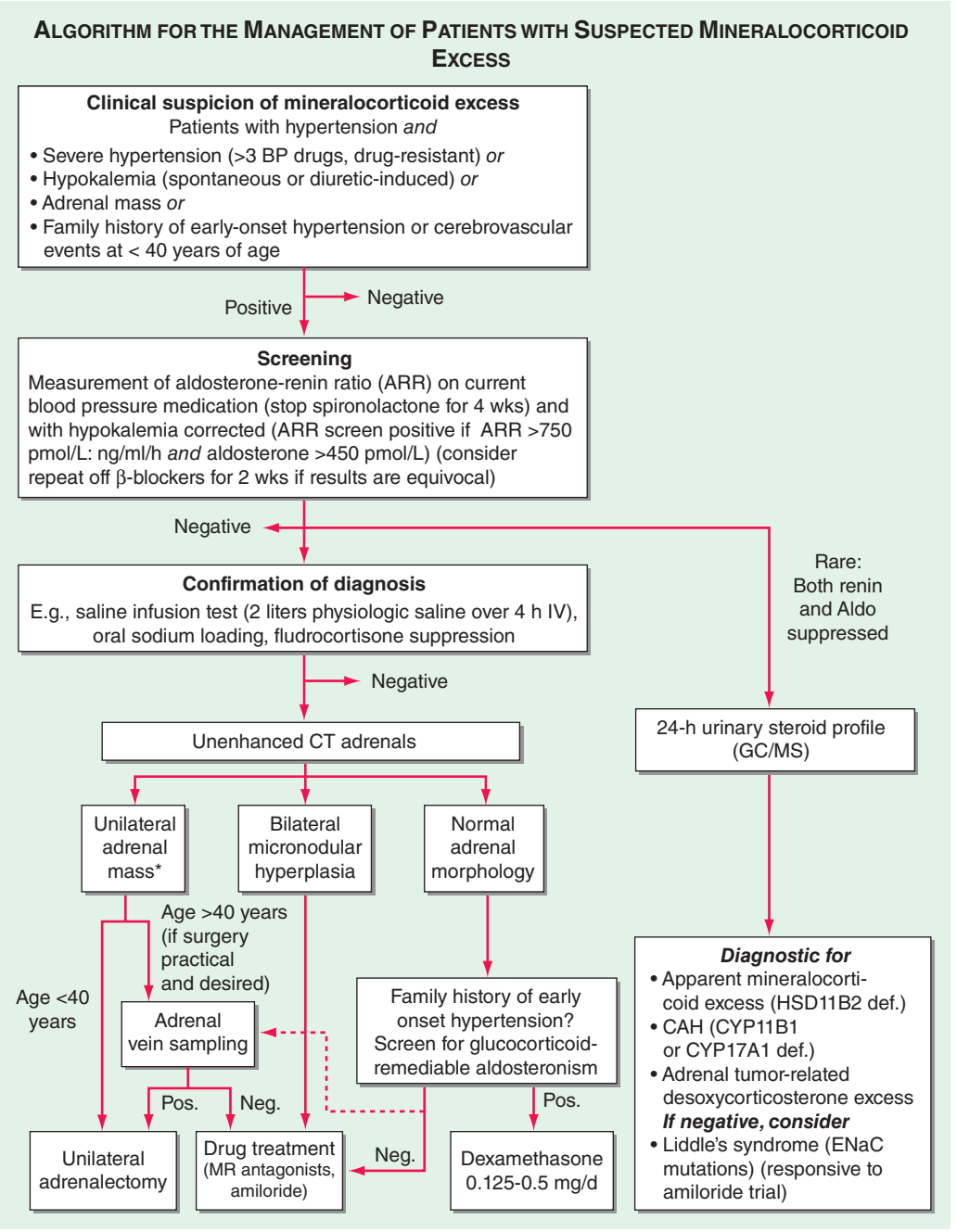

FIGURE 406-12 **Management of patients with suspected mineralocorticoid excess.** *Perform adrenal tumor workup (see Fig. 406-13). BP, blood pressure; CAH, congenital adrenal hyperplasia; CT, computed tomography; GC/MS, gas chromatography/mass spectrometry; PRA, plasma renin activity.

TABLE 406-4 **EFFECTS OF ANTIHYPERTENSIVE DRUGS ON THE ALDOSTERONE-RENIN-RATIO (ARR)**

Drug	Effect on Renin	Effect on Aldosterone	Net Effect on ARR
β Blockers	↓	↑	↑
α_1 Blockers	→	→	→
α_2 Sympathomimetics	→	→	→
ACE inhibitors	↑	↓	↓
AT1R blockers	↑	↓	↓
Calcium antagonists	→	→	→
Diuretics	(↑)	(↑)	→/(↓)

Abbreviations: ACE, angiotensin-converting enzyme; AT1R, angiotensin II receptor type 1.

considered and can be evaluated using genetic testing. Treatment of GRA consists of administering dexamethasone, using the lowest dose possible to control blood pressure. Some patients also require additional MR antagonist treatment.

The diagnosis of nonaldosterone-related mineralocorticoid excess is based on documentation of suppressed renin and suppressed aldosterone in the presence of hypokalemic hypertension. This testing is best carried out by employing urinary steroid metabolite profiling by gas chromatography/mass spectrometry (GC/MS). An increased free cortisol over free cortisone ratio is suggestive of SAME and can be treated with dexamethasone. Steroid profiling by GC/MS also detects the steroids associated with CYP11B1 and CYP17A1 deficiency or the irregular steroid secretion pattern in a DOC-producing adrenocortical carcinoma (Fig. 406-12). If the GC/MS profile is normal, then Liddle's syndrome should be considered. It is very sensitive to amiloride treatment but will not respond to MR antagonist treatment, because the defect is due to a constitutively active ENaC.