Special Article - Cerebral Infarction

Confounding Imaging Findings in Subacute-Chronic Cerebral Infarction

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Abstract

Imaging findings which appear during the course of subacute and chronic cerebral infarct and which can lead to confusion for diagnosis is presented in a series of six patients. These include: luxury perfusion, fogging effect, hemorrhagic transformation, Wallerian degeneration, diaschisis, laminar cortical necrosis and encephalomalacia with exvacuum dilation.

Keywords: Subacute infarct; Chronic infarct; Luxury perfusion; Fogging effect; Hemorrhagic transformation; Wallerian degeneration; Diaschisis; Laminar cortical necrosis; Laminar cortical necrosis encephalomalacia; Exvacuum dilation

Abbreviations

MCA: Medial Cerebral Artery; ACA: Anterior Cerebral Artery; WD: Wallerian Degeneration; CT: Computerized Tomography; MRI: Magnetic Resonance Imaging; FLAIR: Fluid Attenuated Inversion Recovery; CBF: Cerebral Blood Flow; SPECT: Single-Photon Emission Computed Tomography

Introduction

After occlusion of a cerebral artery, imaging studies confirm cerebral infarction during hyper acute and acute phases [1-3]. This allows engaging in therapeutic decisions with the aim of reducing damaged tissue and thus the subsequent sequels.

Nevertheless, imaging findings can occur during subacute and chronic phases of cerebral infarction that could disguise or mimic other neurological processes. Although these findings have been described in the literature, in many occasions they are overlooked and can originate confusion when conducting imaging diagnosis. Among these are: luxury perfusion, fogging effect, hemorrhagic transformation, Wallerian degeneration, diaschisis, laminar cortical necrosis and encefalomalacia with exvacuum dilation [4-9].

During the subacute-chronic phase of cerebral infarction, many physicians can inform the images as negative and may even not associate these imaging findings with primary ischemic damage, interpreting them as independent events, thus modifying the diagnosis

The object of this work was to describe the occurrence of these imaging findings in a series of six patients with cerebral infarction during the subacute-chronic phase. The description of the subjects is represented in Table 1.

Case Presentation

Case 1

Three-year-old female patient with left hemiparesis and previous diagnosis of brain tumor. After a simple (Figure 1A) and contrasted (Figure 1B) CT scan performed between day 3-7 an increased density in the territory irrigated by the MCA and gyriform enhancement
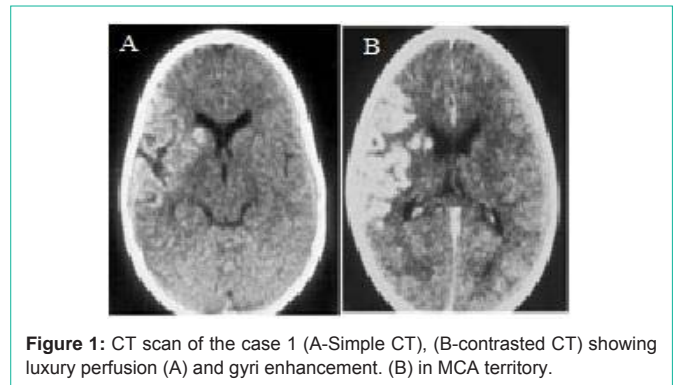

Figure 1: CT scan of the case 1 (A-Simple CT), (B-contrasted CT) showing luxury perfusion (A) and gyri enhancement. (B) in MCA territory.
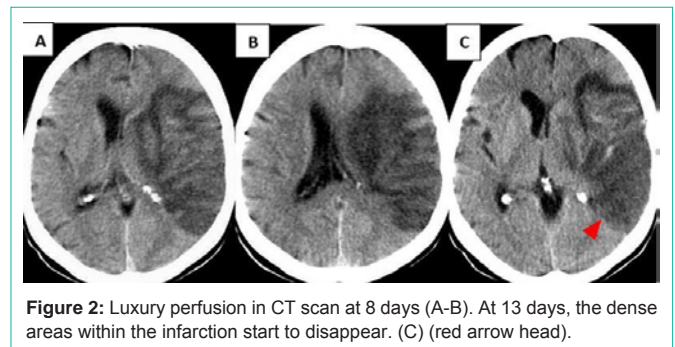

