Bowel obstruction secondary to mesenteric cyst formation

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A rare cause of intra-abdominal obstruction, mesenteric cysts derive from lymphatic tissue. The differential diagnosis should include mesenteric cyst when the patient presents with a history of multiple episodes of partial small-bowel obstruction or with an asymptomatic abdominal mass. A year-old girl was brought to the Family Practice Center with episodic, sharp, nonradiating, left-sided, mid-upper-abdominal pain. Examination indicated a possible diagnosis of mesenteric adenitis. Due to variation in the signs and symptoms of the condition, it appears that the use of ultrasonography is the most effective, nonoperative method of evaluation. Ultrasonography appears to be the most effective, nonoperative method of evaluation. Surgery is the only definitive diagnostic and therapeutic modality.

There are many theories concerning the formation of mesenteric cysts, which are derivations of lymphatic tissue that arise in the mesentery, mesocolon, and omentum. Most investigators attribute the cause to be congenitally malformed or malpositioned lymphatic tissue, while others blame failure on the leaves of the mesentery to properly fuse.¹ Other proposed causes include embryonic abdominal trauma, inflammatory obstruction of existing channels, and localized degeneration of lymph nodes.¹

The rarity of this condition makes preoperative diagnosis very difficult.^{2,3} In 1986, Kurtz and associates⁴ found 152 cases of mesenteric or retroperitoneal cyst reported in the English literature between 1950 and 1985; they presented an additional 10 cases, for a total of 162 cases.

With so few cases to draw from, a general practitioner might well omit the possibility of mesenteric cyst in diagnosing a patient with intermittent abdominal pain. Although surgery may present the only obvious therapeutic modality, a definitive diagnosis may be achieved with the use of ultrasonography.

Report of case

A 4-year-old girl was brought to the Texas College of Osteopathic Medicine Southside Family Practice Clinic because of left-sided, mid-upper-quadrant abdominal pain, which had begun four days earlier. The pain was intermittent, sharp, and nonradiating. Each episode initially had lasted two to three minutes, but the duration increased to 10 minutes. There were pain-free intervals lasting many hours.

The history was negative for trauma, previous surgery, or recent travel. There had been no nausea, vomiting, diarrhea, or constipation. The child's last bowel movement, which had been hard in consistency, had occurred two days earlier. Her appetite was decreased, but she was not anorexic. There were no other related systemic complaints.

The patient was afebrile, and her pulse and respiratory rates were stable. She pointed to the midabdomen as the site of her discomfort. Bowel sounds auscultated in all four quadrants were physiologic in nature. On palpation, a well-defined, movable mass approximately 5 cm to 6 cm in diameter was discovered in the left upper quadrant. There was no rigidity, rebound phenomenon, or peritoneal signs; the child complained of slight tenderness on deep palpation. The rectal examination revealed no occult blood or other abnormal findings.

Because of limited laboratory facilities at the clinic, only urinalysis was performed, with the following results: pH value, 5.0; urobilinogen level, 0.1 Ehrlich unit; +1 proteinuria; and 4+ ketonuria. The microscopic findings were within normal limits.

The differential diagnosis was narrowed to Meckel's diverticulum, partial small-bowel obstruction, or mesenteric adenitis, and the child was referred to the emergency room at a local hospital.

However, against the physician's advice, the parents took the child home. Some eight hours later, she awoke with severe, unremitting abdominal pain, without nausea or vomiting. She was taken to another local emergency room, where she was found to have an oral temperature of 101°F and tachycardia.

On physical examination, the left-sided upper quadrant mass was palpated and peritoneal signs were present. The WBC count was 13,600/cu mm, with a left shift. Serum electrolyte values were within normal limits, and the serum amylase level also was normal. Plain abdominal films revealed fluid-filled loops of bowel and a mass appropriately 5 cm to 6 cm in diameter (Figs 1-4).

The child underwent surgery, which revealed a 6.5-cm, globular, cyst-like mass involving the midileum. The

lesion had caused a volvulus of the small bowel, and approximately 24 inches of markedly ischemic bowel was noted. The small bowel was resected and end-to-end anastomosis was performed. The patient tolerated the procedure well and was discharged on the fifth postoperative day.

