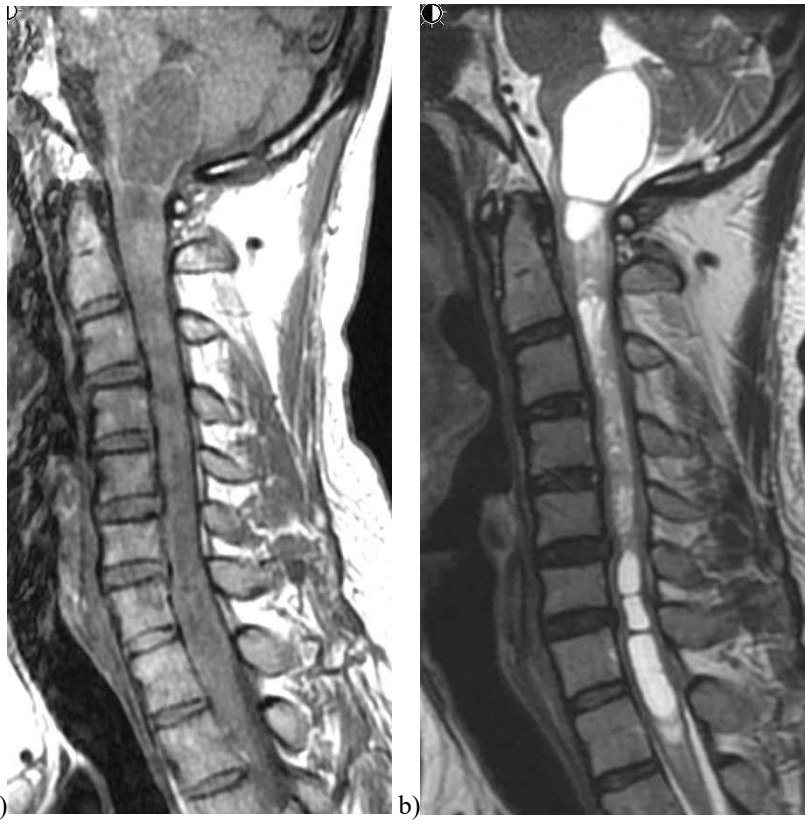
**Example 3.** Ependymoma is a glial tumor in the central nervous system, which includes the brain and the spinal cord. The increased water content or microvascular blood volume associated with tumor angiogenesis makes the tumor hypointense on T1w (Fig.3.27a) and hyperintense on T2w (Fig.3.27b), an MRI feature common to many tumors.



a) b)

**Fig.3.27.** Ependymoma. a) T1w, and b) T2w.

**Example 4**. Intracerebral hemorrhage (ICH) stages are defined on MRI using T1w and T2w imaging in standard radiology textbooks (Table 3.3, Fig.3.28). We encountered subacute ICH in Fig.3.5. The most common risk factor for ICH is hypertension; other factors including aneurysm and anticoagulation medication. The violent onset of ICH causes physical disruption of the brain's cellular architecture. The ICH mass increases intracranial pressure (mass effect), compressing brain regions including blood flow and causing brain herniation, primary brain injury. The primary injury of ICH initiates a cascade of toxic events including inflammation and release of blood degradation products, causing secondary injury. Unfortunately, we know only the devastating damage of ICH with high mortality (about 40% at 1 month), but little of ICH damage process. There is little treatment beyond surgical release of brain pressure when the ICH volume continues increasing.
MRI offers a valuable tool to investigate ICH. Because of the paramagnetic blood degradation products, gradient echo (T2*w) MRI is the most sensitive method to image ICH, more sensitive than the commonly used CT that is more available to emergent patients than MRI. ICH signal intensities on T1w and T2w vary with stage.

**Table 3.3**. Intracerebral hemorrhage stage and appearance on T1w and T2w.

| Stage | Time | Hemoglobin | T1w | T2w |
|---|---|---|---|---|
| Hyperacute | <1d | Oxy+deoxy | Iso | Some hyper |
| Acute | 1-3d | Deoxy | Iso | Hypo |
| Early subacute | 3-7d` | Meta, in RBC | Hyper | Hypo |
| Late subacute | 7-14d | Meta, free | Hyper | Hyper |
| Chronic | >14d | Hemosiderin | Hypo | Hypo |

