Acute Pulmonary-Renal Syndromes*

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INTRODUCTION

THERE ARE A VARIETY OF CLINICAL CIRCUMSTANCES in which the respiratory and renal systems are simultaneously involved. Periarteritis, lupus erythematosus, scleroderma, and dermatomyositis may involve the lung and kidney; however, they initially do not present with acute pulmonary-renal manifestations, and multisystem involvement is present to suggest the diagnosis. Consideration of this broad problem would have limited clinical value, but the differential diagnosis of the patient who initially presented with predominant pulmonary manifestations soon followed by severe renal involvement delimits the diagnostic possibilities.

Case examples of renal vein thrombosis, Wegener’s granulomatosis, Goodpasture’s syndrome, hemolytic streptococcal pneumonia with glomerulonephritis, and pneumococcal pneumonia with renal failure are presented to illustrate this important problem.

RENAL VEIN THROMBOSIS AND THE NEPHROTIC SYNDROME

CASE REPORT

This 31-year-old Negro enjoyed excellent health until January, 1961, when he suddenly developed severe left anterior pleuritic pain followed by hemoptysis. The next day a chest X-ray film disclosed left lower lobe infiltrate with moderate pleural effusion. Although he was afebrile and the complete blood count was normal, pneumonia was diagnosed and penicillin started. Thoracentesis removed 15 ml. of serosanguineous fluid. Urinalysis revealed 4-plus albuminuria and varying pyuria, cylindria and microhematuria. Excretory urograms were normal. On the fifth hospital day, constant severe right lower quadrant pain occurred, but on surgical exploration, only ileus was noted and a normal appendix was re-

moved. Hemoptysis increased, temperature to 102°F. appeared, the white blood cell count rose to 20,000 per mm³ and the roentgenogram now disclosed a right lower lobe infiltrate. The serum albumin was 2.4 gm. per cent. Proteinuria of 3 to 10 grams every 24 hours was present. The blood urea nitrogen was 15 mg. per cent and did not become elevated. There was no evidence of venous stasis or thrombosis in the extremities. Electrocardiogram was normal except for tachycardia.

Six weeks later, right pleuritic pain and a new infiltrate in the right lung base occurred. Severe constant left flank pain and renal colic with the passage of small blood clots were also present. Ileus, acute left flank tenderness with muscle guarding, and a left upper quadrant mass were noted. The white blood count rose to 27,000 per mm³ and the hemoglobin fell to 8.4 gm. per cent. Hypertension, 180/110, was observed, but subsided after two weeks. Excretory urograms revealed a normal-appearing right kidney and a non-functioning, slightly enlarged left kidney. The left collecting system was normal on retrograde urogram; a left ureteral catheter drained 100 ml. of urine over the next 24 hours, although the total urine flow always exceeded 1,000 ml. per 24 hours.

He was transferred to Walter Reed Hospital on March 13, 1961. The two-month history compatible with recurrent pulmonary infarctions, intermittent shifting abdominal and flank pain, loss of function and slight enlargement of the left kidney, transitory anemia, hypertension, and nephrosis suggested inferior vena caval and bilateral renal vein thromboses. Retrograde aortography was confirmatory in that normal renal arteries were visualized, but the nephrogram phase on the left was greatly delayed. Three weeks later, excretory urogram disclosed prompt excretion of the contrast material bilaterally, indicating significant return of function of the left kidney. Serial sodium o-iodohippurate (Hippuran), I¹¹3 renograms also confirmed return to a more normal blood flow.

Anticoagulant therapy, initially heparin and later coumadin, was initiated on March 14, 1961. Prednisone (Meticorten), 60 mg. daily, was given empirically for its anti-inflammatory effect; this amount was slowly tapered and discontinued after two months. Extensive studies failed to ascertain the etiology of the renal thrombosis. Four months after the onset of illness, there were no respira-

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tory symptoms and the chest roentgenogram was normal. Occasional dull aching abdominal pain was present, but the patient was fully ambulatory and had only moderate ankle edema. The nephrosis persisted with a serum albumin of 1.5 gm. per cent and a proteinuria of 10 to 20 gm. every 24 hours. The hematuria, pyuria, and cylindruria cleared. Phenolsulfophthalein test, Fishberg concentration test and creatinine clearance became normal. Serum cholesterol remained elevated at 400 mg. per cent.