Pathologic examination disclosed marked ischemia and mucosal necrosis of the entire segment of resected bowel, and a 6.5-cm, lymphogenous mesenteric cyst.

Discussion

The present case reflects the variation in signs and symptoms of mesenteric cysts. Obviously, the clinical presentation will vary with cyst size and location, as well as with other data related to obstruction. The form and position of the tumor constantly change; therefore, identification is very difficult. In one review,⁵ it was noted that a mass was palpated in 58% of cases. Many cases have been confused preoperatively with acute appendicitis or appendiceal abscess when the cyst was palpated in the right lower quadrant.²

Large cysts may cause obvious abdominal distention or a palpable mass, whereas small ones may be completely asymptomatic. The onset of abdominal pain reflects traction in the mesentery, with or without compression or torsion of the small bowel.

The most common presentation is a slowly enlarging, painless abdominal lesion, which causes symptoms only when bowel obstruction occurs. Warfield⁶ states that an abdominal tumor that is rounded, smooth, nontender, and very mobile in the transverse direction is somewhat indicative of a mesenteric cyst.

There are no definitive diagnostic tests. Roentgenography of the abdomen may reveal soft-tissue density or, rarely, calcification. Ultrasonography and computed tomography definitely demonstrates the cystic structure.⁷ And, also, the thin, intracystic septa may be visualized.⁵ Laboratory data that reveals dehydration or malabsorption may lead to suspicion of obstruction from a mesenteric cyst.

Fig 1. Lateral abdominal chest x-ray showing gas and fluid-filled loops of bowel.

Fig 2. Anteroposterior view of the abdomen showing gas and fluid-filled loops of bowel.

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- ▶ PRINIVIL appeared to attenuate diuretic-induced hypokalemia, hyperglycemia, and hyperuricemia.
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- ▶ Significantly fewer patients receiving PRINIVIL had an increase in serum triglycerides ($p < 0.05$).

1. Bolzano, K. et al.: The antihypertensive effect of lisinopril compared to atenolol in patients with mild to moderate hypertension, *J. Cardiovasc. Pharmacol.* 9(Suppl. 3):S43-S47, 1987.

*Not every product within this therapeutic class has the side effects described.

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[†]Comparable reduction in sitting diastolic blood pressure was seen with PRINIVIL and Tenormin.

^{††}The addition of hydrochlorothiazide caused further reductions ($p < 0.01$) in SSBP/SDBP: PRINIVIL, 16.3/12.5 mm Hg; Tenormin, 18.2/9.8 mm Hg.

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PRINIVIL is contraindicated in patients who are hypersensitive to this product and in patients with a history of angioedema related to previous treatment with an ACE inhibitor. All patients should be cautioned that excessive perspiration and dehydration may lead to an excessive fall in blood pressure because of reduction in fluid volume. Other causes of volume depletion, such as vomiting or diarrhea, may also lead to a fall in blood pressure.

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patients and 4.0% of patients with congestive heart failure. In most cases, these were isolated values which resolved despite continued therapy. Hyperkalemia was a cause of discontinuation of therapy in approximately 0.1% of hypertensive patients. Risk factors for the development of hyperkalemia include renal insufficiency, diabetes mellitus, and the concomitant use of potassium-sparing diuretics, potassium supplements, and/or potassium-containing salt substitutes, which should be used cautiously, if at all, with PRINIVIL® (Lisinopril, MSD) (see Drug Interactions).

Surgery/Anesthesia: In patients undergoing major surgery or during anesthesia with agents that produce hypotension, PRINIVIL may block angiotensin II formation secondary to compensatory renin release. If hypotension occurs and is considered to be due to this mechanism, it can be corrected by volume expansion.

Information for Patients

Angioedema: Angioedema, including laryngeal edema, may occur, especially following the first dose of PRINIVIL. Patients should be so advised and told to report immediately any signs or symptoms suggesting angioedema (swelling of face, extremities, eyes, lips, tongue, difficulty in swallowing or breathing) and to take no more drug until they have consulted with the prescribing physician.

