Hypomanic Episode Following the Excision of a Large Meningioma

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Introduction

Although there remains much to uncover, it is clear that the cingulate cortex plays an important role in the regulation of emotions and cognition which implies an association between the activation of the cingulate cortex and different psychological disorders.

This area was originally divided by Brodmann into the anterior cingulate cortex (area 24, 25, 32, 33) and the posterior (area 23, 29, 30, 31).

The segmentation of the cortex into these sub-areas, based on studies of the structural connections using diffusion tensor imaging, supports the presence of many functional regions which share numerous circuits with the prefrontal cortex, motor cortex, amygdala, spinal cord, hippocampus, as well as areas of the limbic system (Figure 1).

Case History

We present the case of a 65-year-old patient consulted after three days of difficulty speaking and change of behavior.

After scans with Computerized Tomography (CT) and Magnetic Resonance Imaging, (MRI) a voluminous left frontal meningioma was diagnosed measuring about 7 cm of major axis, dependent on the falx cerebri, which compressed the cingulum, corpus callosum, and lateral ventricle (Figure 2).

The patient underwent a surgery one week after and vocal fluency was recovered. Without further incident she was discharged 6 days later. At 20 days after the surgery, the patient comes in the emergency room after progressively change in behavior consisting of impulsive conduct without inhibition, euphoria, increase in activity, and a lack of forethought. The patient presented no prior psychiatric record, nor took corticosteroids.

After excluding complications with the use of an emergency CT scan and a blood test, she was evaluated by the psychiatric service confirming the diagnosis of a hypomanic episode with the presence of rapid thoughts, flight of ideas, tangential thinking, and pressured speech. The patient was not aware of the illness. They had a diminished necessity for sleep, and had even recovered the ability to fluently speak a foreign language they had not used long time ago. Treatment was established with olanzapine and lorazepam, and continued with follow-up mental health service in the proper center (Figure 3).

A few days after treatment began, a stable mood was achieved. After two months, the medication was suspended and the patient appeared to be in their previous base state.

A sample analysis of the meningioma revealed it is of grade III and she received complementary treatment with radiotherapy. Five years later the patient is asymptomatic without psychotropic treatment and with no recurrence.

Discussion

Reviewing the functions that are attributed to the cingulum and its circuital connections, we believe our patient’s disorder is explicable through changes in the structure of the cingulum after being abruptly decompressed with the removal of the tumor. (Probably a cease of the inhibition of the connections between the anterior cingulum and the frontal orbital cortex).

The Anterior Cingulate Cortex (ACC) presents extensive connections with areas knowingly affecting the emotions (amygdala), memory (hippocampus), autonomic nervous system, (hypothalamus and brainstem) and reward mechanism (frontal orbital cortex, ventral striatum).
Specifically, Brodmann’s area 24 has classically been related with behavior, aspects of cognition and emotion, as well as the inhibitory control of these.

The Medial Cingulate Cortex (MCC) or dorsal Anterior Cingulate Cortex (dACC) have extensive cognitive connections (prefrontal cortex) and are related to motor skills (premotor areas and primary motor functions) [1].

The most posterior portion of the medial cingulate cortex is implicated in the visuospatial orientation and the execution of movement.

Both the anterior and medial cortex receive and elaborate perception and the affective dimension of pain [2]. This perception is incrementally related with increased activity in the anterior cingulate.

The components of this pain network include the anterior cingulate cortex, the amygdala, the somatosensory cortex and the periaqueductal grey matter.

Certain evidence suggests that area 25, within the anterior cingulate, in the region below the corpus callosum, is, in fact, a distinct zone of its own. The high concentration of serotonin receptors in this area makes it crucial for serotonergic regulation and transmission, as well as make this area a new target for deep brain stimulation therapy for the treatment of depression [3].

Studies based on psychological tests and their correlation with functional MRI scans reveal the special implication of the subgenual anterior cingulate cortex (area 25) in the genesis of feelings of sadness and grief making the measurement of its activity important in the study of patients in a state of mourning or with depression. Works based on Positron Emission Tomography (PET) imaging, have revealed a metabolic increase together with a decrease in the size of the cortex of this region in subjects with major depressive disorder and bipolar disorder.

