The Turkish Journal of Pediatrics 33: 135-138, 1991

CONTRAST MATERIAL – INDUCED ACUTE RENAL FAILURE IN A DIABETIC PATIENT*

*Nesrin Beşbaş MD**, Rezan Topaloğlu MD***, Ümit Saatçi MD****

SUMMARY: Beşbaş N, Topaloğlu R, Saatçi Ü. (Pediatric Nephrology Unit, Department of Pediatrics, Hacettepe University Faculty of Medicine). Contrast material-induced acute renal failure in a diabetic patient. Turk J Pediatr 33: 135-138, 1991.

A 16-year-old girl diagnosed as having type 1 diabetes mellitus and oliguric acute renal failure which developed 24 hours after computerized tomography with the infusion of contrast material is presented. She required short-term hemodialysis and in two weeks her renal functions returned to normal. All the findings in this high-risk patient suggest the development of contrast material-induced nephropathy. We propose that there should be adequate hydration before radiocontrast material studies are performed on diabetic patients. *Key words: contrast material, acute renal failure, type 1 diabetes mellitus.*

The infusion of radiographic contrast material is considered to be the third leading cause of iatrogenic acute renal failure (ARF)[1]. The incidence of ARF after the administration of radiocontrast material is high in high-risk patients such as those with diabetes mellitus[1,2]. We report a case of type 1 diabetes mellitus (DM) in which ARF was induced by infusion of radiocontrast material.

Case Report

A 16-year-old girl was admitted to the Ankara Social Security Hospital in a semi-comatose state of two days' duration. The patient underwent computerized brain tomography with the infusion of contrast material. A diagnosis of brain edema and encephalitis was established and she was referred to the Hacettepe University Children's Hospital for further evaluation.

Physical examination revealed a stuporus, acidotic girl. The temperature was 36 °C; respiration rate, 35/min; pulse rate, 120/min, and blood pressure, 120/70 mm Hg.

Laboratory studies revealed a hemoglobin of 13 g/dl and white blood cell count of 14,000/mm^3, with normal red blood cell morphology. Urinalysis revealed a specific gravity of 1028, 2 + protein, 4 + sugar, and a urine sediment with

* From the Pediatric Nephrology Unit, Department of Pediatrics, Hacettepe University Faculty of Medicine, Ankara.

** Associate Professor of Pediatrics, Hacettepe University Faculty of Medicine.

*** Pediatrician, Hacettepe University Institute of Child Health.

**** Professor of Pediatrics, Hacettepe University Institute of Child Health.

numerous erythrocytes and rare, fine granular casts. Blood sugar was 600 mg/dl, serum sodium 131 mEq/L, potassium 5.7 mEq/L, and arterial blood pH 6.95. In view of the physical and laboratory findings, a diagnosis of diabetic ketoacidosis was made and she was admitted to the hospital.

Therapy was started which consisted of an appropriate dose of insulin and the infusion of fluids and electrolytes. Despite loading with 400 ml/m^2 intravenous fluids on two occasions, and the administration of 3600-4200 ml/m^2/day maintenance fluids, the patient had a urinary output of only 180 ml/day. The blood sugar was put under control but not the acidosis. THer BUN was 86 mg/dl and the serum creatinine concentration 5 mg/dl; these values were progressively increasing. Other laboratory studies indicated an erythrocyte sedimentation rate (ESR) of 13 mm/h, antistreptolysin O (ASO) titer 625 Todd units, C-reactive protein (CRP) negative, latex fixation test, negative, complement components: C3 76 mg/dl (normal 60-120 mg/dl); C4 24 mg/dl (normal 20-40 mg/dl), anti-nuclear antibody (ANA) negative and anti-DNA 10 U/ml. Urine osmolarity was 320 mOsml, blood osmolarity 294 mOsml, urine sodium 61 mEq/L, fractional sodium excretion 3.1 %, and the urine urea nitrogen/blood urea nitrogen ratio was 2. The renal insufficiency index was compatible with acute renal insufficiency.

Central venous pressure was 17 cm H_2O. The chest X-ray revealed signs of hypervolemia and ultrasonography showed bilateral enlarged kidneys. Two doses of furosemide 5 mg/kg were administered, but there was no response to treatment. The patient was then placed on hemodialysis. After two sessions of hemodialysis, she started to urinate and the BUN and serum creatinine levels progressively fell to 21 and 0.9 mg/dl, respectively within two weeks.