Chronic anticoagulant therapy with warfarin sodium (Coumadin) was continued. Eight months after onset of illness, bilateral pleuritic pain, hemoptysis, and right flank pain recurred. Chest x-ray examination confirmed new infarctions of the right lower lobe. Abdominal exploration was performed in the hope of ligating the inferior vena cava above the area of thrombosis to prevent pulmonary embolization. Extensive venous collateral circulation was present in the pelvis. There was old retroperitoneal scarring with thrombosis of the inferior vena cava from the renal veins to the iliac veins and thrombosis of both renal veins. It was not possible to ligate the inferior vena cava above the renal veins for fear of compromising the collateral circulation, as well as the partially recanalized right renal vein. It was ligated just below the left renal vein. This resulted in persistent and more marked leg edema following surgery. Severe nephrosis persisted and the serum cholesterol rose to 800 mg. per cent. On March 8, 1962, 13 months from onset of illness, severe crushing chest pain occurred. Electrocardiogram confirmed a massive anterior wall myocardial infarction; he died 12 days later in cardiac shock.

Necropsy disclosed thromboses of both renal veins and ligation of the inferior vena cava just distal to the entrance of the left renal vein. The right kidney weighed 140 grams; the left, 190 grams. Their capsules were coursed by many blood vessels. Multiple small depressions representing old infarcts were present throughout both renal cortices. The heart weighed 300 grams and showed a recent circumferential transmural myocardial infarct from extensive atherosclerosis with thrombosis of the left anterior descending coronary artery. The right pulmonary artery was partially occluded with an organized branching embolus. Several small thrombi of varying ages were present in the smaller arteries of both lungs. Microscopically, both renal veins had old, organized canaled thrombi. Peripelvic venules and lymphatics were dilated. Renal parenchymal changes were similar throughout both kidneys and consisted of cortical scarring with small infarcts, diffuse thickening of the glomerular basement membranes, focal lobular thickening of the glomerular tufts, periglomerular fibrosis, and fibrous obliteration of some glomeruli. Mild tubular atrophy with interstitial fibrosis and focal chronic inflammatory exudate was also present. There was no evidence of arteritis about any of the many renal or pulmonary thrombi examined.

**Discussion**

The most characteristic feature of renal vein thrombosis is the sudden onset of pain of a constant, severe nature in the flank, which often radiates into the groin. Loin tenderness, muscle spasm and fullness may be observed due to renal capsule distention from the enlarged and congested kidney. There is modest fever and brisk leukocytosis. Abdominal pain may also occur and should suggest inferior vena caval thrombosis as well. Hematuria, pyuria, and cylindruria may be transient, but marked proteinuria rapidly appears and usually persists. Occasionally, pain does not occur and hypertension with albuminuria may be the only manifestation. The nephrotic syndrome can occur whether one or both renal veins are involved. Thromboembolism, especially to the lungs or legs, is frequently present and in conjunction with the above features should suggest the diagnosis. Superficial venous collateral circulation over the thighs and abdomen may follow and can be more clearly demonstrated by infrared photographs. Early x-ray studies dem-

**Figure 1:** Three weeks from onset of upper respiratory illness, left lower lobe and peri hilar infiltrate.
onstrate an enlargement with poor function of the involved kidney. Aortography will disclose the renal arteries, but clearance of the contrast material during the nephrogram phase will be greatly delayed. Inferior vena cava venography may demonstrate obstruction and absence of retrograde flow into the renal veins with the Valsalva’s maneuver. This latter procedure would seem hazardous because of increased risk of further pulmonary emboli. Sodium o-iodohippurate (Hippuran), I131 renograms will confirm loss of normal vascular flow, but will not differentiate arterial from venous obstruction. Acute renal infarction may be suggested by a non-visualized kidney on excretory urogram and a brisk rise of the SGO-T.

