<table>
<thead>
<tr>
<th>Types</th>
<th>Causes</th>
<th>Gross</th>
<th>Features</th>
<th>Histological Appearance</th>
</tr>
</thead>
</table>
| Coagulative Necrosis | • Due to **Hypoxia/Ischemia** that doesn’t involve<br>  
  o Severe trauma<br>  
  o Toxins<br>  
  o Acute/chronic immune response<br>  
  • Virtually happens in all part of ischeamic organs other than Brain<br>  
  (it is Liquefactive necrosis) | Pale in color, later turn into red during inflammatory response takes part<br>  
  • Dry in cut surface | • Paler staining tissue<br>  
  • **Absence of nuclei (ghost cells)**<br>  
  • Tissue architecture is remained intact<br>  
  • **Inflammatory cells infiltrate** | ![Myocardial Infarction](image1.png) |
| Liquefactive Necrosis | • **Hypoxia/Ischemia** of the Brain tissues<br>  
  • **Fungal and bacterial infection** of the CNS | The tissue is grossly liquify | ![Brain Infarction](image2.png) |
| Gummatous Necrosis | • **Spirocheatal infection**<br>  
  o Long standing Tertiary Syphilis | Soft, non-cancerous growth<br>  
  • Necrotic center with Hyalinization | • Necrotic center same like Coagulative necrosis<br>  
  • **Hyaline deposition**<br>  
  • Numerous inflammatory cells infiltrates (**Giant cells**)<br>  
  • **Fibroblastic ring** surround the necrotic tissues | ![Syphilitic Gumma](image3.png) |
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| Hemorrhagic Necrosis | • Blockage of the venous drainage of an organ or tissue  
  ○ Testicular torsion | Gross  
  • Gross hemorrhage  
  • Reddish in color  
 Microscopy  
  • Numerous Erythrocytes sequestration  
  • Engorgement of veins  
  • Numerous Hemosiderin-laden Macrophages | Pulmonary Hemorrhage |
| Caseating Necrosis | • Most commonly due to Tuberculous infection  
  • Can also be due to  
  ○ Fungal infection  
  ○ Spirocheatal infection | Gross  
  • Chess like appearance  
  • Whitish to yellowish in color  
  • Soft and friable  
 Microscopy  
  • Loss of tissue architecture  
  • Proteinaceous cellular debris  
  • Amorphous necrotic center  
  • Numerous inflammatory cells (Giant cells)  
  • Fibroblastic rings surround the necrotic center | Pulmonary Tuberculosis |
| Fatty Necrosis | • Due to action of Lipases on Adipose tissues in  
  ○ Acute pancreatitis  
  ○ Breast tissue necrosis | Gross  
  • White chalky deposits due to formation of soap  
 Microscopy  
  • Soap deposit (TG interact with calcium)  
  • Numerous Touton Giant Cells | Juvenile Xanthogranuloma |
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<td>Fibrinoid Necrosis</td>
<td>• Immune-mediated vascular damage</td>
<td>• Numerous <strong>Eosinophils infiltration</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>o Infective endocarditis</td>
<td>• Amorphous, basic, proteinaceous material in the tissue matrix with a</td>
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<tr>
<td></td>
<td></td>
<td>o Henoch-Schönlein purpura</td>
<td>staining pattern <strong>reminiscent of fibrin</strong></td>
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Churg-Strauss Syndrome
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<tr>
<th>Infarct</th>
<th>White Infarct</th>
<th>Red Infarct</th>
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<tr>
<td><strong>Causes</strong></td>
<td>• Arterial occlusion</td>
<td>• Venous occlusion</td>
</tr>
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</table>
| **Features** | • Referred as white due to lack of Erythrocytes accumulation  
  • **Pyramid shape necrosis**  
    o Apex to occluded artery  
    o Base at periphery  
  • The area of necrosis is coagulative  
  • Can become red infarct when reperfusion occurs | • Referred as red due to massive Erythrocytes accumulation  
  • Consist numerous fibrin strands  
  • **Irregular shape necrosis (often)** |
| **Organs Involved** | • Solid organs with no dual arterial blood supply such as  
  o Heart  
  o Spleen  
  o Kidneys  
  • This is because solid organ may limit the amount of hemorrhage that can seep into the area of ischemic necrosis from adjoining capillary beds | • Loose organs with dual circulation  
  o Lungs  
  o Kidneys  
  o GIT  
  o Brain  
  • The loose tissue enables Erythrocytes to seep during injury and accumulate inside the tissue |
| **Gross Appearance** | ![Kidney Infarct](image1)  
  ![Lungs Infarct](image2) | |