Figure 2: Luxury perfusion in CT scan at 8 days (A-B). At 13 days, the dense areas within the infarction start to disappear. (C) (red arrow head).

are found, due to luxury perfusion effect (Figure 1A,B). This effect confounds the real diagnostic of ischemic stroke and the images were interpreted as a brain tumor.

Case 2

Seventy-one-year-old female patient who presented sudden right hemiplejia and loss of consciousness. CT was performed 4 hours after the event and was considered normal. Due to unfavorable evolution, the study was repeated after 8 days (Figure 2A,B) and after 13 days (Figure 2C). In A and B an infarct of the right MCA can be seen, with mass effect on the neighboring ventricle (which is collapsed) and slight displacement of the midline. Hyperdensity within the affected vascular territory follows the organization of gyri, contrasting with

Table 1: Description of the subjects included in the review.

Case	Age/Sex	Type of study	Effecto observed/time from infarct onset	Simulation
1	3 F	Simple and contrasted CT	Luxury Perfusion/ 3 days	Tumor
2	71 F	Simple CT	Luxury Perfusion/ 8 and 13 days	Hemorrhagic transformation of the infarct
3	56 M	Simple CT / 2nd simple CT	Fogging effect/17 days	Interpreted as a normal study, no previous evaluation
4	60 M	Simple CT	Hemorrhagic transformation /3, 10, 20, 25 days	Intraparenchymal hematoma at day 10
5	35 F	Simple CT	Fogging effect / 21 days	Informed as normal
		1sr MRI T2, FLAIR	Fogging effect /21 days	Possible Multiple Sclerosis
		Contrasted MRI	Luxury Perfusion, gyriform enhancement/ 28 days	The study was described without offering diagnosis
		Followup Multislice CT	Hypodensity with slight mass effect on the ipsilateral ventricle and laminar cortical necrosis / 35 days	Possible low grade glioma
		MRI T1, FLAIR	Laminar cortical necrosis /40 days	Hemorrhagic transformation of the infarct
			Encephalomalacia with ventricular dilation exvacuum and gliosis/ 3 months	Chronic infarct was informed with ventricular asymmetry andgliosis was interpreted as edema. Edema
		Last MRI T2 y FLAIR	Wallerian Degeneration / 1 year and 9 months	Chronic Infarct in the brain stem independent from the supratentorial infarct
6	65 F	MRI T2, FLAIR y T1	Chronic Infarct of the right MCA Encephalomalacia with exvacuum ventricular dilation/ 2 years and 4 months	The three findings were interpreted as independent conditions (chronic infarct, ventricular asymmetry, atrophia of the right cerebral peduncle and of the left hemicerebellum)
			Wallerian Degeneration 2 years and 4 months	
			Diaschisis/ 2 years and 4 months	

the hypodensity resulting from necrosis and inflammation of the subacute infarct (luxury perfusion). In Figure 2C, it can be observed those 5 days later, the mass effect persists and the dense areas within the infarction start to disappear (red arrow head).

Luxury perfusion is defined as the cerebral blood flow (CBF) reaching the brain post stroke, which is above those levels necessary to fulfill the metabolic needs of the tissue [4]. When an artery is occluded and recanalization does not occur spontaneously or through a therapeutic procedure in a time period during which brain tissue is still viable, permanent neuronal and glial damage occurs, irremediably leading to parenchymal necrosis (cerebral infarction) [10].