Diagnosis may be suspected by renal biopsy. Increased venous pressure primarily affects peritubular capillaries. The most striking changes are tubular atrophy and interstitial fibrosis. The glomeruli are disproportionately less involved and may show only diffuse basement membrane thickening. In our patient, glomerular changes were more extensive.

The rapidity and extent of the occlusion and thus the opportunity for collateral circulation determine the severity and reversibility of many of the clinical features, as well as the degree of recovery of renal function. Rapid and extensive thrombosis may lead to renal infarction and permanent loss of function. The majority of such cases have occurred during the neonatal period. The small caliber of the vessels and relative polycythemia in this age group in conjunction with severe dehydration predispose to intrarenal thrombosis with hemorrhagic infarction. This precludes effective collateral circulation and nephrosis does not follow. Hematuria, abdominal mass, and shock occur. In contrast, in the adult, thrombosis of the renal venous system is usually extra-renal and involves the renal vein following inferior vena caval thrombosis. This may occur following intra-abdominal sepsis, malignancy or trauma, heart failure and constrictive pericarditis. Femoral phlebitis may or may not be present. As in our patient and in one-half of reported cases, these predisposing factors may not be present and the cause of the thrombosis remains obscure. Occasionally, primary intrarenal venous thrombosis occurs in the adult. Previous intrinsic renal disease is present, usually amyloidosis, and there is intensification of the existing renal problem.

Most affected individuals have died within a few months of uremia, cardiac failure, pulmonary embolism, or coronary

artery disease. Our patient had persistent nephrosis without azotemia, but succumbed to a massive myocardial infarction from coronary atherosclerosis. Those surviving usually have persistent albuminuria and/or azotemia, although occasionally complete remission occurs. Chronic anticoagulant therapy is clearly indicated in the hope of preventing further thrombosis as well as embolization to the lung. In the event of acute unilateral hemorrhagic renal infarction, nephrectomy is lifesaving.

**Wegener's Granulomatosis**

**Case Report**

This 23-year-old white man was well until two months before admission to Walter Reed General Hospital. Following a mild upper respiratory infection, a morning productive cough persisted. Three weeks later, left hemithorax tightness and increased production of yellow tenacious sputum prompted taking a chest x-ray film, which disclosed a left lower lobe and left hilar infiltrate (Fig. 1). Penicillin therapy was instituted, but daily temperatures of 103°F., occasional shaking chills, dyspnea on exertion, and cough productive of one-half cup of yellow blood tinged sputum persisted. The pulmonary infiltrate in the left lung increased in size and left pleuritic pain became more severe. Subsequently, a variety of antibiotics in various combinations were given to no avail. On the 21st hospital day, agitation, disorientation and incoherent speech appeared. The following day migratory arthritis of the wrists, elbows and shoulders occurred. The blood urea nitrogen, which had been normal previously, was now 47 mg. per cent. Congestive heart failure was thought to exist and digitalis therapy was started. Prednisone, 75 mg. daily, was also begun. He became afebrile in 24 hours, and the arthritis subsided, but there was no improvement in the mental status. In spite of a urinary output of greater than 1,000 ml. every 24 hours, the blood urea nitrogen rose to 156 mg. per cent. The urinary sediment contained many red cells and hyaline casts.

He was transferred to Walter Reed General Hospital because of severe uremia. He was semistuporous with evident weight loss and muscle wasting. There was severe nuchal rigidity; the reflexes were normal and no localizing neurologic findings were observed. The spinal fluid was grossly bloody with slight xanthochromia. The progressing pulmonary lesions, severe systemic reaction with psychosis, polyarthritis, elevated gamma globulin, and renal failure suggested Wegener's granulomatosis. Therapy consisted of peritoneal dialysis and 1,600 mg. of hydrocortisone intravenously in the first 24 hours. The day after admission the patient lapsed into coma and died.

