Imaging findings of cerebrovascular complications of infective endocarditis

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Learning objectives

The objective of this presentation is to describe the spectrum of cerebrovascular complications (CVC) related to infective endocarditis (IE) observed with CT and MR imagings.

Imaging findings are described with some illustrative cases of patients admitted to our institution with IE diagnosis according to modified Duke Criteria between April 2010 and January 2011.
Background

Despite improvements in health care other the past decades, and however Infective Endocarditis (IE) is a rare disease, it remains a severe affection still associated with high rates of morbidity (20-40% of neurological complications) and a high rate of inhospital mortality (20% ; until 40% at 5-year)\(^1\text{--}^4\).

This apparent paradox seems to be explained by a progressive evolution in risk factors for IE with the emergence of new risk factors. These include intravenous drug use, elderly patients with valve sclerosis, patients with prosthetic valves, those with intravascular prostheses, patients exposed to nosocomial disease, and haemodialysis patients, while chronic rheumatic heart disease is now rare in industrialised countries. Staphylococci and oral streptococci accounted for most cases of disease\(^1\text{--}^4\).

The neurological complications of IE are mostly due to the propensity for heart valvular vegetation to embolize to different organs, leading to cerebrovascular complications (CVC) in brain\(^1\text{,}^2\text{,}^3\). The more frequent CVC include\(^1\text{--}^4\):
- ischemic stroke or transient ischemic attack,
- microbleeds (= micro-hemorrhage),
- intracerebral hemorrhage,
- subarachnoidal hemorrhage,
- mycotic aneurysm,
- brain abcess,
- meningitis,
- and encephalopathy.

CVC in IE contributes to its poor prognosis and may influence the management of patients with IE in several ways\(^2\text{,}^5\). Their presence as minor criteria in Duke classification can help for diagnosis in patients without sustained bacteremia and valvular vegetation. In those patients, nonspecific clinical presentation could delay appropriate diagnosis and urgent therapy. Besides, their presence can also markedly affect medical or surgical therapies, for instance, by changing the type and duration of antibiotic and/or anticoagulation therapies. In the same way, diagnostic of acute brain embolization in patients with large valvular vegetations may lead to urgent valvular surgery during the acute phase of the disease. Alternatively, intracerebral hemorrhage is a transient contraindication for surgery.

Clinically apparent CVC is estimated to occur in 10% to 30%, mostly represented by acute brain embolization with ischemia or transient ischemic attack. Since, the clinic of CVC in IE may be confusing or silent, particularly for brain embolization and microbleeds, its true incidence on the basis of the clinical diagnosis of neurological events remains substantially underestimated\(^2\text{,}^4\text{,}^5\).
Numerous studies have reported the high rate of silent CVC (20-30%), increasing the overall frequency of both symptomatic and asymptomatic lesions (60-80%), by systematic use of cerebral imaging during acute IE. Those results were consistent with previous autopsy studies of proven IE.

Recent studies have suggested the importance and the benefits of performing routinely CT and/or MRI examinations of brain, leading to substantial changes in diagnostic classification or clinical decisions for patients with IE. Those studies have also shown the higher sensitivity of MRI method in comparison to CT examination which is relatively insensitive for the detection of acute stroke or microbleeds.5-10

In the next section of this presentation, the MR and CT findings of most frequent CVC in IE patients are described after a short reporting on epidemiological features. CT and MR images from patients admitted to our institution with IE diagnosis according to modified Duke Criteria are presented as illustration.
ACUTE CEREBRAL ISCHEMIA AND INFARCTION

The major causes of stroke in IE are embolism by migration of a fragment or of the whole vegetation in the cerebral circulation, and mycotic aneurysm rupture. Ischemic stroke is the maincause of death after congestive failure in IE. Clinically apparent acute embolic stroke is estimated to occur in 9-42% of patients with IE. While transient ischemic attack is reported to occur in 1-17% of cases and it preceds the ischemic stroke in 25% of patients with IE. However, the true prevalence of brain embolization in patients with IE is substantially underestimated without systematic brain exploration by imaging modalities. Recent studies based on the systematic recourse to neuroimaging (with MRI++ and/or CT) have shown that silent (subclinical) embolic stroke accounts for lesions in 30-70% of patients with IE in whom clinical neurological evaluation is normal. Therefore, those findings lead to increase the overall incidence of acute brain embolization (symptomatic or not) at about 80%.

