Table 1—*Clinical and metabolic characteristics of NIDDM patients with and without microalbuminuria*

	Diabetic patients		
	Normoalbuminuric	Microalbuminuric	*P* value
n	70	69	
Sex (M/F)	38/32	40/29	NS
Age (years)	56 (39–68)	58 (35–67)	NS
Duration of diabetes (years)	8.0 (1–27)	8.0 (1–26)	NS
HbA_{1c} (%)	7.2 (4.6–12.1)	7.1 (4.4–12.4)	NS
Lp(a) (mg/dl)	8.0 (0.3–69)	9.2 (0.1–116.8)	NS
Cholesterol (mg/dl)	219.5 (125–329)	232 (130–347)	NS
Triglycerides (mg/dl)	124.5 (28–739)	184.0 (60–533)	<0.001
High-density lipoprotein cholesterol (mg/dl)	38 (17–99)	36 (18–73)	NS
Low-density lipoprotein cholesterol (mg/dl)	151.7 (59.6–257)	153.4 (76–257)	NS
Apolipoprotein A (mg/dl)	133.5 (71–189)	129 (95.6–222)	NS
Apolipoprotein B (mg/dl)	124.5 (56.8–333)	142 (92.8–240)	0.025

Data are median (range).

patients likely to develop macrovascular complications.

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References

1. Haffner SM: Lipoprotein(a) and diabetes: an update. *Diabetes Care* 16:835–840, 1993
2. Jenkins AJ, Steele JS, Janus ED, Santamaria JD, Best JD: Plasma apolipoprotein A is increased in type II (non-insulin-dependent) diabetic patients with microalbuminuria. *Diabetologia* 35:1055–1059, 1992
3. Haffner SM, Morales PA, Gruber MK, Hazuda HP, Stern MP: Lack of association of lipids, lipoproteins and Lp(a) with microalbuminuria in NIDDM. *Arteriosclerosis* 13:205–210, 1993
4. Irish AB, Simons LA, Simons J: Lipoprotein (a) concentration in diabetes: relationship to proteinuria and diabetes control. *Aust NZ J Med* 22:329–333, 1992
5. Utermann G: The mysteries of lipoprotein (a). *Science* 246:904–910, 1990
6. Heller FR, Jamart J, Honore P, Derue G, Novik V, Galanti L, Parfonry A, Hondekijn JC, Buysschart M: Serum lipoprotein(a) in patients with diabetes mellitus. *Diabetes Care* 16:819–823, 1993
7. Velho G, Erlich D, Turpin E, Neel D, Cohen D, Froguel P, Passa P: Lipoprotein(a) in diabetic patients and normoglycemic relatives in familial NIDDM. *Diabetes Care* 16:742–747, 1993

Pancreatic Metastases of Grawitz' Tumor Revealed by Ketoacidosis

Pancreatic metastases of renal cell carcinoma are a rare occurrence, expected in 1–3% of patients who develop a metastatic disease (1). Impaired carbohydrate metabolism is a common feature in chronic pancreatic diseases, but multiple metastases of the pancreas from a renal cell carcinoma revealed by diabetic ketoacidosis are exceptional. Only one case has been reported (2). Here we report a new observation in which ketoacidosis was the first sign of the recurrence of a renal cancer.

In 1991, a 55-year-old man was referred to our institute for an inaugural diabetic ketoacidosis. He had no history of diabetes. He had a surgical pituitary insufficiency (substituted by hydrocortisone [30 mg/day] and L-thyroxine [125 μg/day]) after the removal of a craniopharyngioma in 1978. In 1987, a left nephrectomy was performed for a renal cell carcinoma. The tumor was confined within the perirenal fascia (stage T2, N0, M0), and no local recurrence was found in the postoperative follow-up. On admission, the patient complained of asthenia, nausea, polyuropolydipsic syndrome with dehydration, and moderate weight loss. The palpation of his abdomen was normal and painless. Blood pressure was 125/75 mmHg, and body temperature was 37.3°C. Clinical examination and electrocardiogram found no other abnor-

mality. Blood glucose was 29.7 mM, potassium was 5.1 mM, blood sodium, chloride, urea, and creatinine were in the normal range. Arterial blood gas analyses were as follows: pH = 7.18, PCO^2 = 28 mmHg, PO^2 = 91 mmHg, and HCO^{3-} = 17 mM. Urinary ketones and glucose were found highly positive at (+++) and (++++), respectively. Anti-islet cell antibodies were not found in peripheral blood. CT scan revealed multiple metastases developed in the head and the corpus of the pancreas. No sign of local recurrence of the renal tumor was found. A histological examination was determined by fine needle aspiration of the pancreatic tumor and resulted in the diagnosis of a clear-type renal cell carcinoma. A technetium 99 MDP bone scan revealed a bone metastasis in one rib. Classical rehydration and optimalized insulinotherapy were effective rapidly. The patient refused any other treatment and was still alive 2 years later with an insulin total daily dose of 50 IU.