Postmortem examination disclosed two, 2-cm., thick-walled, cavitory lesions in the left lower lobe. Both kidneys were swollen, weighed 300 gm. each, and displayed multiple punctate hemorrhagic areas and many small infarcts. An intracerebral hemorrhage was present in the left temporoparietal area of the brain. On microscopic examination, the pulmonary lesions represented granulomatous necrotizing pneumonitis containing giant cells. Polyarteritis with segmental fibrinoid necrosis of the smaller arterioles and capillaries causing infarction was evident in the myocardial, pulmonary, gastrointestinal, and renal systems. There was glomerulitis and fibroid deposition within the glomeruli; numerous renal infarcts were present. Hemorrhage was present in the right temporal and parietal lobes, but vasculitis was not detected.

**Discussion**

Wegener's granulomatosis is considered to be a variant of polyarteritis nodosa. The sequence of events in this disease, i.e., respiratory tract involvement followed by widespread fibrinoid necrosis of vessels often associated with peripheral eosinophilia and increased gamma globulin, suggests a hypersensitivity phenomenon. An unknown excitant may initially cause tissue alteration within the respiratory tract with resulting autosensitization. It is more prevalent in men during the fourth and fifth decades. Multiple necrotizing granulomatous lesions occur anywhere in the respiratory tract from the nasal sinuses to the lung. Toxicity and fever are disproportionately severe. In three-fourths of the patients, nasopharyngeal involvement with purulent rhinorrhea and/or sinusitis is present. The remainder present with cough, hemoptysis, pleurisy, and single or multiple oval pulmonary infiltrates which tend to cavitate. Occasionally, only a bronchopneumonic infiltrate is present. The usual infectious agents that cause chronic pneumonitis, lung abscess, and granulomatous reactions must be considered. However, the failure to respond to appropriate antibiotic therapy and the early appearance of widespread multisystem involvement culminating in progressive renal failure should suggest Wegener's granulomatosis. Although the re-
spiratory tract involvement may be the only manifestation for several months to three years the progression of the disease is usually rapid and full of incident. Necrotizing vasculitis of the small arterioles and capillaries accounts for the arthritis, necrotizing skin and mucosal lesions, neuritis, and severe focal glomerulitis. Death follows in a few months from severe respiratory tract infection or uremia. The subarachnoid hemorrhage seen in this case has been noted previously.  

Antibiotics are successful only in controlling the secondary mixed bacterial infection that is engrained upon the destructive process. Local x-ray irradiation may be of some benefit for intranasal lesions. Steroids will definitely control many of the systemic manifestations. They were of temporary benefit in two other cases recently seen here, but both succumbed to renal failure within a two-year period.

**GOODPASTURE’S SYNDROME**  

**CASE REPORT**

A 19-year-old white man was hospitalized elsewhere in January, 1961 after five days of hemoptysis and one day of dyspnea. Chest x-ray examination revealed an infiltrate in the posterior segment of the right lobe. The white blood cell count, hemoglobin, hematocrit, and urinalysis were normal. The pulmonary infiltrate rapidly disappeared.

Hemoptysis recurred in February, 1961 for one day and again in March, 1961 resulting in hospitalization. He was pale and there were crepitant rales in the lungs bilaterally. Hemoglobin was 6.0 gm. per cent; urinalysis showed a trace of albumin with many white cells and later microscopic to macroscopic hematuria and cylindruria. A chest roentgenogram showed an extensive patchy infiltrate throughout both lung fields (Fig. 2). The hemoptysis ceased and his chest cleared in five days (Fig. 3). With oral iron therapy, the hemoglobin level improved rapidly. On May, 1961, after four days of vomiting, diarrhea and diffuse abdominal tenderness, oliguria was noted. The blood urea nitrogen had risen to 114 mg. per cent. Over the next four days, he became progressively worse with hemoptysis, epistaxis, dyspnea, tachypnea, tachycardia, pallor, and systolic hypertension. A chest x-ray film showed extensive infiltration bilaterally. Retrograde pyelography was normal. Intermittent positive pressure breathing and intravenous hydrocortisone resulted in moderate improvement, but severe hypertension and anemia persisted. Oral prednisone was also begun. Between May 24 and June 23 hemodialysis was performed 17 times for control of uremia. His urine output never again exceeded 12 ml./day.