"Luxury perfusion" starts about the 5th day post stroke and lasts for several weeks. During this phase CBF increases progressively and frequently reaches values above normal levels at approximately 8 to 12 days, indicating that reperfusion is no longer useful in a tissue irreversibly damaged and probably reflecting futile recanalization [11] or neovascularization with abnormal autoregulation [10]. As demonstrated by SPECT [4,12], oxygen and glucose consumption in this zone is highly reduced or even null. Other mechanisms involved in this phenomenon are ruptured blood brain barrier [4,7], which together with dilated capillary and venous proliferation, lead to increased density of the infarcted area. Luxury perfusion is revealed more clearly with the administration of contrast due to giriform enhancement [13,14].

It can be mistaken with other conditions, mainly with hemorrhagic transformation of the infarct. It can also be mistaken with CNS primary tumors due to the mass effect it can produce on the ventricular system, as during this period vasogenic edema is evident. Diagnostic doubts can be discarded evaluating the initial images, where indirect and early signs of infarction should be looked for. It
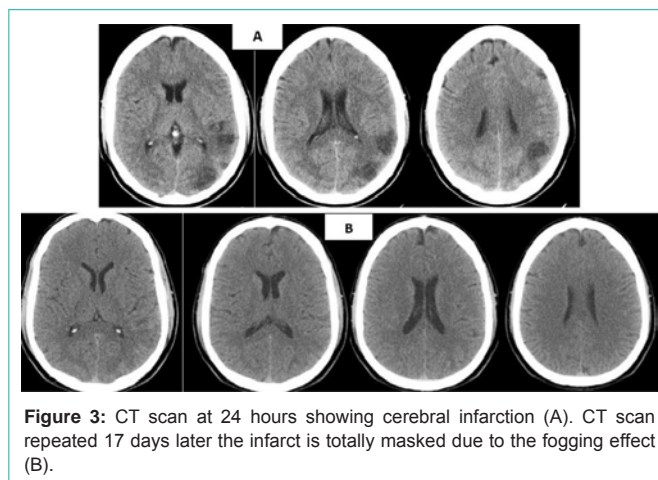

Figure 3: CT scan at 24 hours showing cerebral infarction (A). CT scan repeated 17 days later the infarct is totally masked due to the fogging effect (B).

should be taken into account that CT scanning can be normal when performed during the first hours after symptom onset. Nevertheless, combining neuroimaging studies is always a good option in order to establish differential diagnosis. Luxury perfusion in subacute infarction exhibits giriform enhancement, which most tumors do not show.

Case 3

A CT scan was performed 24 hours after ischemic stroke in a 56-year-old male patient with hypertension and hypercholesterolemia showing a hipodensity in the superficial territory of the left MCA (Figure 3A). CT scan repeated 17 days later and the infarct is totally masked due to the fogging effect, and interpreted as a normal study (Figure 3B), because no previous evaluation was considered.

Fogging effect was described for the first time in 1979 by Becker, et al. [15], involving in brain infarct the transition from hypodensity
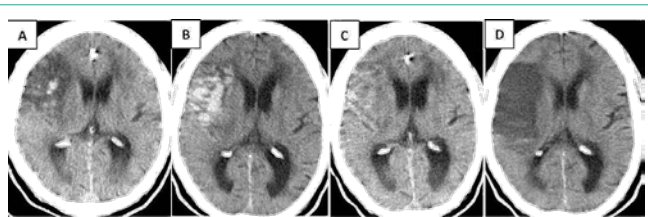
Figure 4: Serial CT scan at 3 (A), 10 (B), 20 (C), 25 (D) days showing hemorrhagic transformation of the infarction until the returning of the hypodensity (D). In C a fogging effect is observed.
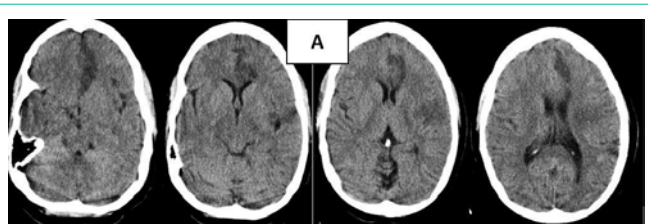

