Full Pattern of Urinary Prostaglandins in Bartter’s Syndrome

L. Calò
S. Cantaro
A. Piccoli
S. Favaro
L. Bonfante
A. Borsatti

First Division of Nephrology, Department of Internal Medicine, University of Padua, Italy

Sir, males and 5 females, age range 19–56 years) and in 9 healthy controls cross-matched for sex and age. After Since Gill et al.’s [1] original description of increased urine extraction with organic solvents and silica gel col-urinary excretion of prostaglandin E\(_2\) (PGE\(_2\)) in Bartter’s syndrome, many controversies have arisen about the role as previously described [4], and 6-keto-PGF\(_\alpha\) and TxB\(_2\) played by prostaglandins in this disease, and it is still by the use of commercially available kits (Amersham, Amersham, UK). As far as the mean urinary excretion is concerned, in involved. Bartter’s syndrome we found a significant increase in different prostanoids, in fact, sometimes exert oppo-

PGE\(_2\) (627.5 ± 275.6 vs. 359.6 ± 126.6 ng/day; t = 2.62, p < 0.02) and 6-keto-PGF\(_\alpha\) (394.8 ± 190.1 vs. 194.7 ± 40.3 ng/day; t = 3.09, p < 0.01), a significant decrease in TxB\(_2\) (123.2 ± 57.3 vs. 184.1 ± 43.9 ng/day; t = 2.47, p < 0.05) in different ways [2]. Therefore, it should be important to know the full urinary pattern of the main arachidonic acid metabolites in patients affected by Bartter’s syndrome to clarify the role played by prostanoids in this syndrome, but it has never been done. We have addressed this issue trying to answer two complicated questions: which prostaglandin of the cyclooxygenase pathway is more frequently involved in Bartter’s syndrome, and which prostanoid is more frequently involved. In fact, PGE\(_2\) values were above the upper main questions: which prostaglandin of the cyclooxygenase pathway is more frequently involved in Bartter’s syndrome, and which prostanoid is more frequently involved. In fact, PGE\(_2\) values were above the upper

main questions: which prostaglandin of the cyclooxygenase pathway is more frequently involved in Bartter’s syndrome, and which prostanoid is more frequently involved.
syndrome and whether a derangement in prostaglandin synthesis is a hallmark of the disease. To this end we have evaluated the urinary excretion of PGE2, PGF2α, 6-keto-PGF1α, the main metabolite of PG12, and thromboxane B2 (TxB2), the main metabolite of TxA2, in 8 patients affected by Bartter’s syndrome. According to these data, we conclude that it is impossible to distinguish between healthy controls and patients affected by Bartter’s syndrome in terms of a single prostaglandin excretion. However, since the trend of the mean values pointed toward an increased production of vasodilatory prostaglandins together with a reduction of TxB2, we have also considered the ratio PGE2 × 6-keto-PGF1α/TxB2. This approach seems to improve the discrimination between patients and controls since the ratio fell above the upper limit (95%) of the controls in 7 patients, but it was still in the normal range in 1 patient (fig. 1).

However, by the use of multivariate discriminant analysis [5], taking into account all 4 prostanoids it was possible to fully discriminate between patients and controls. Moreover, the symmetry of both the weight and sign of the standardized coefficients of the discriminant
function, namely 1.27 PGE₂, 0.70 6-keto-PGF₁α, -1.24 PGF₂α and -0.52 TxB₂, supports the conclusion that a derangement in prostaglandin synthesis is really a constant feature of Bartter’s syndrome, the prevailing picture being that of a rearrangement in prostaglandin production with a shunting toward the vasodilatory ones.


References