He was transferred to Walter Reed General Hospital on June 23, 1961. The uremia was controlled by intermittent peritoneal dialysis with the blood urea nitrogen being kept below 100 mg. per cent. Prednisone, 30 mg./day, orally, was continued. He died of congestive heart failure on September 3, 1961, eight months after onset of illness.

At postmortem examination the kidneys were slightly smaller than normal and covered with petechiae. The lungs were heavy, congested, and covered with a fibrinous exudate. The heart weighed 410 gm. with a thick fibrinous pericarditis and biventricular hypertrophy. Microscopically, the lungs showed areas of recent hemorhages and numerous hemosiderin-laden macrophages. The alveolar walls were diffusely thickened. The septal walls contained intracapillary and pericapillary eosinophilic fibrinoid material, which appeared as subintimal nodules. The interlobular septa were edematous and showed diffuse, round cell infiltration. In the kidneys, there was proliferation of the epithelial cells of Bowman’s capsule, filling Bowman’s space. The glomerular tufts were obliterated by marked thickening and splitting of the basement membrane. There was interstitial fibrosis throughout the cortex.

**DISCUSSION**

In 1919, Goodpasture described a patient having similar clinical and pathologic findings, but it was not until 1955 that this syndrome was re-emphasized. Since then, more than 50 cases have been reported, occurring primarily in men under the age of 35. Recurrent hemoptysis, dyspnea, and disproportionately severe iron deficiency anemia were present and were reminiscent of idiopathic pulmonary hemosiderosis. Variable fever, chills and fatigue were present. Chest films disclose butterfly-wing type infiltrates radiating from each hilar region. These infiltrates rapidly cleared with improvement of the pulmonary symptoms. Progressive nephritis soon intervened. Death resulted from either pulmonary or renal insufficiency within one to six months and rarely beyond a year. Urinary sediment abnormalities may not be present initially, but once apparent usually persist. The ab-
rupt and often severe anemia occurs not only as a result of the hemoptysis, but also from sequestration of blood within the lung parenchyma. The lung becomes saturated with iron, but the latter is not available for utilization since it is contained within macrophages in the intra-alveolar space. The bone marrow iron stores and serum iron level are typical of iron deficiency anemia and oral iron therapy is corrective until azotemia appears. Hypertension is not a predominant feature.

At postmortem examination, the lungs are heavy, red-black, with massive exudate of blood in the alveoli. The walls of the alveoli are thickened with lining cells and hemosiderin-laden macrophages. The kidneys are enlarged and pale with petechial hemorrhages on their surfaces. There is proliferation of the parietal epithelium of Bowman's capsule with crescent formation, as well as lymphocytic interstitial infiltration. The glomeruli are in different stages of degeneration, varying from the early proliferative lesion to complete hyaline replacement. Arteritis is not observed. The etiology remains obscure.

Idiopathic pulmonary hemosiderosis has a similar clinical and pathologic pulmonary component, but it lacks the renal features. However, Heptinstall and Salmon, in a review of 69 patients with idiopathic pulmonary hemosiderosis, found five who had renal lesions at necropsy. Thus, these two syndromes may actually represent different stages of the same basic disorder. In approximately one-half of the reported cases, steroids have been tried. Fairley and Kincaid-Smith have reported three survivors using such therapy, but otherwise steroids have been to no avail. Their use after the onset of renal failure has been unsuccessful in the four cases we have seen.

