

## Bilateral Perinephric Fluid Accumulation: An Unusual Manifestation of Pulmonary Hypertension—A Case Report

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### *Abstract*

**This is the case report of a forty-seven-year-old man with an ostium secundum atrial septal defect and a very high grade of pulmonary hypertension, associated with a large bilateral perinephric fluid accumulation. The fluid accumulation was remarkably reduced after eleven phlebotomies over a twelve-month period. A pathogenetic relation with the Eisenmenger's syndrome is discussed. No previous report of this association has been found in a survey of the literature.**

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### *Introduction*

The perirenal space is an anatomic space bounded anteriorly and posteriorly by the perinephric fascia (Gerota's fascia), medially by the perivascular space, and inferiorly by the retroperitoneal or iliac space. Between the renal capsule and the perinephric fascia is the perinephric fat, which contains dense, fibrous septa that may either localize pathologic fluid accumulations or determine their eventual routes of diffusion.<sup>1,2</sup> Pathologies developing around the kidney are rare, regardless of the age of the patient, and those characterized by a fluid content are most often acquired and rarely congenital.<sup>3</sup>

This paper presents a case of Eisenmenger's syndrome that is associated with a large, bilateral, perinephric transudate of uncertain pathogenesis and nosography.

### **Case Report**

In November, 1990, a forty-seven-year-old man was admitted to our hospital (Clinica Medica II, Pisa) presenting slight discomfort in the right hypocondrium and around the right

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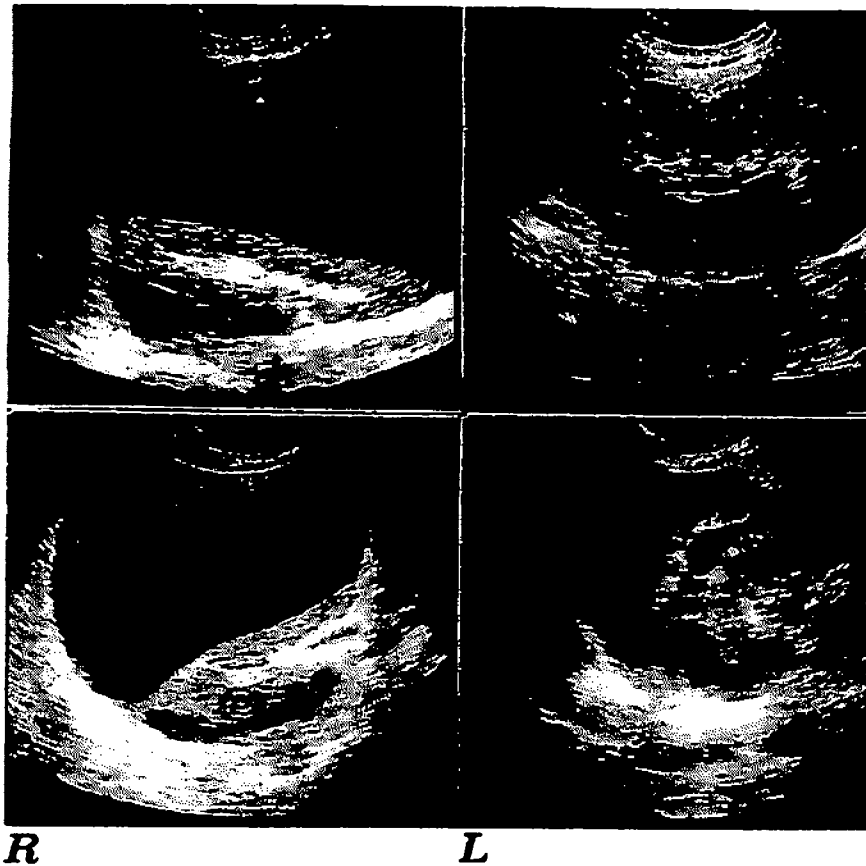


FIG. 1. Massive bilateral anechoic perinephric effusion. The right kidney is partially compressed and displaced.

costovertebral angle for a period of two months. He was diagnosed as having an ostium secundum atrial septal defect with a very high grade of pulmonary hypertension as evidenced by a systolic pressure of 130 mmHg in the right ventricle, bidirectional shunt, secondary erythrocytosis, and central cyanosis. Renal sonography had been performed eight months prior to this admission, in the course of the work-up of an inguinal hernia, and had appeared normal.

