thus avoiding the risk of loss of bands, which is encountered when only a part of the gel is stained,

- The risk of inhibiting an enzyme activity, because of soaking the gel with the buffer used for the previous enzyme staining, is eliminated.

We are now applying our method to other genetic polymorphisms in blood and are looking into the possibility of extending it to other electrophoretic substrates and molecules.

References

Rapid Chemical Diagnosis of Kerosene Ingestion by NMR, Shuichi Yamaguchi,1 Hideaki Yamamoto,2 Rieko Mizukoshi,1 Naoya Koda,1 Shin-ichiro Hamano,1 Hidetsugu Nozaki,2,3 and Masako Fujiiwa4 (1 Div. of Endocrinol. and Metab., 2 Dept. of Radiol., 3 Div. of Neurol., and 4 Dept. of Cardiol., Saitama Children’s Med. Center, 2100 Magome, Iwatsuki, Saitama, Japan 339; 2 present address: Dept. of Pediatrics, Jikei Univ. School of Med., 3-25-3 Nishishinbashi, Minatoku, Tokyo, Japan 105)

Kerosene is sometimes inappropriately stored in the home and accidentally ingested by young children. In pediatric emergency wards, ingestions of kerosene or other hydrocarbons are not rare, accounting for about 3% of all of the poisonings reported to the Poison Center in Tsukuba, Japan (1), and 7% in the U.S. (2).

Ingestion of kerosene is usually diagnosed casually, by the aromatic smell of hydrocarbon. We encountered three cases of kerosene ingestion, but one patient lacked the smell of kerosene. All three were confirmed as kerosene ingestions by analysis of gastric content with nuclear magnetic resonance (NMR) spectroscopy; gastric lavage was also monitored by this method.

Gastric contents were collected after intubation to prevent respiratory aspiration of kerosene. Gastric fluid, 0.5 mL, was mixed with 0.1 mL of sodium 3-trimethylalloylpropionate-2,2,3,3,3-d4 (TSP; MSD Isotopes, Montreal, Canada), 10 mmol/L, in D2O as an internal standard. 1H-NMR was recorded at 299.949 MHz with a Unity-300 spectrometer (Varian Instruments, Sunnyvale, CA). Chemical shifts (ppm) were referenced to TSP. The spectra were acquired with 60° pulses (10.0 ms), 16384 data points, and a repetition time of 1.7 s. An automatic presaturation method was used to suppress signals of water. The kerosene stan-

dard solution was 100 mL/L in distilled water.

1H-NMR spectra in the gastric contents from a patient (Figure 1A) showed doublet signals (0.864, 1.299 ppm). These signals were also observed in the other two patients and in the kerosene standard solution (Figure 1C). After gastric lavage with 1.5 L of isotonic saline, the signals for kerosene disappeared in the lavage fluid (Figure 1B).

This analytical method is rapid (15 min), does not require any treatment of sample before analysis, and gives quantitative and qualitative information (3). During the lavage, NMR spectrometry rather than odor was useful for monitoring the presence of kerosene.

The chemical diagnosis of hydrocarbon ingestions ordinarily depends on analyses with gas chromatography–mass spectrometry, high-performance liquid chromatography, and other analytical techniques—methods that are time-consuming and cannot provide prompt information to the clinical staff. With NMR spectrometry, the rapid diagnosis of hydrocarbon ingestion and some other kinds of poisoning need not depend on a nonscientific smell method.

References

Above-Normal Urinary Excretion of Albumin and Retinol-Binding Protein in Chronic Heart Failure, Gertrude Ellekilde,1 Jan Holm,2,3 Finn Edler von Eyben,1 and Lars Hemmingsen2 (Depts. of 1 Intern. Med. and 2 Clin. Chem., Central Hospital Nykøbing Falster, DK-4800 Nykøbing Falster, Denmark; 3 author for correspondence)

Many patients with chronic heart failure have impaired renal function attributable to a reduced cardiac output and decreased renal perfusion. Thus, an increased concentra-

