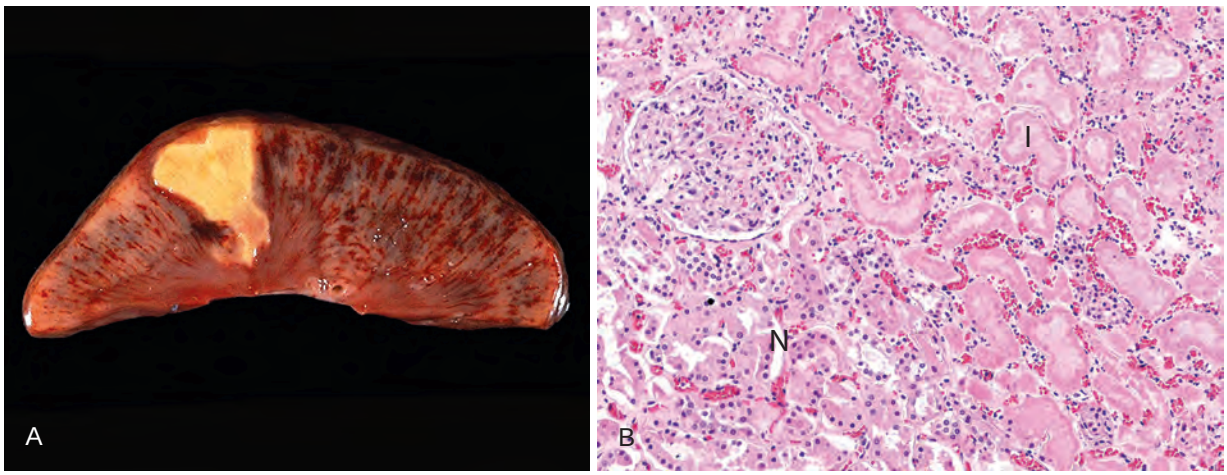

Figure 2-11 Coagulative necrosis. **A,** A wedge-shaped kidney infarct (yellow). **B,** Microscopic view of the edge of the infarct, with normal kidney (N) and necrotic cells in the infarct (I) showing preserved cellular outlines with loss of nuclei and an inflammatory infiltrate (seen as nuclei of inflammatory cells in between necrotic tubules).

Patterns of Tissue Necrosis

The discussion of necrosis has focused so far on changes in individual cells. When large numbers of cells die the tissue or organ is said to be necrotic; thus, a myocardial infarct is necrosis of a portion of the heart caused by death of many myocardial cells. Necrosis of tissues has several morphologically distinct patterns, which are important to recognize because they may provide clues about the underlying cause. Although the terms that describe these patterns are somewhat outdated, they are used often and their implications are understood by pathologists and clinicians.

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Coagulative necrosis is a form of necrosis in which the architecture of dead tissues is preserved for a span of at least some days (Fig. 2-11). The affected tissues exhibit a firm texture. Presumably, the injury denatures not only structural proteins but also enzymes and so blocks the proteolysis of the dead cells; as a result, eosinophilic, anucleate cells may persist for days or weeks. Ultimately the necrotic cells are removed by phagocytosis of the cellular debris by infiltrating leukocytes and by digestion of the dead cells by the action of lysosomal enzymes of the leukocytes. Ischemia caused by obstruction in a vessel may lead to coagulative necrosis of the supplied tissue in all organs except the brain. A localized area of coagulative necrosis is called an **infarct**.

Liquefactive necrosis, in contrast to coagulative necrosis, is characterized by digestion of the dead cells, resulting in transformation of the tissue into a liquid viscous mass. It is seen in focal bacterial or, occasionally, fungal infections, because microbes stimulate the accumulation of leukocytes and the liberation of enzymes from these cells. The necrotic material is frequently creamy yellow because of the presence of dead leukocytes and is called **pus**. For unknown reasons, hypoxic death of cells within the central nervous system often manifests as liquefactive necrosis (Fig. 2-12).

Gangrenous necrosis is not a specific pattern of cell death, but the term is commonly used in clinical practice. It is usually applied to a limb, generally the lower leg, that has lost its blood supply and has undergone necrosis (typically coagulative necrosis) involving multiple tissue planes. When bacterial infection is superimposed there is more liquefactive necrosis because of the actions of degradative enzymes in the bacteria and the attracted leukocytes (giving rise to so-called **wet gangrene**).

Caseous necrosis is encountered most often in foci of tuberculous infection (Chapter 8). The term "caseous" (cheese-like) is derived from the friable white appearance of the area of necrosis (Fig. 2-13). On microscopic examination, the necrotic area appears as a structureless collection of fragmented or lysed cells and amorphous granular debris enclosed within a distinctive inflammatory border; this appearance is characteristic of a focus of inflammation known as a **granuloma** (Chapter 3).

Fat necrosis is a term that is entrenched in medical parlance but does not in reality denote a specific pattern of necrosis. Rather, it refers to focal areas of fat destruction, typically

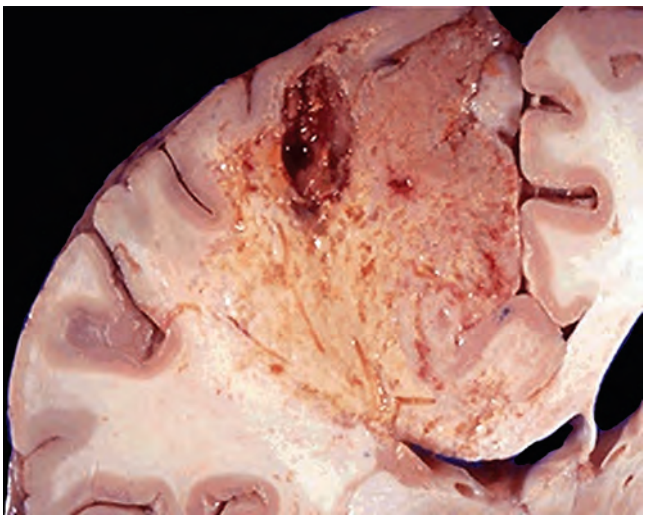

Figure 2-12 Liquefactive necrosis. An infarct in the brain, showing dissolution of the tissue.