The arterial blood pressure was 130/80 mmHg. No peripheral edema or jugular venous distension was seen, and the hepatojugular reflux was negative. On auscultation, there was fixed splitting of the second heart sound with an accentuated pulmonic component, mild tricuspid regurgitation (+ + / + + +) on inspiration maximum along the lower left sternal border. A very large, smooth mass was palpable on the right side, and another, smaller mass that was mobile on inspiration was also perceptible with bimanual palpation on the left side.

Erythrocyte sedimentation rate 2/2; blood urea nitrogen; and serum glucose, creatinine, transaminases, lactic dehydrogenase, amylase, uric acid, calcium, potassium, and sodium were all in the normal ranges; tumor markers were absent. Urine culture was negative. Creatinine clearance was 80 cc/min. Red blood cells were  $7.32 \times 10^6/\text{mm}^3$ ; hemoglobin was 21.7 g/dL; hematocrit was 72.9%; white blood cells were  $6340/\text{mm}^3$ ; and platelets were  $233,000/\text{mm}^3$ .

The EKG showed normal sinus rhythm, heart rate was 80/min, and right atrial and ventricular hypertrophy were detected. The echo Doppler cardiogram showed marked dilatation of

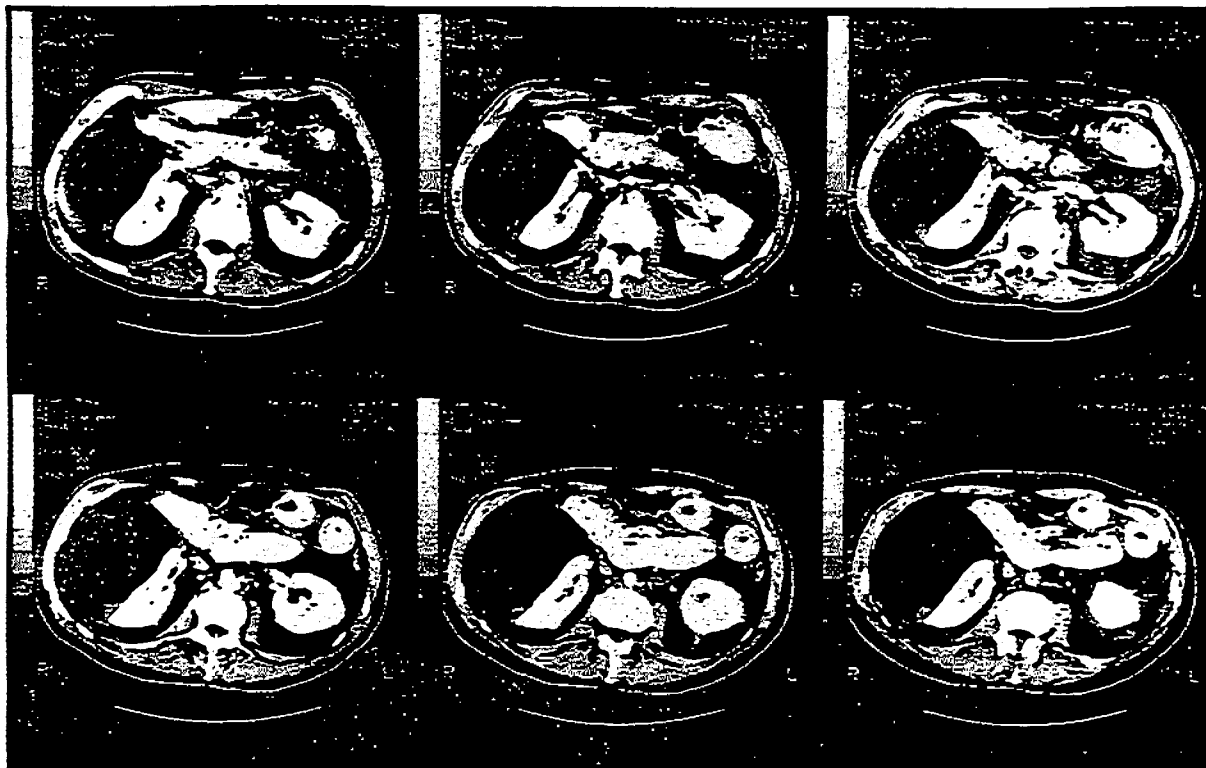


FIG. 2. Fluid collection contained by the renal fascia surrounding both kidneys.