**Hemolytic Streptococcal Pneumonia with Glomerulonephritis**

**Case Report**

Nine days before admission, this 14-year-old white boy developed dry cough, malaise, and fever. Four days later, a physician diagnosed "flu." A chest x-ray examination was negative. Over the next five days, progressive fever, dyspnea, right pleuritic pain, and cough productive of greenish, purulent sputum developed. His admission temperature was 105.0°F.; blood pressure, 130/90; pulse rate, 120 per minute; respiration rate, 45 per minute. He had shaking chills, flared nostrils, an erythematous facial flush, and diffuse cyanosis. The throat was severely inflamed. The right hemithorax was tender and splitting was present. White blood cell count was 20,300 per mm³ with 83 per cent neutrophils, hemoglobin, 14.1 gm.; Wintrobe erythrocyte sedimentation rate, 35, C-reactive protein, 4-plus. Many neutrophils showed toxic granulation. The urine had a specific gravity of 1.022; albumin, 3-plus, and an occasional granular cast was seen. Two days later, the urine showed only a trace of albumin. Sputum culture grew primarily beta hemolytic streptococci with a few hemolytic staphylococci. Roentgen-ray examination showed a 2 x 4 cm. infiltrate in the anterior segment of the right upper lobe. Chest x-ray film the next day disclosed moderate pleural reaction along the right lateral chest wall.

Procaine penicillin, 600,000 units intramuscularly every 12 hours, nasal oxygen, and intravenous fluids were instituted. After culture confirmed the presence of streptococci, penicillin was increased to one million units every six hours. By the second hospital day, there was no movement of the right chest, and dullness, as well as decreased breath sounds were present. The skin remained dusky and an erythematous maculopapular eruption appeared over the face and upper chest. Several attempts at thoracentesis were unsuccessful. Temperature persisted between 102° and 104°F., throughout the initial five days of therapy. However, by the fourth day, the white blood count had decreased and he appeared less toxic. Chest findings and x-ray findings remained unchanged. On the sixth day temperature fell to 101°F., and he became afebrile by the ninth hospital day. Antibiotic therapy was discontinued after two weeks. After one month, only slight pleural reaction could be seen by x-ray examination.

By the 14th day, the white blood cell count had returned to normal. The urine showed 4-plus albuminuria, innumerable red cells, and red cell casts. During the prolonged period of bed rest, there was no peripheral fluid retention or hypertension. The antistreptolysin titer was 250 units three weeks after the onset of illness, and two and six months later was 125 and 50 units, respectively. Urinalysis remained abnormal for one month but was normal at two and six months after onset of glomerulonephritis.

**Discussion**

In preantibiotic days, the hemolytic strep-
Pneumococcal Pneumonia with Acute Renal Failure

Case Report

Five days after the onset of an upper respiratory infection in a 40-year-old Negro, he developed severe right pleuritic pain, chills, and increasing fever. Two days later, physical examination disclosed a seriously ill and dehydrated patient with a temperature of 104°F., pulse, 130; blood pressure, 135/70; respiratory rate, 40 to 50 per minute. Limitation in motion and tenderness of the right anterior hemithorax with rales at both lung bases and abdominal ileus were present. Chest x-ray film disclosed bilateral lower lobe lobar pneumonia. The initial white blood cell count was 2,000 per mm. with 50 per cent polymorphonuclear cells. Sputum cultured Type IV Pneumococcus. Serial blood cultures were negative. Admission blood urea nitrogen was 50 mg. per cent; serum sodium, 124 mEq./L. Although shock was absent and hydration adequate, urine output was only 50 to 100 ml. daily during the initial four days of hospitalization. The urine had a specific gravity of 1.020, 2-plus proteinuria, and innumerable erythrocytes, white blood cells, and red blood cell casts.