Imaging findings of ischemic stroke as complication of IE do not differ from other causes of ischemic stroke, apart from the more frequent association with cerebral microbleeds, subarachnoid or intracerebral hemorrhage at early phase.

CT findings

• Non Enhanced CT (NECT) may show hyperdense vessel caused by acute thrombus ("Dot sign" = occluded MCA branches in sylvian fissure), loss of gray-white matter distinction (in first 3-4 hrs), obscuration of deep nuclei, loss of insular "ribbon", and parenchymal hypodensity (see Fig 1).

• Contrast Enhanced CT (CECT) may demonstrate enhancing cortical vessels (= slow flow or collateralization acutely), absent vessels (= occlusion), Cortical/gyral enhancement (after 48-72 hrs).

• Perfusion CT determines cerebral blood volume (CBV) and/or cerebral blood flow (CBF) (see Fig 2), mean transit time (MTT) (see Fig 3), and assess ischemic core versus penumbra (see Fig 4).

• CT Angiography (CTA) may identify occlusions, stenoses, status of collaterals (see Fig 5).
MR findings

• Early cortical swelling may be seen on both T1WI and T2WI, as a hypointense area with loss of gray-white borders with the first technique (see Fig 6) and as a hyperintense area with the latter technique (see Fig 7).

• FLAIR may be positive (hyperintense) when other sequences are normal (as early as 6 hrs post-ictus) (see Fig 8). MR intra-arterial hypersignal on FLAIR is an early specific sign of major vessel occlusion.

• DWI improves the focal stroke detection by showing restricted diffusion (low apparent diffusion coefficient - ADC-) in cytotoxic edema (see Fig 9).

• T2* GRE image may show an acute blood products highly suggestive of a septic embolic origin for the brain infarction. It may also show thrombosed vessel as arterial "blooming" from clot susceptibility (see Fig 10).

• Gadolinium contrast Enhanced T1WI may show variable enhancement patterns over time, including intra vascular enhancement (immediate), meningeal and gyriform enhancement (early), or parenchymal enhancement (late acute).

• MR Angiography (TOF or Gadolinium enhanced) demonstrates vessel occlusions, stenoses, collateral status (see Fig 11).

• MR Spectroscopy may identify elevated lactate, decreased N-acetyl-aspartate (NAA), and at mid TE (e.g. 135) lactate doublet inverts.

• Perfusion MRI (T2* Gadolinium perfusion imaging) with rCBV map may show larger abnormality than DWI abnormality. With arterial input this technique allow to calculate rCBF, rMTT.

MICROBLEEDS

Cerebral Micorbleeds (CMBs) are small parenchymal microhemorrhages highly associated with IE. CBMs may reflect a subacute inflammatory and/or infectious microvascular process leading in some cases to the development of mycotic aneurysms of distale or pial arteries. The prevalence of CMBs might vary with the type of mico-organism (untill 57% in some series without predominant micro-organism).
They may be associated with cerebral infarction, intracerebral or subarachnoidal hemorrhages, and with mycotic aneurysms (ruptured or not).