Symptomatic Hypotension: Patients should be cautioned to report lightheadedness, especially during the first few days of therapy. If actual syncope occurs, patients should be told to discontinue the drug until they have consulted with the prescribing physician.

All patients should be cautioned that excessive perspiration and dehydration may lead to an excessive fall in blood pressure because of reduction in fluid volume. Other causes of volume depletion such as vomiting or diarrhea may also lead to a fall in blood pressure; patients should be advised to consult with their physician.

Hyperkalemia: Patients should be told not to use salt substitutes containing potassium without consulting their physician.

Neutropenia: Patients should be told to report promptly any indication of infection (e.g., sore throat, fever) which may be a sign of neutropenia.

NOTE: As with many other drugs, certain advice to patients being treated with PRINIVIL is warranted. This information is intended to aid in the safe and effective use of this medication. It is not a disclosure of all possible adverse or intended effects.

Drug Interactions

Hypotension: Patients on Diuretic Therapy: Patients on diuretics and especially those in whom diuretic therapy was recently instituted, may occasionally experience an excessive reduction of blood pressure after initiation of therapy with PRINIVIL. The possibility of hypotensive effects with PRINIVIL can be minimized by either discontinuing the diuretic or increasing the salt intake prior to initiation of treatment with PRINIVIL. If it is necessary to continue the diuretic, initiate therapy with PRINIVIL at a dose of 5 mg daily, and provide close medical supervision after the initial dose for at least 2 hours and until blood pressure has stabilized for at least an additional hour (see WARNINGS and DOSAGE AND ADMINISTRATION). When a diuretic is added to the therapy of a patient receiving PRINIVIL, an additional antihypertensive effect is usually observed. Studies with ACE inhibitors in combination with diuretics indicate that the dose of the ACE inhibitor can be reduced when it is given with a diuretic (see DOSAGE AND ADMINISTRATION).

Indomethacin: In a study in 36 patients with mild to moderate hypertension where the antihypertensive effects of PRINIVIL alone were compared to PRINIVIL given concomitantly with indomethacin, the use of indomethacin was associated with a reduced effect, although the difference between the two regimens was not significant.

Other Agents: PRINIVIL has been used concomitantly with nitrates and/or digoxin without evidence of clinically significant adverse interactions. No clinically important pharmacokinetic interactions occurred when PRINIVIL was used concomitantly with propranolol or hydrochlorothiazide. The presence of food in the stomach does not alter the bioavailability of PRINIVIL.

Agents Increasing Serum Potassium: PRINIVIL attenuates potassium loss caused by thiazide-type diuretics. Use of PRINIVIL with potassium-sparing diuretics (e.g., spironolactone, triamterene, or amiloride), potassium supplements, or potassium-containing salt substitutes may lead to significant increases in serum potassium. Therefore, if concomitant use of these agents is indicated because of demonstrated hypokalemia, they should be used with caution and with frequent monitoring of serum potassium.

Pregnancy Category C: Lisinopril was not teratogenic in mice treated on days 6 to 15 of gestation with up to 1,000 mg/kg/day (625 times the maximum recommended human dose). There was an increase in fetal resorptions at doses down to 100 mg/kg; at doses of 1,000 mg/kg, this was prevented by saline supplementation. There was no fetotoxicity or teratogenicity in rats treated with up to 300 mg/kg/day (188 times the maximum recommended dose) of lisinopril at days 6 to 17 of gestation. In rats receiving lisinopril from day 15 of gestation through day 21 postpartum, there was an increased incidence in pup deaths on days 2 to 7 postpartum and a lower average body weight of pups on day 21 postpartum. The increase in pup deaths and decrease in pup weight did not occur with maternal saline supplementation.

