxidase transforming uric acid into allantoin (Rasburicase) has brought new interest into the pathophysiological mechanisms of hyperuricemia and on the potential applications of the new drug.

This volume has been made possible by Sanofi-Synthélabo thanks to an important organizational effort. We were able to put together a group of internationally recognized experts in the field and put them working together for the preparation of a book that is intended to be a compendium of the present knowledge in the field and at the same time a reference tool for professionals and students who want to expand their knowledge in this topic. The book is multidisciplinary and it takes advantage of the competence and the specific professionalism of each author in the fields of biochemistry, pharmacology, rheumatology, onco-hematology, and nephrology. As we used to say for any multidisciplinary project, we cannot all play the same instrument but we can all be on the same key.

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