the right side of the heart and a flattened left ventricle. A defect was present at the level of the interatrial septum, ostium secundum type; a bidirectional shunt and tricuspid insufficiency were observed. The right ventricular systolic pressure was 130 mmHg.

A chest x-ray showed cardiac enlargement due to dilatation of both the right atrium and ventricle, a large pulmonary trunk with prominence of its primary divisions, normal costophrenic angles, and normal position of the hemidiaphragm.

Abdominal sonography (Fig. 1) indicated the presence of a large, bilateral, anechoic perirenal fluid accumulation more evident on the right. The liver, spleen, pancreas, biliary tract, and large vessels were within normal limits. The vena cava and renal veins were patent with a regular Doppler signal. The right kidney appeared displaced and slightly compressed. Computerized tomography (Fig. 2) revealed formations with a liquid content surrounding the kidneys, contained by the renal fascia, and not modified following use of contrast media. No dilatation of the cavitory excretory structures was seen, and renal function was conserved.

Ultrasound guided drainage (with a 6F catheter) of the right perirenal accumulation resulted in removal of 800 cc of a clear-lipid liquid having a density of 1004, negative Rivalta, protein 0.82 g/dL, lactic dehydrogenase 24 units/L, amylase 12 U/L, and glucose 120 mg/dL. There were no neoplastic cells in the sediment. Culture examinations for ordinary bacteria and bacteria of Koch yielded negative findings.

During the hospitalization there were three additional paracenteses, including one from the left side, of about 900 cc, which were always transudate; then the patient was no longer subjected to paracentesis and the effusion was periodically checked by ultrasonography. After the discharge the patient underwent a series of eleven phlebotomies over a twelve-month

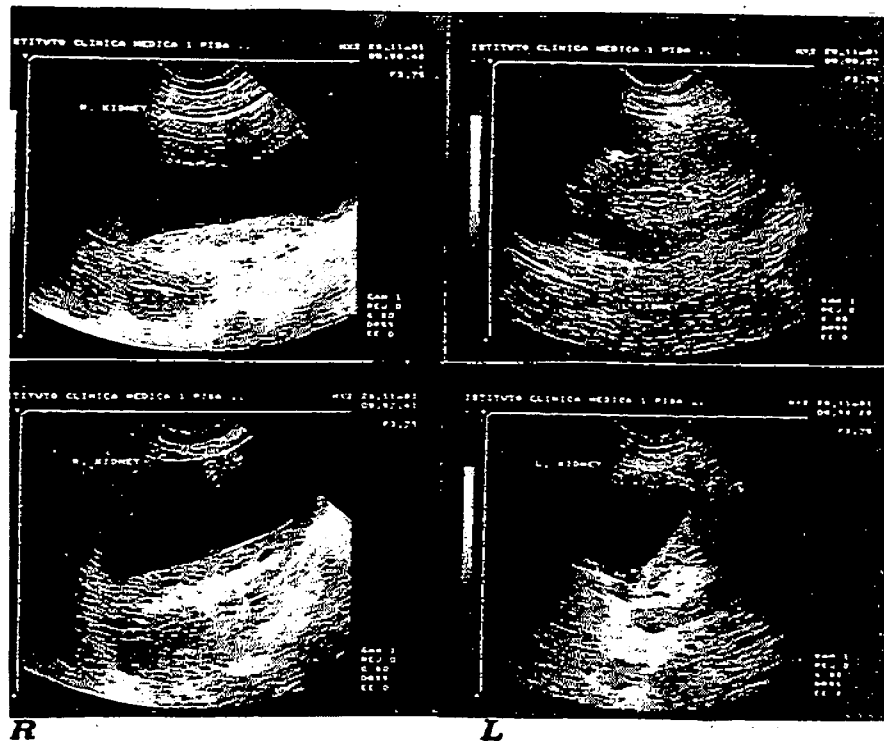


FIG. 3. Significant reduction of the perirenal effusion.

period; the restoration of normal blood values and the reduction and maintenance of the hematocrit at 48% were accompanied by a significant reduction of the perirenal effusion (Fig. 3).

