LETTERS TO THE EDITOR

TO THE EDITOR:

In the past year I have been privileged to practice medicine in a setting which allows me to see a variety of patients. One of the first ones I saw here remains one whom I think of often. Her determination taught me much. She was a 53 year old woman who presented with a common problem—hyperlipidemia.

She had been completely asymptomatic when the problem was detected on routine lab work two years ago. Her family history was significant for cardiovascular disease. Her blood work at the time of presentation revealed a fasting total cholesterol level of 265 mg/dL, an LDL cholesterol level of 190 mg/dL and a triglyceride (TG) level of 404 mg/dL. She was taking 200 mg of gemfibrozil and had been for the past two years. Her TG level ran from 500-1,100 prior to starting medication. She experienced no side effects from the gemfibrozil but desired to discontinue its use and maintain a lower lipid level naturally.

She was perimenopausal and had been on Ogen (estropipate) for two months without complications. She maintained an active lifestyle which included a workout routine four or more times per week consisting of running and weight training. She used no alcohol, tobacco or caffeine. Her diet was standard American fare.

The initial plan consisted of reducing her intake of satured fats, no dairy products, no beef, and an increased consumption of soluble fiber. Essential fatty acids and a multiple vitamin were also instituted with a plan for gradual discontinuation of the gemfibrozil.

She returned three months later and her repeat lab work showed a total cholesterol level of 227, LDL level of 134 and TG level of 298. With the assumption that the slight elevation in total cholesterol was due to discontinuing gemfibrozil she continued on the simple regimen. Ostaderm (by Bezwecken) was substituted for the Ogen at this visit.

Three months later her total cholesterol level was 177, LDL level 99, and TG level 223. She continues to have periodic lab work done and all her lipid levels have now come into the normal range. What most impressed me was the simplicity of the treatment plan and the resulting response. Certainly there are more elaborate supplemental regimes that could have been implemented, but they simply weren't necessary. Sometimes less is more.

Sincerely,

Roberta Bourgon, ND
Yellowstone Naturopathic Clinic
Billings, MT

MUCOPOLYSACCHARIDOSIS II—HUNTER'S SYNDROME

TO THE EDITOR:

Mucopolysaccharidosis II, also called Hunter's syndrome, is an X-linked disorder marked by a deficiency in the enzyme iduronosulfate sulfatase. This causes excessive build-up of mucopolysaccharides, dermatan sulfate and heparan sulfate in the body and their increased urinary excretion. Typical signs are coarse facies with thick lips and flattened nasal bridge, skeletal dysplasia, mental deficiency, short stature and hepatosplenomegaly. The excess mucopolysaccharides cause fluid accumulation in the ears, heart, kidney, and joints. The severe form has a predicted life span of 15 years; the mild form 30-50 years. There is no allopathic treatment other than managing heart and kidney complications as they arise (1).

A two year old boy presented in February 1997 with a case of mucopolysaccharidosis II by previous diagnosis. Initial symptoms included: speech and motor skills behind by 12-18 months, delayed ability to walk, inability to jump, and poor balance. He developed multiple middle ear infections with drainage of honey mustard fluid. Tympanostomy tubes were placed in 1995 and 1997. He wore a hearing aid in the right ear with bilateral hearing impairment at 60 dba. The patient fatigued easily, had dyspnea on exertion, heavy breathing, skipped breaths, and snored. From birth to 18 months he had cystic acne which resolved with ear tubes. There were red, macular skin lesions on his shoulders which were not painful or pruritic. He was 3' 0" and 40 lbs. The patient had multiple sinus infections and nasal congestion. The joints were stiff and painful. The patient could not stand without bending his knees and his gait was stiff. There was audible clicking upon neck rotation. Facial features included flushed cheeks, a large head, protruding eyes, a prominent ridge between the eyebrows, and large tongue. He had daily bowel movements which were soft or watery. Mild hepatosplenomegaly was detected.

