

**Table 2.** Comparison between PHA and FHH.

	<b>Renal PHA</b>	<b>Systemic PHA</b>	<b>Secondary PHA</b>	<b>FHH</b>
Type of inheritance	Autosomal dominant	Autosomal recessive	Not inherited	Autosomal dominant
Mutated gene	<i>NR3C2</i> (4q31.1)	a) <i>SCNN1A</i> (12p13.31), b) <i>SCNN1B</i> (16p12.1), c) <i>SCNN1G</i> (16p12.1)	-	a) 1q31-42, b) <i>PRKWINK4</i> (17q21.31), c) <i>PRKWINK1</i> (12p13.33), d) unidentified locus
Mutated protein	MR	ENaC (subunit $\alpha$ , $\beta$ or $\gamma$ )	-	a) Unidentified, b) WNK4, c) WNK1, d) unidentified
Age of onset	Neonatal or early infant period	Neonatal period	Neonatal or early infant period	Childhood
Salt loss/retention	Salt loss (kidneys)	Salt loss (kidneys, colon, sweat glands, salivary glands, manifestations from lungs)	Salt loss (kidneys)	Salt retention (kidneys)
Blood pressure	Hypotension	Hypotension	Hypotension	Hypertension
Renin levels	↑	↑	↑	↓
Aldosterone levels	↑	↑	↑	Variable (usually almost normal)
GFR	Normal	Normal	↓	Normal
Duration of manifestations and treatment	Until early childhood	Life-long	Up to a few days to one week after the initiation of treatment	Life-long
Prognosis	Good	Poor	Good	Good

PHA: pseudohypoaldosteronism; FHH: familial hyperkalemic hypertension; MR: mineralocorticoid receptor; ENaC: epithelial sodium channel; WNK: with-no-lysine (K) kinase; GFR: glomerular filtration rate.