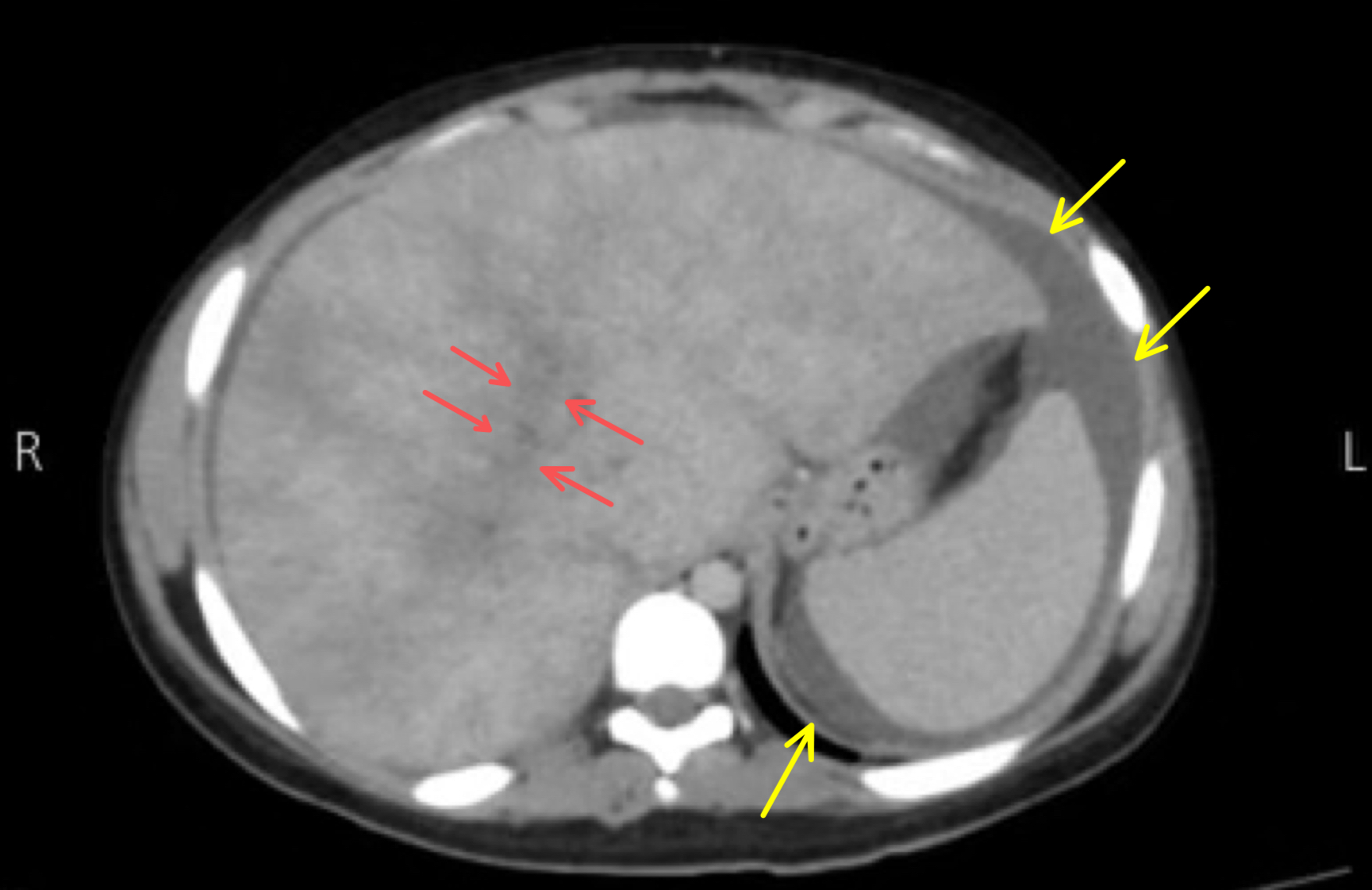
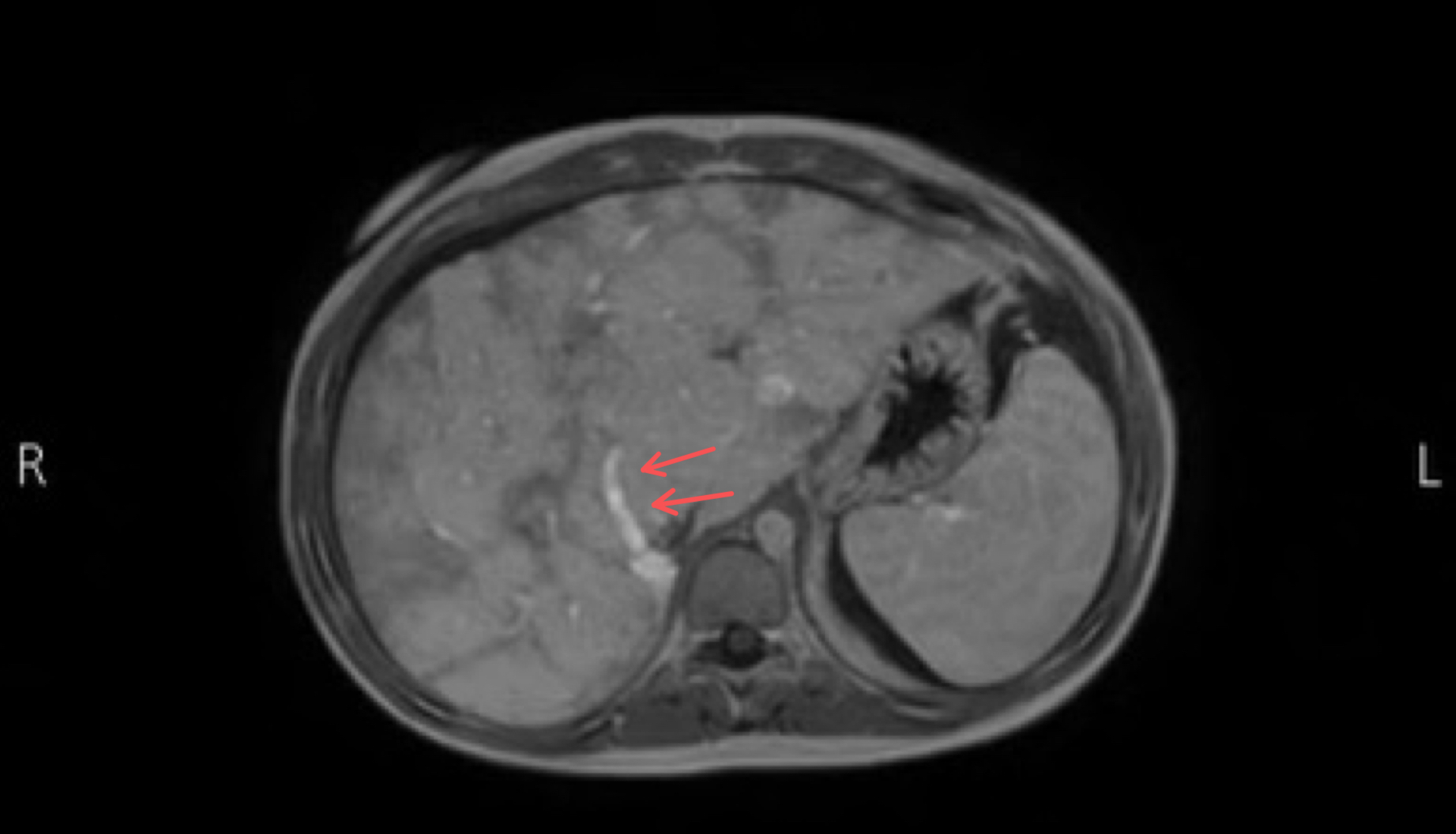
**Figure 1:CT scan with contrast reveals an Occluded intrahepatic segment of the IVC and hepatic veins, which are relatively small in caliber (Red arrow), Large ascites (yellow arrow).**

