Female patient, 37 years old, coming from China, with a history of resistant arterial hypertension since the age of 25 years old associated with spontaneous hypokalemia and classic ACTH-independent Cushing’s syndrome. Radiological imaging exams showed bilateral adrenal nodules. Diagnosed primary hyperaldosteronism (Baseline aldosterone 103ng/dL (2.5-39.2), Renin 2.7uUI/mL (4.4 - 46.1), Aldo/renin ratio 38.1 and K 3.3) and confirmed laboratory hypercortisolism (Cortisol after 1mg of dexamethasone overnight 17.9mcg/dL (<1.8), salivary cortisol at midnight 0.466mcg/dL (<0.274), ACTH <2pg/mL, basal serum cortisol 19.4 (3.7–19.4)). Abdominal CT identified bilateral adrenal nodules, one on the right and two on the left, with <10 HU, suggestive of adenomas. In the pandemic, we proposed the same surgical treatment for primary adrenocortical macronodular hyperplasia (total adrenalectomy of the largest gland and with greater uptake in PET-FDG and subtotal of the smallest and with less uptake). The patient was therefore submitted to total videolaparoscopic adrenalectomy on the left and subtotal on the right. The anatomopathological evaluation showed bilateral adenomas (Weiss 1) being the immunohistochemistry of the right adrenal nodule diffusely positive for CYP11B1 and negative for CYP11B2, inferring only cortisol production. In contrast, left adrenal nodules were positive only for CYP11B2, inferring exclusive aldosterone production. However, we also found bilateral subcapsular cell clusters with positivity for CYP11B2. This may infer a possibility of hyperaldosteronism recurrence. The patient developed secondary adrenal insufficiency postoperatively, requiring replacement with hydrocortisone, and cure of hyperaldosteronism with normalization of potassium and release of renin. Six months after the operation, there was no need for antihypertensive drugs. Currently, it is using only one class of antihypertensive, amlodipine 5mg a day, and it still needs to be replaced with hydrocortisone 20mg on waking up and 10mg at 2 pm with still undetectable serum cortisol concentrations, despite the ACTH being unblocked (17.3 pg/ml). Now we are working on genetic background of this case report.

Presentation: No date and time listed.