Neurospect applications in Psychiatry.
AJ45-1

Abstract.
During the last years, functional imaging by means of Neuro SPECT has been progressively incorporated to the differential diagnosis of psychiatric disorders, particularly mood disorders, psychosis, schizophrenia, cognitive impairment, Alzheimer's Disease, obsessive-compulsive disorder, attention deficit disorder, and brain effects of neurotoxic exposure. This review will be initiated evaluating the findings in brain hyperacute infarction before and post arterial thrombolisis treatment in order to demonstrate the sensitivity and specificity of this imaging modality in clinical work. On methodology, we have to comment, furthermore, that we perform double image reconstruction, one without attenuation correction for the lateral images, posterior and anterior images and the superior image of the brain cortex. A second reconstruction is performed with attenuation correction by means of a Chang coefficient of 0.1. This is applied to the parasagittal images and to the inferior image of the brain. In this last image, we remove the cerebellum in order to facilitate inspection of the inferior aspects of occipital and temporal lobes. We apply this modality also for the study of basal ganglia. The software that we have used is the Oasis Software of the Segami corporation of USA and brain imaging is performed by means of the Ecam Dual Head Siemens camera.

We compare the clinical data with a normal, age matched database and display the results using a color scale defined by Standard Deviation below and above the normal mean for HMPAO Tc99m, while the normal range is depicted in color gray.

Clinical correlations

Hyper acute brain preinfarct.

Figure 1: We observe in the series of 8 images an area of deep hypo perfusion, deeper than 5 standard deviations below the normal mean. Depicted in color black and projecting on the left parietal lobe, and surrounded by an area of systemic penumbra in the colors green, light blue and dark blue, denoting 2, 3, and 4 standard deviations below the normal means (normal range is depicted in color gray). We observe also hyperperfusion in the lateral aspects of the left temporal lobe and at the level of the interhemispheric fissure we observe hyperperfusion of the parietal lobe, and particularly of the intermediate cingulate gyrus, (area 23 of Brodmann). In these images, we observe the extension of the preinfarct in the left hemisphere in the superior image, that demonstrates that practically the totality of the territory of the left medial cerebral artery is involved. It calls our attention also the increased perfusion in the right hemisphere at the level of the occipital lobe, at 2 and 3 standard deviations above the normal mean (colors red and pink). The irreversibility of areas of deep ischemia is demonstrated when the levels of hypo perfusion reaches below 10 standard deviations below the normal mean. This does not occur in this particular patient[1, 2]. Figure 2 demonstrates these findings at the left mesial image and also at the left lateral image.
In figure 3, it calls to our attention the fact that the basal ganglia show increased perfusion in the contralateral hemisphere; namely there is marked and extensive increased perfusion in the right thalamus and in the lateral aspects of the lentiform nucleus with a very slight increase in perfusion of the left thalamus. This observation has recently been reported and demonstrates the fact that the loss of function of one hemisphere due to acute ischemia, introduces a loss of regulation of cerebral blood flow in particularly the basal ganglia in the opposite hemisphere. And for this reason, there is increased perfusion very marked at the level of the right thalamus and right lentiform nucleus (normal range in color red, color white larger than 4 standard deviations above normal mean).

Post intra-arterial thrombolysis performed before six hours of evolution of the symptomatology demonstrates a significant reduction of hypoperfusion at the level of the left parietal lobe, with a small remanent observed in the intermediate aspects of the left parietal lobe, particularly at the level of the convexity. Fig. 4.
The abnormalities demonstrated at the inter-hemispheric fissure have been corrected and at the level of the basal ganglia hyperfusion, thalamus and right lentiform nucleus have been corrected in Figure 5. In this particular patient, there is persistence of hypoperfusion of the caudate nucleus that correlates with hypoperfusion of the posterior cingulate and mesial temporal lobe, that points to the possibility of cognitive impairment in this patient.

**Mood disorders:**

We will analyze in this review neurospect findings in depression and bipolar disorders.

**Depression**
Figure 6: In this figure there is a predominant hypo perfusion in the orbital frontal region, and also at the level of anterior cingulate gyrus and in the left subgenual region (area 25 of Brodmann). There is also frontal temporal bilateral hypo perfusion and marked diminution of function in both posterior cingulate, while there is increased perfusion in thalamus and marked hypo perfusion in the head of the caudate nucleus. Figure 7. This depicts secondary cognitive impairment induced by depression and expressed at the level of the posterior cingulate gyrus and hippocampus in the mesial temporal lobe. There are also the previously described abnormalities of the caudate nucleus [3, 4, 5].

Figure 7.