MRI findings

• CBMs are only detectable by MRI with $T2^{*}WI$ (hardly visible on the other MR images or on CT). They appear as a focal hypointense lesions (micronodular, "dot"-shaped) and are mostly homogeneous and <5mm. They are predominantly located in cortical areas and in cortical sulci than in subcortical areas (see Fig 12-13-14). They can be observed as well in supra tentorial brain as infra tentorial brain.$^{9,13}$

INTRACEREBRAL HEMORRHAGE

Intracerebral hemorrhage (ICH) occurs in about 5-7% of patients with IE and is usually lobar. In IE, ICH is usually attributed to ruptured mycotic aneurysm or (less frequently) following cerebral infarction. However, non-aneurysmal hemorrhages are well known and described, but their pathogenesis remains unclear. Some advanced hypothesis include septic erosion of the arterial wall resulting in rupture, rupture of a venous or capillary vessel, and focal arteritis without the presence of a well-delineated aneurysm.$^{2,4}$ They are variable in size (small to large) and shape (ovoid to rounded), but they prevail in supratentorial brain. Staphylococcus aureus carries a high risk of bleeding and accounts for most cases of ICH.

CT findings

• $NECT$ shows at acute phase a hyperdense mass (50-70UH), usually lobar, associated or not with mass effect. The hematoma becomes isodense then hypodense at subacute phase (1-6 weeks) and chronic (>6 weeks) phase respectively, and the mass effect decreases. Associated SAH adjacent to the hematoma or acute brain infarction may be seen, suggestive of complications of IE (Fig 15-16).

• $CECT$ may show an active bleeding, as a contrast pooling within the hematoma at acute phase. A peripheral "ring"-enhancement may be observed within a few days in vascularized capsule of the ICH and it disappears usually in 2-6 months.

• $CTA$ may identify infectious aneurysms adjacent to the ICH.

MR findings
• On *T1WI* and *T2WI*, the signal of the ICH varies with its composition, specially according to the different stages of hemoglobin (Hbg) degradation, as reported in the following table (see Fig 117-18 : hematoma at subacute early-late stages)\(^{13}\).

<table>
<thead>
<tr>
<th>Stages</th>
<th>T1(^{†})</th>
<th>T2(^{†})</th>
<th>T2(^{*})(^{†})</th>
</tr>
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<tbody>
<tr>
<td>HYPERACUTE</td>
<td>hypo-/iso-</td>
<td>hyper-</td>
<td>hypo-</td>
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<tr>
<td>ACUTE</td>
<td>hypo-/iso-</td>
<td>hypo-</td>
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<td>SUBACUTE-early</td>
<td>hyper-</td>
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<td>SUBACUTE-late</td>
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<tr>
<td>CHRONIC-early</td>
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<tr>
<td>CHRONIC-late</td>
<td>hypo-/iso-</td>
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</table>

**Intracerebral hemorrhage Staging based on T1WI and T2WI appearances**\(^{14}\).

\(^{†}\) Signal in intensity.

• *DWI* shows findings parallel that of T2WI, apart from more marked susceptibility artefacts. It may also demonstrate associated ischemic foci, highly suggestive of complicated IE.

• As with *CECT*, Gadolinium enhanced *T1W* may show a "ring"-shaped enhancement in the vascularized capsule of the hematoma.

• *MRA* may demonstrate infectious aneurysms adjacent to the ICH.

**SUBARACHNOIDAL HEMORRHAGE**

Subarachnoid hemorrhage (SAH) in IE as a presenting feature or after the start of antibiotic therapies ar rare. Most cases of SAH in IE are related to the rupture of a mycotic aneurysm occuring after the infection has been controlled. In cases of SAH as early manifestations of IE, no mycotic aneurysm may be identified\(^{2,4,12}\). SAH prevail in supratentorial compartement\(^{12,13}\).

Extraventricular Obstructive Hydrocephalus (EVOH) often occurs as a complication of SAH.
CT findings

- **NECT** is positive in 95% in first 24h, <50% by 1 week, and shows hyperdense cerebrospinal fluid (CSF) in cortical sulci and cisterns. An associated EVOH may be seen (see Fig 19).

- **CTA** may show arterial narrowing or occlusion adjacent to the SAH.

MR findings

- Blood in sulci or cisterns appears isointense to brain ("dirty" CSF) on **T1WI**, hyperintense on **T2WI** and **FLAIR**, and hypointense on **T2*GRE WI** (see Fig 20-21). Those features on **T1WI** and **T2WI** are not specific for SAH.