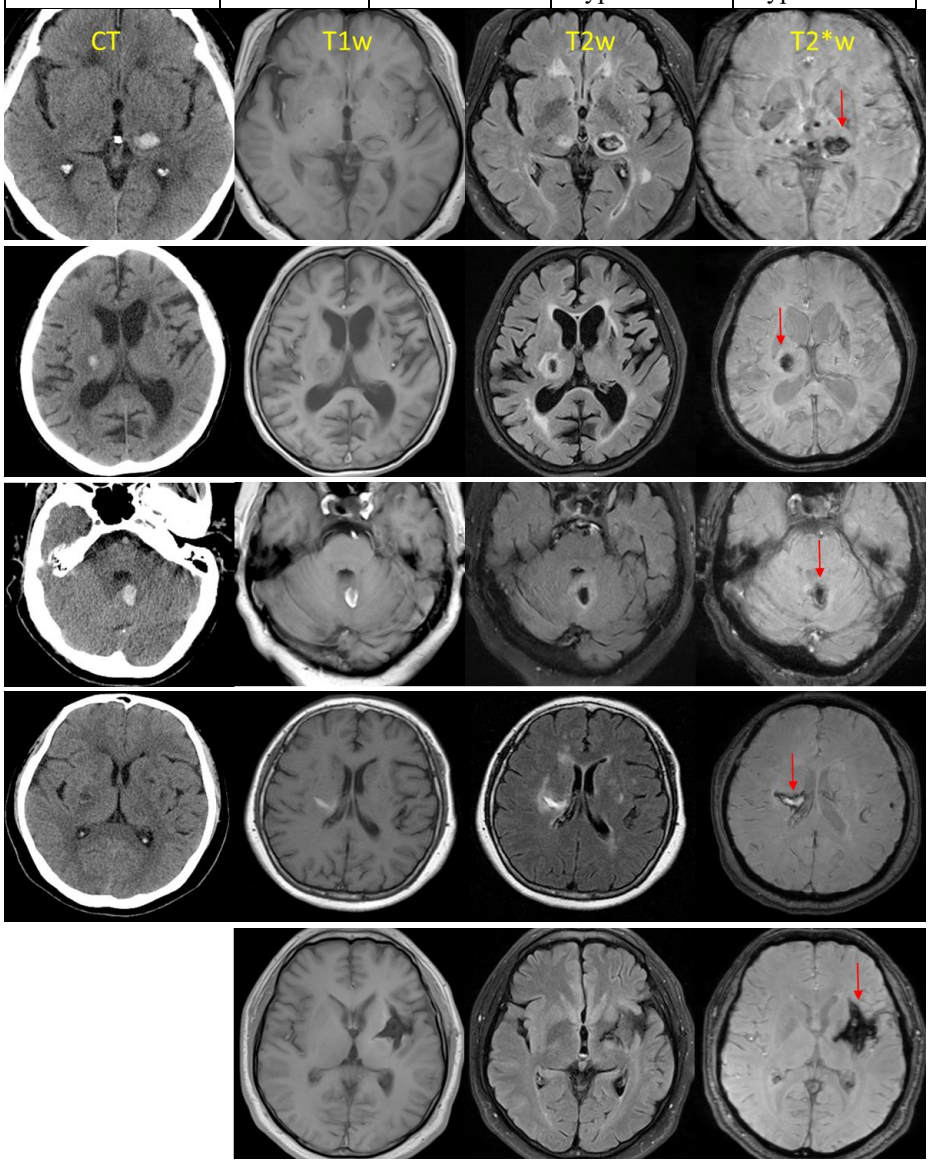



**Fig.3.28**. ICH at hyperacute (1st row), acute (2nd row), early subacute (3rd row), late subacute (4th row), and chronic (5th row) stages imaged with CT, and MRI with T1w, T2w and T2*w. These images are interpreted as follows:
**1st row, hyperacute ICH**. Oxyhemoglobin converts to deoxyhemoglobin(dHb) within a few hours of ICH onset. dHb already appears at hyperacute stage MRI, as paramagnetic dHb causes hypointesity on T2*w (also see Chapter 5). There is little

T1 enhancement by dHb that is confined with little mobility in red blood cells (RDB), leading to isointensity on T1w and some hyperintensity (ICH core) on T2w. This ICH has a complex rim, possibly composing edema and high dHb concentration.

**2nd row, acute ICH**. Abundant dHb causes strong 1/T2 that depends on the static interaction energy or dHb concentration. dHb with little mobility causes little T1 enhancement, leading to isointense on T1w and hypointense on T2w.

**3rd row, early subacute ICH**. Now dHb has turned into more paramagnetic metahemoglobin (mHb) and causes some T1 enhancement; perhaps some RBCs are lysing with some mHb gaining mobility. This leads to hyperintensity on T1w, in addition to hypointensity on T2w, for ICH at the early subacute stage.

**4th row, late subacute ICH**. During subacute stage, the RBCs in ICH lyse, and their contents and damaged neural cells get removed rapidly by macrophages. ICH turns into a lesion with little cellular contents, becoming undetectable on CT and hyperintense on T2w. The residual mHb (or released iron), though diminished in concentration, is highly mobile, causing significant T1 shortening but only marginal T2 shortening (similar to Gd effects on shortening blood T1 and T2). Therefore, late subacute ICH is also hyperintense on T1w.

**5th row, chronic ICH**. Residual mHb/iron in ICH turns into highly or super paramagnetic hemosiderin with magnetic domain. Chronic ICH becomes ultrashort T2 species with little signal on standard MRI. Edema may be resolved. Therefore, chronic ICH is hypointense on both T1w and T2w.