Discussion

Hyperglycemic hyperosmolar nonketotic coma, diabetic ketoacidosis, radiocontrast material-induced nephropathy, renal papillary necrosis, glomerulonephritis and septicemia are common causes of ARF in diabetic patients[2]. ARF is a rare complication of diabetic ketoacidosis and occurs as a result of hypovolemia due to profound dehydration. The development of ketoacidosis causes patients to present to the hospital sooner than those with hyperglycemic hyperosmolar nonketotic coma, and thus, dehydration is less severe and hypotension less common, as was the case in our patient[2, 3].

Thus the possibility of renal papillary necrosis should always be considered in a diabetic patient with rapidly progressive renal failure. Elderly female diabetics are most frequently affected, and usually present with recurrent fever, hematuria and loin pain. Ultrasonography may indicate obstruction and loss of medullary tissue[2, 4]. These findings however were not observed in our patient.

Renal disease, other than that caused by diabetic glomerulosclerosis, may occur in three percent of diabetics and may lead to acute renal failure[2]. Idiopathic membranous nephropathy, acute post-infectious glomerulonephritis and rapidly progressive glomerulonephritis are most commonly found in diabetic patients[5]. With such laboratory findings as mild proteinuria, normal C3, C4, and anti-DNA levels, negative ANA and the serum creatinine level and urine analysis returning to normal within two weeks, glomerulopathies were easily excluded.

The incidence of contrast material-induced nephropathy in normal patients following intravenous urography, angiography or computerized tomography scanning ranges between two to five percent in normal patients and nine to 16 percent in high-risk patients with such conditions as pre-existing renal insufficiency, DM and congestive heart failure[6, 7]. The mechanism of renal damage is uncertain, but it is probably related to direct tubular toxicity and to a reduction in renal blood flow, causing renal ischemia and a decrease in the glomerular filtration rate[8].

Typical contrast material-induced ARF is a mild, reversible condition of short duration. Serum creatinine levels begin to rise within 24 h and peak in 3-5 days returning to normal within 7-10 days[2, 8]. Patients may require short-term dialysis, but irreversible renal failure is rare[9]. Our patient developed oliguric ARF 24 h after infusion of the contrast material and the condition progressed in 48 hours. She required hemodialysis. After two sessions of hemodialysis, she started to urinate and the renal functions returned to normal within two weeks.

The findings and clinical course of this particular high-risk patient strongly suggest that the contrast material used had induced the ARF.

In conclusion, we propose that radiologic studies with contrast material be avoided in diabetics, but if contrast material has to be administered, proper precautions should be taken such as ensuring adequate hydration prior to injection.

REFERENCES

1. Hou SH, Bushinsky DA, Wish JB, et al. Hospital-acquired renal insufficiency: a prospective study. *Am J Med* 74:243, 1983.
2. Grenfell A. Acute renal failure in diabetics. *Intensive Care Med* 12:6, 1986.
3. Tunbridge WM. Factors contributing to the deaths of diabetics under fifty years of age. On behalf of the Medical Services Study Group and British Diabetic Association. *Lancet* 2:569, 1981.
4. Eknoyan G, Qunlbi WY, Grissom RT, et al. Renal papillary necrosis: an update. *Medicine* 61:55, 1982.
5. Kasinath BS, Mujais SK, Spargo BH, Katz AI. Nondiabetic renal disease in patients with diabetes mellitus. *Am J Med* 75:613, 1983.
6. Brezis M, Epstein FH. A closer look at radiocontrast-induced nephropathy. *N Engl J Med* 320:179, 1989.

7. Parfrey PS, Griffiths SM, Barrett B, et al. Contrast material-induced renal failure in patients with diabetes mellitus, renal insufficiency, or both. A prospective controlled study. *N Engl J Med* 320:143, 1989.
8. Berkseth RO, Kjellstrand CM. Radiologic contrast-induced nephropathy. *Med Clin North Am* 68:351, 1984.
9. Weinrauch LA, Healy RW, Leland OS Jr, et al. Coronary angiography and acute renal failure in diabetic azotemic nephropathy. *Ann Intern Med* 86:56, 1977.