Table 1 summarizes the treatment initiated in March 1997. By May 1997 the patient was talking for the first time. His mother reported a decrease in the patient's joint stiffness and pain with no ear or sinus infections since March. His eyes were no longer prominent and the ridge between his eyebrows had decreased. The patient had greater energy, needed fewer naps, had no dyspnea on exertion, did not skip breaths, and no longer used his hearing aid. The patient appeared to be only 3-6 months behind in speech and 3 months behind in motor skills. His balance was better and he could jump and ride a pony. His stools were solid for the first time. The click previously

heard on neck rotation was absent. The mother believed the diet was making the biggest difference, noting the patient's face and abdomen swelled after consuming carbohydrate.

In November 1997 there were still no ear or sinus infections reported. Nasal congestion and discharge were greatly reduced. Joint stiffness returned whenever Lymphomyosot was not taken. The mother reported the skin lesions came and went; none were seen on examination. Motor and speech skills continued to improve. The patient could stand with knees unbent. He still reacted to excessive carbohydrate intake with bloating and facial swelling. The patient was beginning to look more normally proportioned. A probiotic supplement, HMF Forte (containing 5 billion live organisms per capsule), 1 cap qd, was instituted due to excessive past antibiotic use and this appeared to keep the patient's stools firm. His height was now 3' 4" and weight 45 lbs.

It appears the low carbohydrate diet had a significant impact on the patient's symptoms, growth and development. It is the author's belief that this is due to decreasing precursors to mucopolysaccharides and thereby reducing their build-up. It also appeared the Lymphomyosot had a direct influence on the joint symptoms. The impact of the other therapies was unclear. Continuation of the regime including strict dietary restrictions appears warranted given the improvements in the condition, until a true cure can be found. There was some concern about consumption of so much lipid in the diet, but the child was fed entirely organic meats, eggs, and fish and was eating vegetables. Even if there is an increased risk of atherosclerosis on such a diet, it would still greatly outweigh the risks of allopathic management alone for his Hunter's syndrome. It is this author's hope that the regime will lead to normal or near normal mental function, height, appearance, and overall physical health and function for this and other patients.

Sincerely,

Marleen Haverty, ND, LAc
Natural Health Clinic of Bastyr University
Seattle, WA

TABLE 1
NATUROPATHIC APPROACH TO MUCOPOLYSACCHARIDOSIS II

Treatment Agent	Ingredients/ Potency	Dose	Rationale
Very low carbohydrate diet	n/a	n/a	Decrease mucopolysaccharide precursors.
Biomins	100 mg Ca, 90 mg Mg, 30 mg K, 7 mg Zn, 5 mg Mn, 5 mg Fe, 1 mg B, 0.3 mg Cu, 68 mcg Cr, 50 mcg Se, 30 mcg Mb, 30 mcg V, 30 mg KI (all minerals aspartate salts)	1 cap qd	Mineral cofactors to assist in driving enzymatic reactions.
Bromelain	2000 mcu/g potency	250 mg qd	Proteolytically break down substrates and remove fluids.
Calcarea carbonica	homeopathic remedy, 200 C potency	3-5 pellets 4 times q2h on three occasions	Best matched specific symptom picture.
Lymphomyosot	*Geranium robertinum 4X, Nasturtium aquaticum 4X, Ferrum iodatum 12X, Juglans nigra 3X, Myosotis arvensis 3X, Scrophularia nodosa 3X, Teuctrium scorodonia 3X, Thyroidonium 12X, Calcarea phosphorica 12X, Sarsaparilla 6X, Aranea diadema 6X, Gentiana lutea 5X*	10 gtts qd	Increase lymph and fluid drainage.

Formulated by the author with the assistance from Dr. Dirk Powell, ND.

REFERENCE

1. Shapiro LJ. Mucopolysaccharidoses, glycoproteinoses, and mucolipidoses. in: Rudolph AM, Hoffman JIE, Rudolph CD (eds) *Rudolph's Pediatrics* 20th ed. (Stamford, CT: Appleton & Lange, 1996):356-64.