Renal Medullary Necrosis

Friedreich first described renal medullary necrosis in 1877 in a man with an enlarged prostate and hydronephrosis. The lesion is a circumscribed, wedge-shaped necrosis of the medullary pyramids especially affecting the distal parts of the papillae. It is usually greyish-yellow in colour, and microscopically it consists of acellular necrotic material separated from the remainder of the parenchyma by a zone of acute inflammation. While in most cases there is a superimposed acute pyelonephritis, the condition may occur in the absence of infection, and the alternative name necrotizing papillitis is to be deprecated. The necrotic papillae may slough off into the urine, leaving a ragged surface at the point of separation. Most patients die of rapidly progressive renal failure, but some have survived.

The condition is described mainly in adults, and the two usual predisposing causes are diabetes mellitus and obstruction of the urinary tract, both being frequently complicated by acute pyelonephritis. More recently medullary necrosis has been described in association with interstitial fibrosis, a condition in which a contracted kidney is associated with generalized interstitial scarring, especially at the junction of the necrotic and the normal tissue. The microscopical appearance resembles chronic pyelonephritis, but these cases usually do not show infection; there is often a history of abuse of analgesics. Phenacetin is especially incriminated, but aspirin and other analgesics probably also play a part.

Renal medullary necrosis may occasionally occur in children. D. J. Davies and his colleagues have recently described 18 cases in infants. Of these only two occurred in children of over six months, and five were found during the first week of life. Thirteen children died during the first week after the onset of the illness, and their kidneys showed haemorrhagic infarction of the medulla, with no clear line of demarcation. The five who died two weeks or more after the onset showed a lesion more closely resembling the adult one; it was better demarcated, and the necrotic areas were acellular. The aetiological factors were asphyxia, dehydration, renal-vein thrombosis, septicaemia, haemorrhage, urinatry tract obstruction, and cyanotic congenital heart disease. The basic cause of the medullary necrosis appeared to be renal ischaemia due to severe, prolonged hypotension, complicated in some cases by thrombosis of the renal veins.

The cause of adult medullary necrosis appears to be more complex. Once again ischaemia is probably an important factor. It could be engendered by a raised intrapelvic pressure in cases of urinary-tract obstruction, by sclerosis of the intrarenal arteries and arterioles in diabetes mellitus, by compression of the medullary vessels from oedema in acute pyelonephritis, and by vasospasm following the hypotension due to dehydration in diabetic ketosis. Infection itself could cause some of the lesions. The aetiology of medullary necrosis in analgesic nephropathy is obscure. It is possible that a metabolite of phenacetin has a directly toxic effect. Rats given a compound structurally related to phenacetin developed a necrotic lesion of the terminal parts of the proximal convoluted tubules, and this was followed by an extensive chronic inflammatory reaction in the interstitial tissue beyond the zone of injury. Whether this is a true counterpart of the human lesion remains to be seen. In any case the relation between interstitial fibrosis and medullary necrosis is not apparent.

Nonspecific Lung Abscess

About one-half of all lung abscesses apart from those due to pulmonary tuberculosis can be neatly classified according to their antecedents. They are complications of a recognizable cause—for example, a specific pneumonia, bronchial occlusion, pulmonary infarction, or trauma. Though the rest are a mixed bag they have much in common. They often follow the inhalation of foreign material, their anatomical distribution is characteristic, and they usually resolve with thorough antibiotic therapy.

The idea of an aspiration lung abscess derives principally from the studies of Lord Brock, who showed radiologically the most likely resting-places of small quantities of iodized oil when they were inhaled in the recumbent position. These places were the posterior segment of the upper lobe and the apex of the lower lobe when the patient was supine and the axillary part of the lung when he was lying on his side—precisely the sites where nonspecific lung abscesses are most often seen. Some confirmation that lung abscesses arise in this way comes from the frequent finding that the bacteria causing them are often the same as those associated with dental sepsis, upper respiratory infections, and preceding nasopharyngeal operations in patients who developed such abscesses.

The inception of an acute, febrile illness with a profuse mucopurulent sputum and radiological evidence of cavitation is ordinarily sufficient for a diagnosis of lung abscess, the abscess having arisen from suppuration and necrosis of a pulmonary process. Indeed, a lung abscess is inevitably part of a pneumonia, and either may predominate. In the early stages no more than an aspiration pneumonia is seen. If necrosis is then widespread, a lung abscess is diagnosed; alternatively a “spreading supplicative pneumonia” may develop. When the aspired material is liquid or semifluid, such as vomit, a diffuse bronchopneumonia with a predisposition to abscess formation may occur. H. Nicholson favours the term “nonspecific supplicative pneumonia” for the whole group once necrosis has started.

Several recent American articles have referred to “primary lung abscess,” but this would seem to be an undesirable choice of words. Admittedly the infection of infected material

1 Friedreich, N., Virchows Archiv fur pathologische Anatomie und Pathologie, 1877, 69, 308.
7 British Medical Journal, 1965, 1, 673.