Bipolar disorder:
Figure 8a and 8b demonstrates marked increased perfusion in both frontal lobes predominantly at the level of the intermediate gyrus, with the extension to the orbital frontal region. There is also increased perfusion in both posterior parietal lobes at the level of the interhemispheric fissure. Finally there is increased perfusion (2, 3 standard deviations above the normal mean in both temporal lobes). At the subcortical structures, figure 9, there is markedly increased perfusion in both thalami predominantly in the ventral segment and in both lentiform nuclei.\(^\text{[6,7]}\)
In first episode of schizophrenia in drug naïve patients Fig 10 NeuroSPECT demonstrates markedly lateralized to the left hemisphere hypoperfusion in the lateral aspects of temporal lobes, there is also marked and extensive hypoperfusion of the limbic system with involvement of bilateral in this case anterior, intermediate and posterior cingulate gyr.
Fig. 11 demonstrates marked hypoperfusion of the head of caudate nuclei, of both thalami in the dorsal aspects while the lentiform nucleus appears within normal range.

**Obsessive Compulsive Disorder.**

Fig. 12 The functional imaging characteristics of this process are frontal hyperperfusion, predominantly in the inferior gyrus, and with extensive projection into the fronto-orbital area. There is also increased perfusion of the anterior poles of both temporal lobes, in this case predominantly in the left hemisphere and hyperperfusion in the occipital lobe at the level of the visual associative cortex, areas 17 and 18 of Brodmann, that are in contact with the posterior cingulate gyrus (area 30 of Brodmann). In this patient there is also increased perfusion adjacent to the anterior cingulate gyrus, probably due to a comorbidity of attention deficit disorder.
In Fig. 13 in the subcortical structures there is increased perfusion on both lentiform nuclei and focal increase of the right thalamus.\textsuperscript{1,8,9}

**Attention deficit disorder.**

Fig. 14. These patients present hyperfrontality at the level of the intermediate and inferior gyr and increased perfusion adjacent to the anterior cingulate gyrus in the subgenual area. In this case there is also increased perfusion in the poles of temporal lobes. At the level of subcortical structures there is focal hypoperfusion in the head of the right caudate nucleus with increased perfusion of the ventral thalamus. Fig. 15.
Neurotoxic Exposure.

Fig. 16. The characteristic of neurotoxic exposure due to cocaine, crack cocaine, marihuana, edasis, etc. is a multifocal, disorganized hypoperfusion involving in this case both temporal lobes, extensively the limbic system at the level of the anterior and posterior cingulate gyri, the later with bilateral involvement, there is also hypoperfusion presentation in the convexity of the cortex in the projection of parietal lobes. This is highlighted in the superior images and in Fig. 17 in the subcortical structures there is increased perfusion in the ventral aspects of both thalami. [10, 11, 12]
Cognitive Impairment: Alzheimer’s Disease.

In advanced Alzheimer’s Disease, Fig. 18 the characteristic findings are symmetrical or a symmetrical bilateral hypoperfusion, which is always bilateral. In this case there is extensive hypoperfusion at 5 Standard Deviations below the normal mean (for this age group), depicted in color black, there is extensive and deep hypoperfusion in the posterior cingulate gyrus bilaterally. Finally there is also bilateral temporal hypoperfusion at the level of the mesial aspects in the projection of the hippocampus, while in the subcortical structures, Fig 19 there is deep hypoperfusion of both caudate nuclei, with marked hypoperfusion of both thalami and the right lentiform nucleus. [13, 14, 15 ]
In conclusion

Fig 20 demonstrates that the clinical entities that evolve with hyperfrontality (color red), are represented by Bipolar Disorder (B.P), Obsessive Compulsive Disorder (OCD) and Attention Deficit Disorder (ADD).

Figure 20.

B.P. is characterized by increased frontal perfusion in areas 9 and 10 in the Executive Cortex and also in Area 7 in the posterior parietal lobes, there is also increased perfusion in the thalami.

Furthermore, in OCD and ADD increased frontal perfusion is present in the inferior aspects of the Executive Cortex, namely area 10 of Brodmann, in ADD there is also characteristically increased perfusion adjacent to area 24, the anterior cingulate gyrus.

The clinical entities characterized by constant hypoperfusion are Depression (color blue) with hypoperfusion located in the orbito-
frontal area (area 11 of Brodmann) and the anterior cingulate and subgenual areas (areas 24 and 25 of Brodmann respectively). There is also increased thalamic perfusion.

In Schizophrenia the classical findings are hypoperfusion of temporal lobes lateralized more frequently to the left hemisphere in area 21 and 22 of Brodmann, anterior cingulate (area 24 and 25), intermediate cingulate (area 23) and area 30 in the posterior cingulate gyrus. There is also hypoperfusion in the hippocampus and in the caudate nucleus.

In drug exposure the presentation is of disorganized, multifocal hypoperfusion, while in Alzheimer’s Disease there is bilateral temporal-parietal hypoperfusion in advanced cases, and in mild cases there is predominantly hypoperfusion of the posterior cingulate and hippocampus projection, caudate nucleus and thalamus.

The functional imaging NeuroSPECT characteristics described are an effective addition the differential diagnosis workup of these psychiatric patients, thus becoming effective information for the clinical management of complicated cases, where the presence of comorbidities have to be defined for proper care of their illness.

Bibliography


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