- **DWI** may show associated multifocal restrictions related to associated acute embolic stroke or vasospasm.

- **Gadolinium enhanced T1WI** may show cortical ant pial enhancement adjacent to the SAH, related to associated meningitis.

- **MRA** may show arterial narrowing or occlusion adjacent to the SAH.

**INTRACRANIAL INFECTIOUS ANEURYSM**

Intracranial infectious aneurysms (IA or Mycotic Aneurysm or Pseudoaneurysm) account for 0.7-6.6% of all intracranial aneurysms. They are present in 1-5% IE patients and may be multiple in 30% of cases. However, they remains a rare but potentially life threatening condition2,4.

IA is characterized by a focal arterial dilatation not contained by layer(s) of normal arterial wall. In IE, the destruction of the vessel wall secondary to infection and/or inflammation of a segment of the artery leads to the occurrence of the IA. Spread of infection is mostly endovascular. However and rarely, IA may also occur via extravascular contiguous spread from a leptomeningitis or a parenchymal abscess adjacent to the segment of the artery.

Their evolution is unpredictable even after start of antibiotic therapy (regression, development de novo, or rupture).

In IE, IA are mostly encountered in the distal branches of the Circle of Willis (50% middle cerebral artery, 25% anterior cerebral artery, 25% vertebrobasilar aretries). They are
variable in size and morphology (irregular/lobulated arterial outpouching), usually more irregular than typical saccular aneurysm\textsuperscript{6,12,13}.

**CT findings**

- **NECT** may show focal hematoma or SAH adjacent to the parent vessel.
- On **CECT** enhancing focus, or « dot », can be visualized within the hematoma or the SAH.
- CTA identifies contrast accumulation and the communication of this latter with the true lumen of parent vessel (see Fig 22-23-24).

**MR findings**

- ICH or SAH may be visible as usually hypertense on **FLAIR**, hyperintense on **T1WI** at subacute phase, hypointense on **T2*GRE** images. The signal of ICH or SAH varie with the age of the bleeding on T2WI, but usually hyperintense at acute phase (see § INTRACEREBRAL HEMORRHAGE).
- **DWI** usually demonstrates susceptibiltiy artefact (due to the bleeding) and may show associated ischemic foci.
- **MRA** (contrast-enhanced MRA recommended) may identifie the contraste accumulation and the communication of this latter with the true lumen of parent vessel.

**BRAIN ABSCESS**

Pyogenic brain abscess is a rare complication of IE and may occur in 0.5-4% of IE patients (1-8.6% of neurological complications). It results from hematogenous spread of the infection process and is characterized by four pathological stages: early cerebritis, late cerebritis, early capsule and late capsule. Microscopic, usually multiple, brain abscesses are more frequent that macroscopic abscesses (about 0.9-4% vs 0.5% respetively). In particular group of IE patients, as parenteral drug users, they tend to be more frequent (2-4%) and macroscopic. Associated ischemic stroke lesions or CBMs are common. They usually are associated with an uncontrolled sepsis or a delayed diagnosis, and contribute to the poor prognosis of IE. Staphylococcus aureus (+++) and Streptococcus pneumonie accounted for most cases of brain abscesses in IE patients\textsuperscript{1,2,4}. 
Imaging findings of brain abscess vary with stage of abscess development, but prevail in anterior and middle cerebral arteries territory, at grey-white matters junction (hematogenous spread) and in supratentorial brain\textsuperscript{13}.

**CT findings**

- Early *NECT* of brain may be normal or may show an ill-defined hypodense subcortical lesion in early cerebritis. At an advanced stages, a low density area with mass effect and peripheral edema may be seen in late cerebritis, or a hypodense masse with vasogenic edema and increasing mass effect in early capsule. Late capsule is characterized by an edema and a diminishing mass effect.

