A 31 years male presented with diabetes mellitus, hypertension and increasing body pigmentation over one year. He had history of low backache for last 3 months. On examination his body mass index was 23.23kg/m² and had BP of 150/90mmHg. He had diffuse hyperpigmentation, including that of nail bed (Fig. 1a), knuckles and gingival margin. He did not have striae and bruises but had severe proximal muscle weakness. Other systemic examination was essentially normal except lumbar lordosis with local tenderness at L2-3. On investigation, fasting plasma glucose was 127 mg/dl; postprandial plasma glucose, 240 mg/dl; serum potassium 2.9 mmol/L and arterial blood gas analysis showed metabolic alkalosis. Cortisol dynamics revealed: 8 AM cortisol, 1200 nmol/L (N, 400-600); at 10 PM cortisol and paired ACTH were 1200 nmol/L (N,200-400) and 950 pg/ml respectively. Plasma cortisol after overnight, low- and high-dose dexamethasone was non-suppressible. Radiology showed mediastinal widening and compression wedge fracture at L2-3 vertebrae. CECT chest and abdomen showed an anterior mediastinal mass and bilateral adrenal hyperplasia respectively. A diagnosis of ectopic Cushing’s syndrome due to thymic carcinoid with secondary diabetes mellitus and hypertension was made, and he was subjected to median sternotomy with thymectomy. Histopathology was consistent with thymic carcinoid.

At 3 months follow-up, his blood pressure and blood glucose were controlled without medications; hyperpigmentation had started waning. His nail-bed pigmentation had considerably decreased though distal nails were hyperpigmented. At 6 months follow-up, serum cortisol at 8 AM was 600 nmol/L and at 10 PM cortisol and paired ACTH were 300 nmol/L and 10 pg/ml respectively. He continued to be euglycemic and normotensive and hyperpigmentation over nails and nail beds completely disappeared.

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