Figure 5A: CT scan demonstrated an acute infarct of the left MCA and ACA at 36 hours.
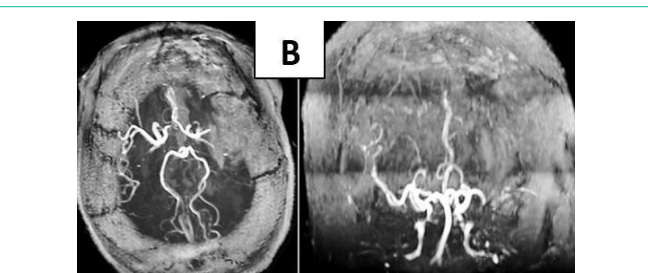

Figure 5B: Occlusion of the MCA and late recanalization of the left ACA is visualized in the angioresonance.

to a zone with normal attenuation. It occurs through the combination of several processes including the invasion by macrophages loaded with lipids [16], capillary proliferation, leukocyte infiltration and extravasation of blood cells through the walls of the lesioned blood vessels [5].

This effect is related with luxury perfusion, with increased blood flow concealing the radiological hypodensity in CT scans [3] associated with decreased brain edema [14,15]. Transient changes in the affected zones have also been described in MRI, where it can appear even from the sixth day after acute stroke [11].

It appears in up to 50% of subacute infarcts, more frequently between the 2nd and 3rd weeks after the event, and it does not last more than 6 months [16-17]. This phenomenon could be mistaken with a normal study, because when fogging effect appears false negatives increase during follow up and the size of infarcts can be underestimated.

Case 4

A male of 60 years-old, which CT scan, shows an infarct in the territory of the right MCA, which evolution demonstrate a hemorrhagic transformation, but at day 10 it was confused with and intraparenchymal hematoma. The first scan (Figure 4A) shows an infarct with petechial hemorrhage, while in Figure 4B there is

increased bleeding with petechial confluence producing a slight mass effect on the adjoining ventricle. After 17 days (Figure 4C) the blood is almost isodense with respect to the neighboring brain parenchyma, demonstrating that fogging effect in the course of subacute hemorrhagic transformation of infarcts, due to the degradation of hemoglobin, causes a transition of the infarct from hypodensity to isodensity. In Figure 4D the hypodensity of the infarct returns.

The hemorrhagic transformation is a complication, which appears in 90% of cardioembolic infarcts and in 65% of arterial infarcts spontaneously, or post fibrinolysis [18]. It occurs as a consequence of blood extravasation to the infarcted tissue due to early damage of the blood brain barrier and the secondary inflammatory effect in response to the arrival of plasmatic components to the brain tissue. This blood reaches the damaged vessels through recanalization of the occluded artery or through collaterals [19,20]. Diagnostic errors could be with hypertensive intraparenchymal hematoma, amyloid angiopathy, bleeding tumor, luxury perfusion and cortical laminar necrosis.

Case 5

A CT scan in a 35-year-old female patient, receiving oral anticonception drugs, demonstrated an acute infarct of the left MCA and anterior cerebral artery (ACA) (Figure 5A). Occlusion of the MCA with scarce collateral circulation and late recanalization of the left ACA is visualized in the angioresonance (Figure 5B).

21 days later, due to the fogging effect the image was interpreted as normal (Figure 5C), where CT scan only shows partial blurring of the left Sylvian valley. In MRI study, the sequences FLAIR (Figure 5D) and T2 (Figure 5E) shown crown radiated hyperintensities near the left lateral ventricle (red arrow head), these images were evaluated as possible multiple sclerosis.