- *CECT* may show a mild patchy enhancement (= early cerebretis), or irregular peripheral rim enhancement (= late cerebretis), or a low density center lesion with a thin, well defined, enhancing capsule (= early capsule), or a shrinking cavity (may be multiloculated) with a capsule thickening (late capsule). Daughter abscesses may be seen adjacent to the main lesion.

**MR findings**

- *T1WI* and *T2WI/FLAIR*: Early cerebritis appears as poorly marginated mass, mixed hypo-/isointense on *T1WI* and ill-defined and hyperintense on *T2WI/FLAIR*. In late cerebritis, the lesion presents a center hypointense on *T1WI* and hyperintense on *T2WI/FLAIR*, and a rim iso-/hyperintense (mildly) on *T1WI* and hypointense on *T2WI/FLAIR*, associated with a hyperintense peripheral edema. In early capsule, the lesion center is hyperintense to CSF and the rim isointense (to hyperintense) to white matter (WM) on *T1WI*. On *T2WI/FLAIR*, the rim appears hypointense because of collagen, hemorrhage or paramagnetic free radicas. Late capsule appears as a shrinking cavity and a hypointense thickening capsule on *T1WI*, and hyperintense edema on *T2WI/FLAIR* (see Fig 25-26).

- *DWI* demonstrates an increased signal intensity both in cerebretis and abscess, but corresponding to a decreased ADC (restricted diffusion) only in latter lesion (see Fig 27).

- *Gadolinium enhanced T1WI* may show a patchy enhancement in early cerebretis, or an intense but regular rim enhancement in later cerebritis, or a well-defined, thin wall enhancing rim in early capsule, or a collapsed cavity associated to a thickened enhanced capsule, in later capsule (see Fig 29).

- MR Spectroscopy may demonstrate the presence of acetate, lactate, alanine, succinate, pyruvate and amino-acids in the central necrotic area, without abnormalities of N-acetyl-aspartate or choline.
Resolving abscess appears hyperintense on T2WI and FLAIR and hypointense rim resolves. Small ring or punctate enhancing focus may persist for months.

MENINGITIS

The report frequency of all meningitis in IE varie from 0% to some 20%, considering as well aseptic as septic meningitis. However, bacterial (suppurative) meningitis, with positive CSF cultures, is reported in only 7% of all patients with IE. It contributes to the poor prognosis of IE. Over one third of meningitis patients with IE develop additional neurologic complications like ischemic stroke, ICH or brain abscesses.

As for brain abscess, Staphylococcus aureus (+++) and Streptococcus pneumonia are most frequent isolated micro-oganisms in IE patients with pyogenic meningitis. Meningitis complicates 59% of IE due to Streptococcus pneumonia.

It is characterized by an inflammatory and/or infectious infiltration of the pia mater, arachnoid and CSF, related to hematogenous dissemination from the cardiac infection. EVOH often occurs as early complication of meningitis.

Imaging findings of meningitis complicating IE do not differ from other causes of meningitis (viral, chemical, or chronic granulomatous), apart from the more frequent association with other cerebrovascular complications of IE.

CT findings

- Normal study is most common with NECT. However, it may show mild subarachnoid space enlargement filled with a mildly hyperdense exudate (see Fig 30). Low density of adjacent cortical and su cortical areas may be seen, related to perfusion alterations and vasogenic edema. Enlarged Ventricules enlargement and effaced basal cisterns are more rare.

- CECT may demonstrate an enhancing exudate in sulci or in cisterns (see Fig 31).

- Arterial narrowing or occlusion may be associated and visible on CTA.

MR findings

- Exudate filling the subarachnoid space, within sulci and cisterns appears isointense on T1WI and hyperintense in T2WI and FLAIR. Abnormalities of adjacent parenchyma usually appears hypointense on T1WI and hyperintense on T2WI.

- DWI may shows restriction and associated infarction.
• 
  *Gadolinium enhanced T1WI* shows a typical smooth, intense enhancement of exudate and pia. Enhancement may be (rarely) nodular.

• Arterial narrowing or occlusion may be associated and visible on MRA.