Antibiotic therapy consisted of chloramphenicol (Chloromycetin) for four days followed by tetracycline for two weeks. By the fourth hospital day, control of the overwhelming infection was apparent. However, the blood urea nitrogen had risen to 180 mg. per cent and the patient had become stuporous. Hypertension to 160/100 was now present and persisted for the next three weeks. Right retrograde pyelogram was normal. A 72-hour peritoneal dialysis decreased the blood urea nitrogen to 86 mg. per cent and serum potassium, sodium, chlorides, and CO₂ returned to normal. The uremic symptoms rapidly cleared. The urine output began to rise and was 460, 1,200, and 2,000 ml. by the eighth, 11th and 13th hospital days, respectively. The blood urea nitrogen remained at 110 to 128 mg. per cent until the 15th hospital day and then rapidly fell to 15 mg. per cent over the next five days. The albuminuria and abnormal urinary sediment cleared by the third week. The serum complement and antistreptolysin titer remained within normal limits. The pneumonic infiltrates resolved within a month.

Discussion

Transitory febrile proteinuria with an increase in the urinary formed elements is often noted at the height of pneumococcal
pneumonia. These urine sediment abnormalities have been attributed to focal nephritis. Glomerular congestion, endothelial proliferation, leukocytic infiltration, and cloudy swelling of tubular epithelium, all of a spotty nature, have been described. Similar observations have been made in a variety of pyogenic infections. Older observations have indicated that pneumococcal pneumonia may be followed by more significant renal involvement (2 to 8 per cent). Abnormal urinary findings were seen during the crisis or within one to two weeks, but hypertension, edema, and azotemia were uncommon.

In the described case, the lack of history of ingestion of a nephrotoxic substance prompted initially the consideration of acute tubular necrosis, bilateral cortical necrosis, septicemia with multiple abscesses of the kidney, diffuse nephritis associated with subacute bacterial endocarditis, acute glomerulonephritis, and a hematuric exacerbation of chronic glomerulonephritis. In 100 cases of acute tubular necrosis, Blue- mle and co-workers found two associated with pneumococcal pneumonia. Tubular necrosis had also been observed with staphylococcal pneumonia. Severe infection and hypotension were common features in these reports. However, we have observed acute tubular necrosis in severe infections in the absence of hypotension. Disproportionate renal vascular spasm with ischemia was presumed to have been present. Bilateral cortical necrosis initially cannot be differentiated from acute tubular necrosis. However, irreversible renal failure regularly follows. It is usually associated with septicemia and shock from a gram-negative organism, and is part of a generalized Shwartzman-like reaction. Staphylococcal septicemia may cause multiple abscesses of the kidneys with pyuria and positive urine cultures, but is rarely associated with severe renal failure. On rare occasions, diffuse glomerulonephritis occurs with acute bacterial endocarditis. Sherry et al. reported a patient who developed a staphylococcal wound infection following a laminectomy. Several weeks later, the local infection flared resulting in a severe staphylococcal septicemia, multiple pulmonary abscesses, and acute renal failure. Necropsy disclosed a tricuspid valve endocarditis, multiple pulmonary abscesses, and an acute proliferative glomerulonephritis similar to that seen following streptococcal infections. When the tricuspid or pulmonic valves are involved by endocarditis, recognition is more difficult since heart murmurs are often absent and the source of the septicemia may not be appreciated. Its inclusion in the differential diagnosis of acute pulmonary-renal syndromes may be helpful in suggesting the source of a septic clinical course with multiple lung abscesses and abnormal urinary sediment.

In the described case, the oliguria, hypertensive, proteinuria, hematuria, red cell casts, hypertonic urine, and progressive anemia suggested acute glomerulonephritis. The red blood cell casts and the persistent hypertonic urine excluded acute tubular necrosis. Absence of gross hematuria and shock, but more important, prompt recovery from renal failure, ruled out cortical necrosis. Multiple abscesses of the kidney were excluded on the basis of the absence of septicemia, prompt control of the febrile illness, and negative urine cultures. There was no evidence of bacterial endocarditis or septic pulmonary emboli. Acute hematuric exacerbation of chronic glomerulonephritis or an initial episode of acute glomerulonephritis remained the most likely possibilities. The former seemed unlikely, since subsequent clinical evaluation did not suggest residual renal disease.