Lisinopril, at doses up to 1 mg/kg/day, was not teratogenic when given throughout the organogenic period in saline-supplemented rabbits. Saline supplementation (physiologic saline in place of tap water) was used to eliminate maternotoxic effects and enable evaluation of the teratogenic potential at the highest possible dosage level. The rabbit has been shown to be extremely sensitive to ACE inhibitors (captopril and enalapril) with maternal and fetotoxic effects apparent at or below the recommended therapeutic dosage levels in man. Fetotoxicity was demonstrated in rabbits by an increased incidence of fetal resorptions at an oral dose of lisinopril at 1 mg/kg/day and by an increased incidence of incomplete ossification at the lowest dose tested (0.1 mg/kg/day). A single intravenous dose of 15 mg/kg of lisinopril administered to pregnant rabbits on gestation days 16, 21, or 26 resulted in 88% to 100% fetal death.

There are no adequate and well-controlled studies in pregnant women. PRINIVIL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers: It is not known whether this drug is secreted in human milk. Because many drugs are secreted in human milk, caution should be exercised when PRINIVIL is given to a nursing mother.

Pediatric Use: Safety and effectiveness in children have not been established.

Adverse Reactions

PRINIVIL has been found to be generally well tolerated in controlled clinical trials involving 2,003 patients and subjects.

The most frequent clinical adverse experiences in controlled trials with monotherapy with PRINIVIL® (Lisinopril, MSD) were: dizziness (6.3%), headache (5.3%), fatigue (3.3%), diarrhea (3.2%), upper respiratory symptoms (3.0%), and cough (2.9%), all of which were more frequent than in placebo-treated patients. For the most part, adverse experiences were mild and transient in nature. Discontinuation of therapy was required in 6% of patients. In clinical trials, the overall frequency of adverse experiences could not be related to total daily dosage within the recommended therapeutic dosage range.

Other adverse experiences occurring in greater than 1% of patients and subjects treated with monotherapy with PRINIVIL in controlled clinical trials were: nausea (2.3%), hypotension (1.8%), rash (1.5%), orthostatic effects (1.4%), asthenia (1.3%), chest pain (1.3%), vomiting (1.3%), and dyspnea (1.1%). For information on adverse experiences occurring in greater than 1% of patients and subjects treated with PRINIVIL plus hydrochlorothiazide in controlled clinical trials, see Prescribing Information. Clinical adverse experiences occurring in 0.3% to 1% of patients treated with monotherapy with PRINIVIL in the controlled trials and rarer, serious, possibly drug-related events reported in uncontrolled studies or marketing experience included: **Body as a Whole:** Chest discomfort, fever, flushing. **Cardiovascular:** Angina pectoris, orthostatic hypotension, rhythm disturbances, tachycardia, peripheral edema, palpitation. **Digestive:** Abdominal pain, anorexia, constipation, flatulence. **Metabolism:** Gout. **Musculoskeletal:** Joint pain, shoulder pain. **Nervous System/Psychiatric:** Depression, somnolence, insomnia, stroke. **Respiratory System:** Bronchitis, sinusitis, pharyngeal pain. **Urogenital:** Oliguria, progressive azotemia, acute renal failure. **Other:** Blurred vision, pruritus, urinary tract infection, vasculitis of the legs. **Angioedema:** Angioedema has been reported in patients receiving PRINIVIL (0.1%). Angioedema associated with laryngeal edema may be fatal. If angioedema of the face, extremities, lips, tongue, glottis, and/or larynx occurs, treatment with PRINIVIL should be discontinued and appropriate therapy instituted immediately (see WARNINGS). **Hypotension:** In hypertensive patients, hypotension occurred in 1.2% and syncope occurred in 0.1% of patients. Hypotension or syncope was a cause for discontinuation of therapy in 0.5% of hypertensive patients (see WARNINGS). In patients with congestive heart failure, hypotension occurred in 5% and syncope occurred in 1% of patients. These adverse experiences were cause for discontinuation of therapy in 1.3% of these patients.

Clinical Laboratory Test Findings

Serum Electrolytes: Hyperkalemia (see PRECAUTIONS). **Creatinine, Blood Urea Nitrogen:** Minor increases in blood urea nitrogen and serum creatinine, reversible upon discontinuation of therapy, were observed in about 2% of patients with essential hypertension treated with PRINIVIL alone. Increases were more common in patients receiving concomitant diuretics and in patients with renal artery stenosis (see PRECAUTIONS). Reversible minor increases in blood urea nitrogen and serum creatinine were observed in approximately 9.1% of patients with congestive heart failure on concomitant diuretic therapy. Frequently, these abnormalities resolved when the dosage of the diuretic was decreased.