At 28 days, a contrast T1-MRI was performed showing a gyriform enhancement due to a damaged blood brain barrier and luxury
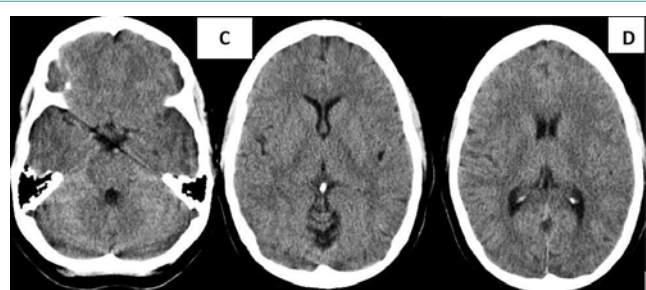

Figure 5C-D: The fogging effect in CT scan at 21 days. The image was interpreted as normal, and in D CT scan only shows partial blurring of the left Sylvian valley.
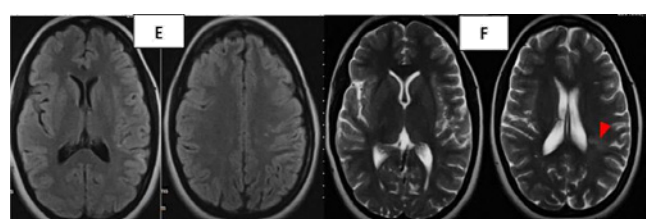

Figure 5E-F: MRI study visualizing crown radiated hyperintensities near the left lateral ventricle (red arrow head) due to the fogging effect.
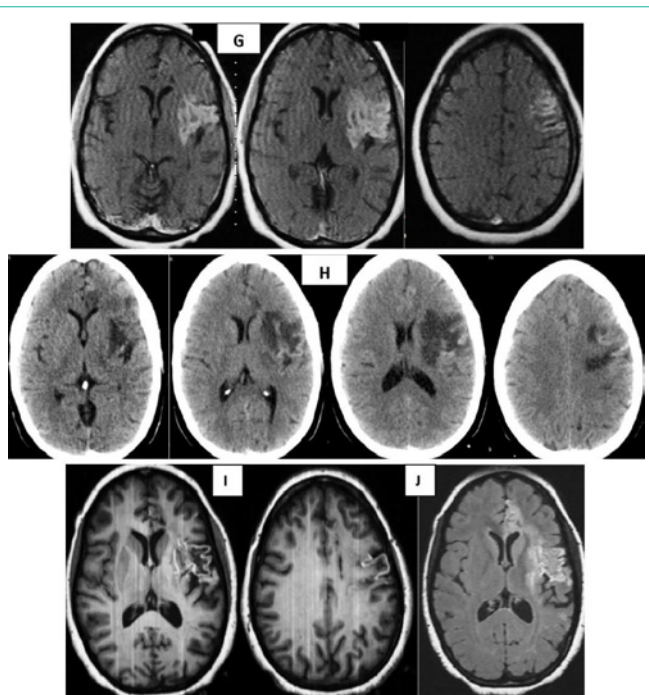
Figure 5G-J: MRI performed 40 days after the previous CT scan show laminar cortical necrosis in the superficial territory of left MCA and ACA with associated gliosis. Area of encephalomalacia in axial T2 sequence is shown (J).

Figure 5K-L Areas of encephalomalacia in axial (K) and sagital T2 (L) MRI sequences 3 months after the ischemic event.

Figure 5M-N: In T2 axial a small hyperintensity in the left border of the pons and cerebral peduncle ipsilateral to ischemic damage due to Wallerian degeneration (red arrows).

Figure 5O-P: Two years later the sequences axial T2 and FLAIR show hyperintensity due to gliosis and atrophy in the left cerebral peduncle (red arrows), greater dilation of the temporal horn as well as hippocampal atrophy on that side.

Figure 6 A-G: Cerebral infarction, hypointense in FLAIR (A) and hyperintense in T2 (B-C). Wallerian degeneration causes assymetry of the right cerebral peduncle (red arrow in D). Atrophy of the contralateral cerebellar hemisphere (left) secondary to diaschisis is shown with black arrows (D-G).

perfusion (Figure 5F).

