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 co-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, debated whether prostaglandin overproduction is a con- stant feature and which prostanoid is more frequently involved. Bartter’s syndrome we found a significant increase in different prostanoids, in fact, sometimes exert oppo- site effects on renal handling of electrolytes, regulation of renin release and renal action of antidiuretic hormone, TxB\(_2\) and can counteract the intrarenal action of angiotensin II (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 consider the cutoff values corresponding to either the one-sided 95th percentile, respectively), the picture becomes more complicated. In fact, PGE\(_2\) values were above the upper limit in 4 patients, no PGF\(_\alpha\) values were above the upper main questions: which prostaglandin of the cyclooxyge- nase pathway is more frequently involved in Bartter’s syndrome, but it has never been done. or 5th percentile, respectively), the picture becomes more complicated. In fact, PGE\(_2\) values were above the upper limit in 4 patients, no PGF\(_\alpha\) values were above the upper
syndrome and whether a derangement in prostaglandin synthesis is in a hallmark of the disease. 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-PGF2α/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 (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 PGF₁α, 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