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**Fig. 1.** 1H-NMR spectra in (A) gastric contents before gastric lavage, (B) gastric contents after gastric lavage, and (C) kerosene in distilled water (100 mL/L).
tion of creatinine in serum is common in these patients (1). Proteinuria is also caused by renal dysfunction and may predict cardiovascular events (2). Increased urinary albumin excretion is mainly due to glomerulopathy. Retinol-binding protein (RBP) is a low-molecular-mass (21 kDa) protein that, after ultrafiltration in the glomerulus, is reabsorbed in the proximal tubules. Hence, increased urinary RBP excretion indicates a dysfunction of the proximal tubules with an impairment of the reabsorption of low-molecular-mass proteins. We recently observed an increase in the urinary concentrations of albumin and RBP in some patients with hypertension (3). In the present study we examined the urinary excretion of albumin and RBP in patients with moderate to severe chronic heart failure.

We studied 23 patients (19 men and 4 women; median age 74 years, range 66–87 years). They were consecutively admitted to our hospital with a diagnosis of chronic heart failure and dyspnea during physical exercise. Serum creatinine concentrations were normal (<125 μmol/L) in 17; the remaining 6 had values between 125 and 190 μmol/L. All were Albustix-negative for albumin in urine. None of the patients had diabetes mellitus, arterial hypertension, myocardial infarction within six months, valvular disorder, acute pulmonary edema, or primary renal disease. None of the patients received angiotensin-converting enzyme (ACE) inhibitors. We measured the concentration of albumin and RBP in overnight urine samples by immunochemical assays (4, 5).

As Figure 1 shows, 13 had increased urinary albumin concentrations: 9 with values between 0.45 and 4.5 μmol/L (microalbuminuria), and 4 with values >4.5 μmol/L (macroalbuminuria); 10 had values <0.45 μmol/L (normoalbuminuria). Seven had increased urinary RBP concentrations (>10 nmol/L), and 16 had normal values. Six of seven patients with increased urinary concentrations of RBP also had above-normal urinary concentrations of albumin.

This pilot study shows that 13 of 23 patients with chronic heart failure had above-normal urinary albumin concentrations despite a negative dipstick reaction. In addition, six of these patients had above-normal RBP concentrations. The serum concentration of creatinine increased in only 4 of these 13 patients. Hence, the urinary albumin concentration seems to be a more sensitive index of glomerular dysfunction than is the serum creatinine concentration. The increased urinary albumin values could not be ascribed to the treatment of the heart failure: ACE inhibitors may cause proteinuria, but none of our patients were treated with these drugs (3).

In conclusion, negative Albustix reaction and normal serum creatinine concentrations do not exclude abnormalities in renal function in patients with chronic heart failure. The present study demonstrates pathological changes in the renal handling of the two proteins: albumin (a marker of glomerular dysfunction) and RBP (a low-molecular-mass protein marker of proximal tubular dysfunction) in some patients with chronic heart failure. The prognostic implications of this finding remain to be settled.

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References

Plasma Concentrations of Lipid Peroxidation Products in Children with Acute Lymphoblastic Leukemia, Abd-El-Rahman M.A. Hammouda,1 Sayed F. Soliman,1 Koth A. Tolba,2 Zeinab A. El-Kabbany,2 and Madiha S. Makhlouf2 (1 Biochem. Dept., 2Pediatrics Dept., Faculty of Med., Ain Shams Univ., Cairo, Egypt)

Acute leukemia is the most common childhood malignancy (1). Leukemic patients are subjected to a combined cytotoxic therapy to induce remission; thereafter, they are maintained on a special regimen for two to three years (2). Follow-up of patients is mandatory for detection of relapse and initiation of therapy when needed. Clinical manifestations are extremely variable and the routine blood picture may not be conclusive (3). Bone marrow examination is mandatory whenever a relapse is suspected. Although membrane lipid peroxidation has been related to leukemia (4), the degree of lipid peroxidation has not been tested as a marker of the disease activity. We have investigated the possible role of the plasma concentration of lipid peroxidation products as a marker of disease activity in acute lymphoblastic leukemia (ALL).

Heparinized fasting blood samples were collected from children with different stages of ALL (confirmed by bone marrow examination) and from matching healthy controls. Patients with other coincident diseases such as hepatitis or lymphoma and those who had received blood transfusions