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**Figure 2: Liver MRI with contrast presents partial recanalization after long treatment by anticoagulant.**

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**Table 1: Results of Thrombophilic Genetic Testing**

|  |  |
| --- | --- |
| **Genetic factor** | **Results** |
| MTHFR (Ala1298Cys) | Heterozygous |
| Factor V Leiden (FV Arg506Gln) | Heterozygous |
| MTHFR (C677T) | Heterozygous |
| PAI-I 5G/4G | Heterozygous |
| MTRR (Ala66Gly) | Heterozygous |

**Table 2: Demonstrates the Most common etiologies of BCS**

|  |  |
| --- | --- |
| **Etiology** | **Notes** |
| **Myeloproliferative Disorders (MPD)** | The leading cause of BCS. This occurs due to the associated hypercoagulability. Polycythemia vera constitutes 10%-40% of MPD-related cases, while essential thrombocythemia and myelofibrosis are fewer common contributors (6,8). |
| **Malignancy** | Responsible for 10% of cases. The mechanism can be direct, through vessel compression or invasion, or indirect by inducing hypercoagulability. The most common cancer associated with BCS is hepatocellular carcinoma (6). |
| **Pregnancy and oral contraceptives’ use** | By inducing hypercoagulability, they account for 20% of BCS cases (6). |
| **Liver lesions** | These space-occupying conditions compress the vasculature directly resulting in obstructive BCS. Such lesions include hepatic cysts, adenomas, amoebic and pyogenic liver abscesses (6,8). |
| **Inherited thrombophilia** | These include deficiencies in protein C, protein S, and antithrombin III, as well as the Factor V Leiden (FVL) mutation. The FVL mutation is the most prevalent among them (8). |
| **Idiopathic** | 20% of cases are idiopathic (6). |