Hemoglobin and Hematocrit: Small decreases in hemoglobin and hematocrit (mean decreases of approximately 0.4 g% and 1.3 vol %, respectively) occurred frequently in patients treated with PRINIVIL but were rarely of clinical importance in patients without some other cause of anemia. In clinical trials, less than 0.1% of patients discontinued therapy due to anemia.

Other (Causal Relationship Unknown): Rarely, elevations of liver enzymes and/or serum bilirubin have occurred.

Overall, 2.0% of patients discontinued therapy due to laboratory adverse experiences, principally elevations in blood urea nitrogen (0.6%), serum creatinine (0.5%), and serum potassium (0.4%).

Dosage and Administration

Initial Therapy: In patients with uncomplicated essential hypertension not on diuretic therapy, the recommended initial dose is 10 mg once a day. Dosage should be adjusted according to blood pressure response. The usual dosage range is 20 to 40 mg per day administered in a single daily dose. The antihypertensive effect may diminish toward the end of the dosing interval regardless of the administered dose, but most commonly with a dose of 10 mg daily. This can be evaluated by measuring blood pressure just prior to dosing to determine whether satisfactory control is being maintained for 24 hours. If it is not, an increase in dose should be considered. Doses up to 80 mg have been used but do not appear to give a greater effect. If blood pressure is not controlled with PRINIVIL alone, a low dose of a diuretic may be added. Hydrochlorothiazide 12.5 mg has been shown to provide an additive effect. After the addition of a diuretic, it may be possible to reduce the dose of PRINIVIL.

Diuretic-Treated Patients: In hypertensive patients who are currently being treated with a diuretic, symptomatic hypotension may occur occasionally following the initial dose of PRINIVIL. The diuretic should be discontinued, if possible, for 2 to 3 days before beginning therapy with PRINIVIL to reduce the likelihood of hypotension (see WARNINGS). The dosage of PRINIVIL should be adjusted according to blood pressure response. If the patient's blood pressure is not controlled with PRINIVIL alone, diuretic therapy may be resumed as described above.

If the diuretic cannot be discontinued, an initial dose of 5 mg should be used under medical supervision for at least 2 hours and until blood pressure has stabilized for at least an additional hour (see WARNINGS and PRECAUTIONS, Drug Interactions).

Concomitant administration of PRINIVIL with potassium supplements, potassium salt substitutes, or potassium-sparing diuretics may lead to increases of serum potassium (see PRECAUTIONS).

Use in Elderly: In general, blood pressure response and adverse experiences were similar in younger and older patients given similar doses of PRINIVIL. Pharmacokinetic studies, however, indicate that maximum blood levels and area under the plasma concentration time curve (AUC) are doubled in older patients so that dosage adjustments should be made with particular caution.

Dosage Adjustment in Renal Impairment: The usual dose of PRINIVIL (10 mg) is recommended for patients with a creatinine clearance > 30 mL/min (serum creatinine of up to approximately 3 mg/dL). For patients with creatinine clearance ≥ 10 mL/min and ≤ 30 mL/min (serum creatinine ≥ 3 mg/dL), the first dose is 5 mg once daily. For patients with creatinine clearance < 10 mL/min (usually on hemodialysis), the recommended initial dose is 2.5 mg. The dosage may be titrated upward until blood pressure is controlled or to a maximum of 40 mg daily.

For more detailed information, consult your MSD Representative or see Prescribing Information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, PA 19486.

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Contraindications:

PRINIVIL® (Lisinopril, MSD) is contraindicated in patients who are hypersensitive to this product and in patients with a history of angioedema related to previous treatment with an ACE inhibitor.