Recently, Gentile et al. have reported the findings in renal biopsies obtained 10 to 16 days from onset of pneumonias of diverse etiology. The clinical spectrum varied from acute renal insufficiency to that of no urinary abnormalities. The histologic picture was constant and could not be correlated with the clinical or laboratory studies. Fifteen of the 16 patients had thickening of the glomerular basement membrane, periglomerular thickening, and thickening...
of the arteriolar walls most compatible with membranous glomerulonephritis. It is apparent that further studies with serial renal biopsies will be necessary to clarify the role of bacterial pneumonia in the etiology of renal disease.

**Summary**

Cases were selected to emphasize the differential diagnosis of the patient who initially and predominantly manifests acute pulmonary and renal involvement. In renal vein thrombosis, pulmonary emboli commonly occur and their recognition in an individual with recent flank pain and rapidly developing nephrosis should suggest this diagnosis. Goodpasture's syndrome is seen in young men and is characterized by recurrent hemoptysis, dyspnea, transitory diffuse lung mottling on chest x-ray films, and an anemia of iron-deficiency type. Renal involvement with uremia soon follows. At necropsy, the lung shows extensive hemorrhage, hemosiderosis, and mild interstitial fibrosis. Vasculitis is not present. The glomeruli are involved with an exudative and proliferative reaction. Unlike post-streptococcal glomerulonephritis in which the involvement is diffuse and uniform, the glomerular changes vary from normal to complete obliteration. The renal picture is similar in Wegener's granulomatosis; however, this variant of polyarteritis is seen in both sexes and at any age. The upper respiratory tract is more commonly involved with a necrotizing granulomatous reaction of the nasal or sinus mucosa. When the lung is affected, single to multiple nodular lesions appear which have a tendency to cavitate. A diffuse angiitis is also present. Acute glomerulonephritis may follow beta hemolytic-streptococcal pneumonia. Usually, a mild upper respiratory tract illness is interrupted by high fever, toxicity, dyspnea, severe pleuritic chest pain, and rapidly appearing extensive pleural effusion. The acute phase is slowly controlled with penicillin, and low-grade fever and pleural pain persist for several days. Renal disease occasionally follows pneumococcal or staphylococcal pneumonia. Overwhelming sepsis with shock is usually present and tubular necrosis results. Exceptionally acute glomerulonephritis may occur.

**Zusammenfassung**


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ACUTE PULMONARY-Renal Syndromes

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SPONTANEOUS RUPTURE OF THE ESOPHAGUS

Spontaneous rupture of the esophagus should be considered in any patient in whom chest and upper abdominal pain follow a bout of retching or vomiting, or in patients sustaining a sudden increase in intra-abdominal pressure due to a variety of causes. The pertinent radiographic studies consist of an upright chest roentgenogram, a Bucky chest film, a scout film of the abdomen, and an esophagram.

The roentgen findings essential to diagnosis include mediastinal emphysema, hydrothorax or hydro pneumothorax, and spillage of contrast material through the perforation. Early roentgen diagnosis will lead to prompt surgery, a requisite for survival.


HIGH OXYGEN PRESSURE FOR RESPIRATORY DISTRESS SYNDROME

Eight newborn infants with progressive hypoxia were placed in oxygen at pressures greater than atmospheric for periods of 3 to 20 hours. These infants had the clinical picture of respiratory distress usually associated with hyaline-membrane disease. All died. In six, hyaline membranes were found at necropsy; all eight showed severe atelectasis. Arterial oxygen tension of all infants was raised when the infants were first placed in high oxygen pressure. This elevation of oxygen tension was sooner or later followed by a fall so that increasingly high ambient pressures were required to sustain arterial oxygen pressures. Little or no change was observed in pH or arterial carbon dioxide tension. Though calculated shunts minimally and briefly fell, they soon increased in high oxygen pressure.