In an evolutive evaluation, a simple CT scan performed 35 days after symptom onset shows an hypodense lesion with a slight mass effect on the ipsilateral ventricle and hyperdense curved lines following some gyri (laminar cortical necrosis); which was mistakenly interpreted as a possible tumor (Figure 5G). Sequences T1 and FLAIR in an MRI performed 40 days after the previous CT scan show laminar cortical necrosis in the superficial territory of left MCA and ACA with associated gliosis (Figure 5H,I).

Laminar cortical necrosis is due to unequal destruction of the neocortex with preservation of some layers and impairment of others (layers 3-5), accompanied by gliosis [21], and appearing during the late subacute and chronic stages of cerebral infarct. It occurs when the influx of oxygen and glucose are inadequate for maintaining regional cerebral metabolism as in cerebral infarct, hypoxia, cerebral hipoperfusion and hipoglicemia or due to an increased demand (status epilepticus) [22]. They are visualized in MRI as cortical curve-lined hyperintensities that follow the gyri pattern in T1 sequences without contrast and FLAIR. This hypersignal in T1 is caused by the accumulation of macromolecules, lipid-laden macrophages and no for the presence of blood or calcium [21]. It can be mistaken with the presence of blood in the infarct. Cortical necrosis is evident two weeks after infarction, with a peak of highest intensity at about one month, slowly disappearing after 3 or more months [23]. Occasionally it can be seen a year after infarction.

Evolution of the infarct to chronicity 3 months after the ischemic event. Area of encephalomalacia in axial (Figure 5J) and sagital T2 (Figure 5K) sequences. Impairment of the left half of the genu and
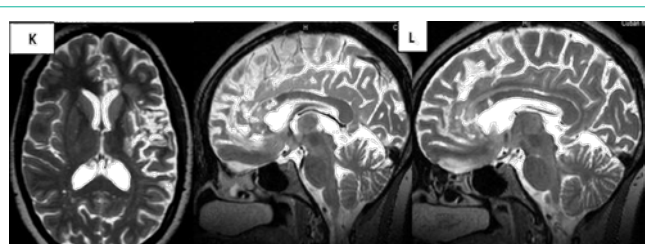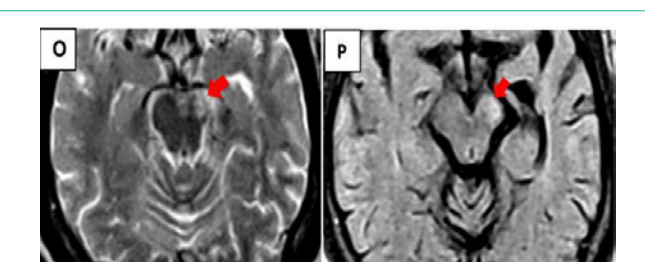
anterior part of the corpus callosum´s truncus, typical of cerebral infarcts is evident, because tumors cross the midline. The anterior

cingulum is also affected, concurring with cognitive decline in this patient.

Figure 5L shows fronto temporo insular atrophy with increased frontal subarachnoid space and of the left Sylvian valley in a coronal T2 slice; hyperintensity of the left cortico-spinal tract starting in the area of residual encephalomalacia from chronic infarction (red arrow). In T2 axial (Figure 5M) a small hyperintensity in the left border of the pons and cerebral peduncle ipsilateral to ischemic damage due to Wallerian degeneration (red arrows).

Encephalomalacia is the residual cavity remaining as a sequel after brain damage, with neuronal and glial death that interrupts the architecture of normal brain parenchyma, secondary to any acute event: ischemic (chronic infarct), hemorrhagic, traumatic, surgical, etc [24,25]. This leads to loss of local volume, gliosis and dilation of the ventricle ipsilateral to the lesion, thus being easily identified in neuroimaging studies [24,25].