When renal cell carcinoma is diagnosed, metastases are present in ~25% (3–5). Most frequently, the involved sites are the lungs, lymph nodes, bones, and liver. However, metastatic renal cell carcinoma also occurs in a number of other organs and body regions, such as thyroid, spleen, bowel, and skin and rarely the pancreas (4–6). In most instances, pancreatic metastases are discovered at autopsy. Willis (7) found pancreatic metastases in only 3% of 500 patients with a malignant disease at autopsy. The primary tumor is a small cell carcinoma of the lung or renal cell carcinoma. With regard to renal cell carcinoma, Lubarsch (8) diagnosed pancreatic metastases in 1.3% of 320 autopsy cases. Pancreatic metastases also are diagnosed during life. The disease proved to be of metastatic origin in 4.5 (9) and 3.7 (10) of cases. Of these, the source was renal cell carcinoma in 1 and 1.7%, respectively. Clinical manifestations of pancreatic metastases vary: exocrine and/or endocrine function disturbances lead to exocrine and/or endocrine explorations (2,8). Jaundice, abdominal pain, weight loss, diarrhea, and digestive tract bleeding are the most common clinical features of these pancreatic metastases. Diabetes is exceptional, and sometimes an impaired carbohydrate metabolism is observed (1). Ultrasonography and CT-scan studies usually disclose nonspecific evidence of a tumor (11). Endoscopic ultrasonography seems promising because of its high sensitivity.

The occurrence of diabetes in a patient with a history of lung or renal neoplasia necessitates looking at pancreatic metastases. Pancreatic metastases of renal cell carcinoma carry a less grim prognosis because slowing evolution and surgical removal may be possible.

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References

1. Tongio J, Peruta O, Wenger JJ, Warter P: Métastases duodénales et pancréatiques du néphro-épithéliome: a propos de quatre observations. *Ann Radiol* 20:641–647, 1977
2. Guttman FM, Ross M, Lachance C: Pancreatic metastasis of renal cell carcinoma treated by total pancreatectomy. *Arch Surg* 105:782–784, 1972
3. Holland JM: Cancer of the kidney: natural history and staging. *Cancer* 32:1030–1042, 1973
4. Ritchie AW, Chisholm GD: The natural history of renal cell carcinoma. *Semin Oncol* 10:390–400, 1983
5. Fuselier HA Jr, Guice SL III, Brannan W, Ochsner MG, Sangisetty KV, Beckman EN, Barnes CA: Renal cell carcinoma: the Ochsner medical institution experience (1945–1978). *J Urol* 130:445–448, 1983
6. Klugo RC, Detmers M, Stiles RE, Talley RW, Cerny JC: Aggressive versus conservative management of stage IV renal cell carcinoma. *J Urol* 118:244–246, 1977
7. Willis RA: *The Spread of Tumours in the Human Body*. 3rd ed. London, Butterworths, 1973, p. 216–217
8. Lubarsch O: Handbuch der speziellen. In *Pathologischen Anatomie und Histologie Niere*. Vol. 6. Berlin, Springer-Verlag, 1925, p. 607–720
9. Whittington R, Moylan J, Dobelbower RR, Kramer S: Pancreatic tumors in patients with previous malignancy. *Clin Radiol* 33:297–299, 1982
10. Opocher E, Galeotti F, Spina GP, Battaglia G, Hernandez C: La diagnosi dei tumori secondari del pancreas: analisi di 13 casi. *Minerva Med* 73:577–581, 1982
11. Wernecke K, Peters PE, Galanski M: Pancreatic metastases: U.S. evaluation. *Radiology* 160:399–402, 1986

Dorchy's Recipes Explaining the "Intriguing Efficacity of Belgian Conventional Therapy"

In the January 1993 issue of *Diabetes Care*, Bougnères et al. (1) published the results of a French multicenter study comparing a three-injection insulin regimen (called intensified insulin therapy) with a conventional two-injection therapy in patients aged 7–18 years with >1-year duration of insulin-dependent diabetes mellitus. They were evaluated after 1 year of treatment. The mean HbA_{1c} levels decreased from 9.8% (i.e., 146% of normal values) in the two-injection group to 9.3% in the three-injection group (i.e., 139% of normal values).