vels of sFlt-1, immediately after delivery, with values at 888, 667 and 123 pg/mL. Conversely, serum creatinine and uric acid remained unchanged, 30 days after delivery at 141 and 428 μmol/L, respectively.

Comment. This case illustrates the difficulty in differentiating a superimposed pre-eclampsia from an isolated deterioration of a pre-existing renal disease during pregnancy. The increase in serum uric acid can be explained by a superimposed pre-eclampsia or by the impact of pregnancy in worsening pre-existing nephropathy, similar to the onset of hypertension. The high levels of circulating sFlt-1 found before delivery were in the same range as the mean serum level of 4382 pg/mL found by Levine *et al.* in the group of women with pre-eclampsia at a similar gestational age (as compared with 1643 pg/mL, in the control group of women with normal pregnancy) and strongly suggests an excess placental production related with superimposed pre-eclampsia [2,3]. Because of the possibility of false-positive values, histological placenta data would have been better to confirm the diagnosis [4]. Unfortunately, the latter was not preserved. However, these high levels of circulating sFlt-1 cannot account for the concomitant renal dysfunction. Indeed, renal failure is usually associated with a significant, but very moderate, increase in circulating sFlt-1 [5]. Moreover, in the present case, circulating levels of sFlt-1 fall rapidly after delivery despite the persistence of renal failure and hyperuricaemia.

Variations of sFlt-1-circulating levels have been mainly studied among population groups of gestational women. The present case demonstrates that the results of several sFlt-1-circulating assays performed on the same patient can also assist better birth management in difficult situations.

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Pleiotropic effects of vitamin D in an early stage of chronic kidney disease—effect on insulin resistance

Disturbances of carbohydrate metabolism are common in patients with chronic kidney disease (CKD). Possible pathogenetic mechanisms involve unresponsiveness of insulin receptor and/or a bland response of the beta cell at the stimulus of hyperglycaemia. It is known that vitamin D has an important role in the endocrine function of the pancreas, particularly in the insulin release process, and is one of the determinants of insulin resistance [1]. This metabolic complication may be implicated in the accelerated atherosclerotic process and is common in CKD. The aim of our study was to investigate the effect of vitamin D treatment on insulin resistance in patients with CKD stage 3.

We included in our study 37 (25 men/12 women, age 51 ± 13 years old) non-diabetic patients with CKD stage 3 (eGFR 30–59 mL/min/1.73 m^2 calculated with the Modification of Diet in Renal Disease formula). Patients with history of diabetes mellitus and previous therapy with vitamin D were excluded. The underlying kidney disease was obstructive nephropathy, secondary focal segmental glomerulosclerosis, membranous glomerulonephritis and unknown cause of CKD. In all patients, CKD–mineral and bone disorder (CKD–MBD) was established. All patients were treated at the beginning with dietary phosphorous restriction, phosphate-binding agents and calcium supplements. If intact parathyroid hormone (iPTH) values were persistently >70 pg/mL, administration of 1-alpha-hydroxyvitamin D3 in a single daily dose of 0.25 μg was initiated. Fasting glucose concentration, insulin levels, HbA1c, iPTH, Ca, P and insulin resistance evaluated by homeostasis model assessment index [HOMA index, calculated as: fasting glucose (mmol/L) × insulin (mU/mL) / 22.5] were measured before (T0) and 12 weeks after (T1) initiation of treatment. In all patients, an oral glucose tolerance test (OGTT) with an oral dose of 75 g of glucose was performed. No patient was under treatment with corticosteroids or erythropoietin for anaemia.

Plasma levels of fasting glucose and insulin levels were comparable before (T0) and after (T1) treatment with vitamin D (Table 1). The HOMA index was statistically higher in time T0 than T1 indicating that insulin resistance was improved. Mean glucose OGTT levels were higher in T0. There was no statistical difference in calcium, phosphorous, HbA1c, pre- and post-treatment concentration, although the T1 calcium concentration was higher than T0.

Vitamin D insufficiency is correlated with increased risk of all-cause and cardiovascular (CV) mortality in patients

Table 1. Plasma levels of fasting glucose and insulin levels before and after treatment with vitamin D

	T0	T1	P
Fasting glucose (mg/dL)	103 ± 2.3	89 ± 2.4	n.s.
Insulin level (mU/mL)	8.9 ± 1.6	9.8 ± 1.8	n.s.
HOMA index	3.4 ± 0.3	2.58 ± 0.4	0.035
Glucose OGTT 120[a]	153 ± 25	134 ± 29	<0.05
Calcium (mg/dL)	8.2 ± 0.6	8.9 ± 1.3	n.s.
Phosphorous (mg/dL)	4.3 ± 1.4	4.9 ± 1.6	n.s.
HbA1c[b] (%)	6.7 ± 1.1	6.2 ± 1.4	n.s.

[a]Glucose levels 120 min after administration of 75 g of glucose.
[b]Glycated haemoglobin.

with stage 3 and 4 CKD [2]. Studies demonstrate a link between abnormalities in vitamin D metabolism and insulin resistance, a very common condition in patients with CKD. It is proven that insulin resistance is an independent predictor of morbidity and mortality for CV causes in non-diabetic patients with end-stage renal disease [3]. A few works have investigated the effect vitamin D has on insulin resistance in early stages of CKD.

Our study has shown a beneficial effect of vitamin D supplementation in patients with CKD stage 3 on insulin resistance. The HOMA index and OGTT mean glucose plasma levels have significantly decreased after 1-alpha-hydroxyvitamin D3 treatment.

Changes in PTH, a factor implicated in insulin resistance, were not significant during the study period and were not correlated with the amelioration of the HOMA index. Our conclusion was that vitamin D affected insulin metabolism directly and not through reduction of PTH in patients with stage 3 CKD.

Insulin resistance is present in early stages of CKD and is associated with vitamin D metabolism. According to our data, treatment with vitamin D in patients with CKD stage 3 has a beneficial effect on insulin resistance assessed with the HOMA index. Of course, further randomized controlled large-scale studies are needed in order to assess long-term benefits in morbidity and mortality of those patients.

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