ovascular damage mechanisms is clearly defined. That is, the higher incidence of cardiovascular complications in patients with PHA, as compared to patients with EH, may be associated with an increased prevalence of metabolic syndrome in patients with PHA (46-48).

The presence of more than three signs of MS in patients with PHA emphasizes that this secondary form of arterial hypertension is not benign by its course. It is strongly associated with metabolic changes, which can cause a high risk of developing cardiovascular complications.

CONCLUSIONS

Thus, the analysis of literature data indicates that PHA is the main endocrine cause of secondary hypertension and is much more frequent than previously considered.

Symptoms of primary hyperaldosteronism are not specific and do not give an impression of it as a disease with clearly expressed clinical manifestations. An early and leading symptom of PHA is an increase in blood pressure. It is common to many diseases that cause primary and secondary arterial hypertension. Obviously, this can be explained by the low level of diagnosis of PHA as causes of arterial hypertension at all levels of medical care.

Certain values in the diagnosis of PHA have non-specific clinical and laboratory characteristics. Among them, it is possible to distinguish, without indicating priority, such as sleep apnea, inadequate response to hypotensive therapy of the combination of three drugs, manifestation of arterial hypertension at the age of 30, rapid increase in blood pressure even in the elderly and/or loss of efficacy of antihypertensive therapy, expressed increase in blood pressure more than 180/110 mm Hg, hypokalemia, especially when combined with increased creatinine plasma concentrations, proteinuria or hematuria.

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