Figures 5N,O,P two years later (axial T2 and FLAIR sequences respectively) show hyperintensity due to gliosis and atrophy in the left cerebral peduncle (red arrows), greater dilation of the temporal horn as well as 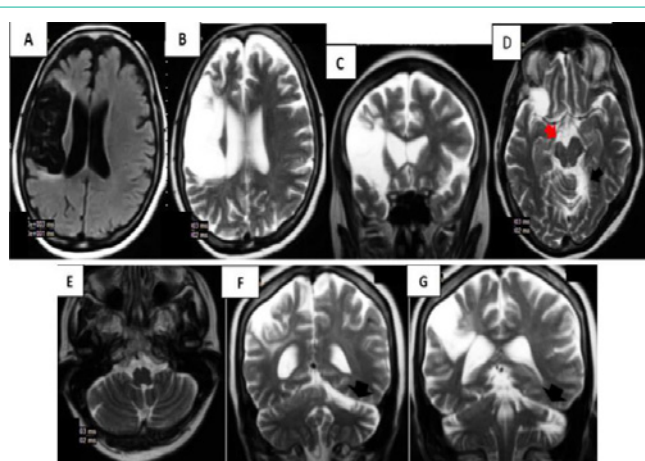hippocampal atrophy on that side. The later images were interpreted as another brain stem infarct and not as Wallerian degeneration secondary to the chronic infarct in the territory of the left MCA two years ago, the same as hippocampal atrophy and dilation of the temporal horn.

Wallerian degeneration (WD) is a progressive demyelinating process of the distal ending of the axon after proximal damage that leads to axonal atrophy, gliosis and secondary loss of volumen. It is a distant sequel of the primary event [4]. The most affected pathway is the pyramidal tract, but damage in the optic pathway, corpus callosum and pontocerebellar fibers has also been described [26-28]. Although the most frequent cause is cerebrovascular disease, it can appear after any other event damaging the origin of the pathways as in infarcts, hemorrhages, encephalic trauma, surgery, frontotemporal dementias, multiple sclerosis and others [29].

Degeneration of the corticospinal tract is easily visualized in coronal T2 sequences, displaying hyperintensity throughout the length of the damaged tract from the chronic infarct in the territory of the medial cerebral artery to the ipsilateral cerebral peduncle. During the following years the cerebral peduncle and pons on the same side of the infarcted territory can appear hyperintense in T2 and FLAIR and atrophied; both could appear in CT scans and MRI [30]. In this case, atrophy and hyperintensity of the cerebral peduncle could be mistakenly reported as isolated events and not as a consequence of the primary event, in this case, chronic infarct.

Case 6

Is a sixty eight year old female patient with a story of arterial hypertension and a cerebral infarct two years and 4 months ago? In Figure 6 is observed encephalomalacia with exvacuum dilation of the ipsilateral ventricle secondary to a chronic infarct in the territory of the right MCA. The infarct appears hypointense with surrounding hyperintense gliosis in FLAIR (Figure 6A) and hyperintense in T2 (Figure 6B,C). Assymetry of the right cerebral peduncle is evident, which appears smaller (red arrow in Figure 6D) due to Wallerian degeneration. Black arrows denote atrophy of the contralateral

cerebellar hemisphere (left) secondary to diaschisis (Figure 6D-G).

Diaschisis is a functional inhibition leading to loss of excitability because of a unilateral lesion in a distant zone, not having been lesioned directly, but still interconnected [31]. Crossed cerebellar diaschisis refers to a unilateral lesion (in this case a cerebral infarct) that can result in decreased CBF in the contralateral cerebellar hemisphere [31,32] that can be reversible or not and can evolve in time towards atrophy due to deafferentation or selective neuronal death.

CT and MRI can show a chronic infarct of MCA territory and atrophy of the contralateral hemicerebellum [33]. Confounding diagnosis in this case is reporting cerebellar hemiatrophy as an event, which is independent from the primary ischemic damage.

Conclusion

It is very important to consider in the subacute phase of a cerebral infarct the imaging findings previously described, which appear frequently. Evaluating previous images, the use of contrast substances and combining images contributes to achieve more precise diagnoses. In the chronic phase of an infarct, local and distant sequels of the primary ischemic event should be sought.

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