• EVOH, ventriculitis, choroid plexitis, abscess, empyema may be associated.

**ENCEPHALOPATHY**

Encephalopathy in IE occurs in 5-24% of patients. It occurs usually during uncontrolled infection and is associated with an increase of mortality (60% in some series). Its pathogenesis, probably multifactorial, remains still unclear In two third of IE patients with encephalopathy, multifocal brain ischemia lesions are present, suggesting the potential role of microscopic septic emboli (focal encephalopathy). About one third of IE patients with encephalopathy do not present any neurological deficit (nonfocal encephalopathy)\(^2,4\).

In those cases, MRI may shows non specific areas of abnormalities hyperintense on *T2WI* and *FLAIR*.
Fig. 0: NECT of brain showing the parenchymal hypodensity of left middle cerebral artery territory and focal hyperdense vessel in left sylvian fissure ("Dot sign") caused by acute thrombus.

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Fig. 0: CT perfusion (axial analysis) in a IE patient: the Mean Transit Time (MTT) map shows an allongement of MTT in the left middle cerebral artery related to an ischemic stroke.

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**Fig. 0:** CT perfusion (axial analysis) in the previous patient: the Cerebral Blood Flow (in ml/100g/min; CBF map shows a decrease of CBF in the left middle cerebral artery related to the ischemic stroke.

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**Fig. 0:** CT perfusion (axial analysis) in the previous patient allows to assess ischemic core (red area) versus penumbra (green area) in the left middle cerebral artery.

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**Fig. 0:** CTA (coronal view) in the same patient showed an occlusion of the M1 segment of the middle cerebral artery.

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**Fig. 0:** Brain T1WI (sagittal view) shows an early cortical swelling as a hypointense fronto-insular area with loss of gray-white borders.

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Fig. 0: T2WI (coronal view) shows an early fronto-insular cortical swelling as a hyperintense area, in the right superficial middle cerebral artery territory.

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**Fig. 0:** FLAIR (axial view) is positive, showing an early cortical swelling as a hyperintense area in right fronto-insular lobes.

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**Fig. 0:** DWI (axial view) shows in this case a hyperintense area of the left superficial middle cerebral artery territory corresponding to a restricted diffusion in cytotoxic edema (low apparent diffusion coefficient on ADC-map: not showed).

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Fig. 0: T2* GRE shows hypointense acute blood products in left cortico-insular ischemic area highly suggestive of a septic embolic origin for the brain infarction.

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**Fig. 0:** TOF (Time of Flight) MRA in this patient demonstrates the left middle cerebral artery occlusion (M1 segment). Anterior communicant artery and both posterior communicant arteries are permeable.

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**Fig. 0:** T2WI shows two microbleeds appearing hypotense and located in the cortical parenchyma and in a sulci of the left frontal lobe.

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**Fig. 0:** T2WI shows an hypotense and focal lesion located in a sulci of the left occipital lobe corresponding to cerebral microbleeds.

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Fig. 0: T2WI shows microbleed as an hypointense cortical "dot" in the left frontal lobe.

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**Fig. 0:** NECT of posterior fossa (axial view) shows a voluminous hematoma of right cerebellar hemisphere, resulting in moderate mass effect on fourth ventricle and basal cisterns, with a subarachnoidal hemorrhage in adjacent cistern.

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**Fig. 0:** NECT of supra tentorial brain (axial view) shows a focal cortical hematoma of left occipital lobe with a subarachnoidal hemorrhage of left hemisphere.

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Fig. 0: T2WI of posterior fossa (coronal view) in same patient as in Fig 12 demonstrates a voluminous hematoma of right cerebellar hemisphere at subacute late stage, appearing hyperintense at center, with a peripheral hypointense rim (hemosiderin deposits).

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**Fig. 0:** T1WI of posterior fossa (axial view) in same patient as in Fig 12 shows a voluminous hematoma of right cerebellar hemisphere at subacute late stage, appearing hyperintense at center (extra cellular Methemoglobin phase) with a peripheral hypointense rim (hemosiderin deposits).