### Discussion

The perinephric fluid may vary from a merely anechoic mass, which sometimes displaces the kidney, to an evident echogenic mass. Ultrasonography is known to be effective in the demonstration of perirenal fluid accumulations.<sup>4</sup> It is not always able, however, to recognize the fascial structures when the echogenic collection merges with normal echogenic fat.<sup>5</sup> Computerized tomography, on the other hand, permits a higher degree of contrast between the fat and the perirenal fascia and therefore provides the most precise anatomic information.<sup>6</sup>

If we exclude the so-called hygroma or renal hydrocele, which is a congenital cyst whose walls are composed of the renal capsule,<sup>3</sup> true perinephric fluid accumulations are most often acquired. Based on the characteristics of their content, these acquired fluid accumulations can be inflammatory, hematic, or urinary.<sup>4</sup> A perinephric abscess originates most frequently from the direct extension or rupture of an abscess within the renal parenchyma into the perinephric space. More rarely, this space may also be infected by hematic or regional lymphatic seeding from distant foci of infection (skin, liver, pancreas, colon, vertebrae). In such a case the fluid accumulation is monolateral, purulent, usually confined to the space or extending beyond Gerota's fascia into the pararenal space or along the flank muscles and toward the psoas, and directed by the renofascial septa.<sup>7,9</sup> Hemorrhagic effusions are most often the result of trauma or percutaneous renal biopsy but may also occur spontaneously in patients with arteritis, bleeding diathesis, or aneurysms.<sup>5</sup> Urinary effusion sometimes referred to as a urinoma, often

results from postoperative or posttraumatic complications and rarely from percutaneous renal stone removal, which was used prior to the advent of extracorporeal shock wave lithotripsy.<sup>10,11</sup> Small perinephric fluid accumulations are seldom demonstrated in the transplanted kidney.<sup>12</sup>

In the patient studied, all three causes can be excluded. The patient's history gives no indication of abdominal trauma, surgery, or invasive diagnostic procedures. The patient was afebrile and never presented signs or symptoms of pyelonephritis or other septic renal processes. The fluid accumulation was bilateral and confined to the perirenal space, did not communicate with other anatomic spaces of the retroperitoneum, and had typical characteristics of a transudate. The fluid accumulation appeared to be acquired, as verified by normal-appearing sonography performed several months prior to hospitalization. When aspirated, the fluid reformed maintaining its transudative characteristics, and if not drained, it tended to remain stationary and did not cause hypertension or compromise renal function significantly.

### *Conclusions*

A relation with Eisenmenger's syndrome is very probable. The transudate recurred after the drainage, but after eleven phlebotomies (each of 500 cc), with normalization of the hematocrit values, twelve months later, the transudate was remarkably reduced. When we speculate on the pathogenesis, the role of hyperviscosity due to secondary polycythemia seems unquestionable. Yet, to explain the unusual, strange event in our patient, there is the possibility that the pulmonary arterial hypertension itself can determine, by an unknown pathogenetic mechanism, a localized increase of hydrostatic pressure at the level of the venous anastomosis between the perinephric area and lumbar veins with resulting transudation of fluid from the vessel walls. The high degree of compliance of the perirenal space is due to the presence of adipose tissue; hence, the tension developed by the fluid accumulation does not compress the kidney or compromise kidney function but is sufficient to oppose the intravascular pressure since the quantity of perirenal fluid remains constant, until the hematocrit values remain high. By keeping the hematocrit values within the normal range, the local hydrostatic pressure and, consequently, the transudation, are reduced.

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