Warnings:

Angioedema: Angioedema of the face, extremities, lips, tongue, glottis, and/or larynx has been reported in patients treated with ACE inhibitors, including PRINIVIL. In such cases, PRINIVIL should be promptly discontinued and the patient carefully observed until the swelling disappears. In instances where swelling has been confined to the face and lips, the condition has generally resolved without treatment, although antihistamines have been useful in relieving symptoms. Angioedema associated with laryngeal edema may be fatal. **Where there is involvement of the tongue, glottis, or larynx, likely to cause airway obstruction, appropriate therapy, e.g., subcutaneous epinephrine solution 1:1000 (0.3 mL to 0.5 mL), should be promptly administered (see ADVERSE REACTIONS).**

Hypotension: Excessive hypotension was rarely seen in uncomplicated hypertensive patients but is a possible consequence of the use of PRINIVIL in salt/volume-depleted persons, such as those treated vigorously with diuretics or patients on dialysis (see PRECAUTIONS, Drug Interactions and ADVERSE REACTIONS). In patients with severe congestive heart failure, with or without associated renal insufficiency, excessive hypotension has been observed and may be associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death. Because of the potential fall in blood pressure in these patients, therapy should be started under very close medical supervision. Such patients should be followed closely for the first 2 weeks of treatment and whenever the dose of PRINIVIL and/or diuretic is increased. Similar considerations apply to patients with ischemic heart or cerebrovascular disease in whom an excessive fall in blood pressure could result in a myocardial infarction or cerebrovascular accident.

If hypotension occurs, the patient should be placed in a supine position and, if necessary, receive an intravenous infusion of normal saline. A transient hypotensive response is not a contraindication to further doses, which usually can be given without difficulty once the blood pressure has increased after volume expansion.

Neutropenia/Agranulocytosis: Another ACE inhibitor has been shown to cause agranulocytosis and bone marrow depression, rarely in uncomplicated patients but more frequently in patients with renal impairment, especially if they also have a collagen vascular disease. Available data from clinical trials of PRINIVIL are insufficient to show that PRINIVIL does not cause agranulocytosis at similar rates. Periodic monitoring of white blood cell counts in patients with collagen vascular disease and renal disease should be considered.

Precautions:

General: Impaired Renal Function: As a consequence of inhibiting the renin-angiotensin-aldosterone system, changes in renal function may be anticipated in susceptible individuals. In patients with severe congestive heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with ACE inhibitors, including PRINIVIL, may be associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death.

In hypertensive patients with unilateral or bilateral renal artery stenosis, increases in blood urea nitrogen and serum creatinine may occur. Experience with another ACE inhibitor suggests that these increases are usually reversible upon discontinuation of PRINIVIL and/or diuretic therapy. In such patients renal function should be monitored during the first few weeks of therapy.

Some hypertensive patients with no apparent preexisting renal vascular disease have developed increases in blood urea nitrogen and serum creatinine, usually minor and transient, especially when PRINIVIL has been given concomitantly with a diuretic. This is more likely to occur in patients with preexisting renal impairment. Dosage reduction of PRINIVIL and/or discontinuation of the diuretic may be required.

Evaluation of the hypertensive patient should always include assessment of renal function (see DOSAGE AND ADMINISTRATION).

Hyperkalemia: In clinical trials, hyperkalemia (serum potassium > 5.7 mEq/L) occurred in approximately 2.2% of hypertensive

Fig 3. Anteroposterior view showing gas and fluid-filled loops and basic obliteration of organs.

Treatment consists of complete enucleation of the cyst without disruption of surrounding nonpathologic structures.² If the cyst is located close to the abdominal wall, or if obstruction or strangulation of bowel occurs, small-bowel resection may be necessary.

Summary

Although mesenteric cysts are uncommon, they may be seen by the general practitioner. Multiple episodes of partial small-bowel obstruction or asymptomatic abdominal masses should alert the physician to include mesenteric cysts in the differential

Fig 4. Lateral decubitus view showing the basic outline of the cyst.

diagnosis. Ultrasonography and computed tomography appear to be the most accurate and noninvasive mode of evaluation. As in this case, surgical exploration appears to be the only definitive diagnostic, as well as therapeutic, modality. Postoperative recovery generally is free of sequelae.

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