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**Fig. 0:** NECT of supra tentorial brain (axial view) shows a massive subarachnoidal hemorrhage, as well in all basal cisterns as in both sylvian fissures, associated with an extraventricular obstructive hydrocephalus. A hypodense area in left temporal lobe related to an ischemic stroke lesion is also visible.

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**Fig. 0:** T2*GRE image of supra tentorial brain (axial view) shows multiples foci of blood within sulci, predominantly in left hemisphere, related to a subarachnoidal hemorrhage.

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**Fig. 0:** T2*GRE image of supra tentorial brain (axial view) shows a focal blood within few sulci of left frontal lobe, related to a subarachnoidal hemorrhage.

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Fig. 0: CTA MIP reconstruction (coronal oblique reconstruction ; 7mm thickness) shows an anterior interhemispheric massive subarachnoidal hemorrhage (« hematoma-like »), left paramedian. An associated focal arterial dilatation on a distal branche of left anterior cerebellar artery is visible and related to an infectious aneurysm that was probably the cause of the bleeding. An important mass effect on left frontal lobe is also visible.

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Fig. 0: CTA MIP reconstruction (coronal reconstruction; 7mm thickness) shows a focal arterial dilatation on an insular branche of middle cerebral artery (M3 segment) related to an infectious aneurysm without visible intracranial bleeding.

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Fig. 0: CTA Volume Rendering reconstruction of the previous patient (Fig 20) shows the left middle cerebral artery infectious aneurysm previously described.

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**Fig. 0:** T1WI of brain (coronale view) shows a well defined necrotic mass of left frontal lobe, with a hyperintense signal at the lesion center (hyperintense to CSF) and rim appearing isointense to white matter (WM). There is an associated moderate mass effect. It corresponds to an early capsule stage abscess.

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**Fig. 0:** FLAIR of brain (axial view) of the previous patient (Fig 22) shows a well defined necrotic mass of left frontal lobe, with a heterogeneous signal at the lesion center, hyperintense and isointense to WM, and a rim appearing isointense to WM. There is an associated moderate mass effect. It corresponds to an early capsule stage abscess.

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Fig. 0: DWI of brain (axial view) of the previous patient (Fig 22) demonstrates a hyperintense signal at center of the lesion associated with a low ADC (ADC-map not showed) related to a diffusion restriction. It corresponds to an early capsule stage abscess.

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**Fig. 0:** T2*GRE of brain (axial view) of the previous patient (Fig 22) also demonstrates a microbleed, as hypointense signal and punctiform lesion, in the cortex of anterior right frontal lobe. The abscess appears as hyperintense at center, with linear hyposignal (hemorrhage or paramagnetic free radicals) with an isointense rim.

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**Fig. 0:** Gadolinium enhanced T1WI of brain (sagittal view through the right cerebral hemisphere) of the previous patient (Fig 22) shows a regular and well-defined, thin wall, and enhancing rim. It corresponds to an early capsule stage abscess.

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**Fig. 0:** NECT of supra tentorial brain (axial view) in a IE patients shows a mild enlargement of left central sulcus that appears mildly hyperdense without any significative abnormalities of adjacent parenchyma, related to a focal meningitis.

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**Fig. 0:** CECT of supra tentorial brain (axial view) of the previous patient shows a markedly enhancement of the previously described enlarged spontaneously hyperdense central sulcus and of the adjacent cortex, related to a focal meningitis complicating an IE.

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Conclusion

Infective Endocarditis is a rare disease, but it remains a severe affection still associated with high rates of morbidity and mortality. Cerebrovascular complications of infective endocarditis, symptomatic or asymptomatic, are highly frequent and contribute to its poor prognosis.

Recent studies have suggested the importance and the benefits of performing routinely CT and/or MRI (highly sensitive) examinations of brain, leading to substantial changes in diagnostic classification or clinical decisions for patients with IE.
References