This hypothesis has been backed up by postmortem histological studies in which a decrease in grey matter with a loss of glial cells (but not of neurons) have been found in subjects diagnosed with bipolar disorder [4].

Injury to the ACC in humans typically results in an overall cerebral slowdown, a reduction in the production of language and an impossibility to understand one’s errors.

Data which exists in the field of studies over anatomy and metabolites in schizophrenia associate a decrease of N-Acetylaspartic acid together with an increase of glutamine in the anterior cingulate cortex and the medial frontal cortex analyzed with spectroscopy.

They suggest a relation to the susceptibility to suffer a primary episode, not being consistent over time [5,6].

Patients with major depression show deficits in glutamate in the ventral region of the ACC. The disequilibrium in the proportion of glutamine/glutamate in this region has been related to mood disorders [7].

Focal injuries located in the ACC caused by ischemia or hemorrhage have been described as producing episodes of amnesia and akinnesia, mutism, attention deficit, dysexecutive disorder and changes both emotional-affective, and of verbal memory [8]. In the case of acute ischemia in the medial and posterior cingulate cortex, disorders of visuospatial memory and topographical disorientation, together with emotional and behavioral changes, have been described [9].

The cerebral infarction of the anterior and posterior cingulum is described as being associated with difficulty in the recuperation of autobiographical information and the lack of capacity to plan for the future suggesting an important implication about the requirements of memory [10].

Cases of hyperlexia and even echolalia have been described with cerebral infarctions of the left anterior cingulum interpreted as a lack of inhibition in the last intact connections with the supplementary motor area [11].

Massive destruction of the cingulum translates to a complete impossibility to begin any action. Epileptic seizures with origin in the cingulum can occur with intense motor manifestations such as

Figure 1: Simplified description of the areas described by Brodmann in the cingulum according to their cytoarchitectonic composition.

Figure 2: Pre-surgical MRI. The tumor displaces the primary motor cortex posteriorly and thins left structures; ventricle, cingulate and premotor cortex. The patient had slight difficulty in language production.

Figure 3: MRI one month after the intervention and performed after the onset of the manic episode.
intense fright, screams, aggressive verbalizations and/or complete gestural automatisms and hallucinations with an incomplete loss of consciousness.

This knowledge has given us targets within the ACC, for cerebral stimulation therapy with promising results for depression and neuropathic pain with the most recent testing including treatment for anorexia nervosa [12,13].

Anterior cingulotomy has been used for decades to treat cases of chronic pain, typically using radiofrequency in stereotactic conditions, being effective as well on the depressive symptoms that accompany such pain. The result may be due to the inhibition of nociception at the outlet from the somatosensory cortex transmitted by the anterior cingulum to the grey matter in the center of the spinal cord. Studies with an fMRI have revealed an increment of activity in the ACC when nociceptive perception is amplified [14].

Studies of nuclear medicine in patients undergoing those systems of stimulation demonstrate decreases in activity in the medial frontal gyrus, anterior and subcallosal cingulate gyrus, left caudate nucleus, thalamus pulvinar nuclei, and cerebellum. Oppositely, they show an increase of the metabolism in the parahippocampal gyrus, medial temporal lobe, and parietal bilateral lobe. It is not surprising, taking into account the high complexity level of the connections and their extension, that the cingulum cortex plays a role in the pathogenesis of schizophrenia (more concretely the anterior cingulum being the object of multiple studies.) The importance of the loss of grey matter, neural disruption, and the decrease in volume of circumscription is hypothesized.

In the case of our patient, we lack a ruled psychological evaluation, as well as a study of images with metabolic quantification, which could allow us to confirm this causation.

Notwithstanding, we seem to be given a coherent explanation given the ever more extensive literature regarding this fascinating structure.

Conclusion

Taking into account its multiple circuital connections, the cingulate cortex can be considered an axis responsible for emotions, sensations, and actions. Focal injuries are rare, but can help us better understand the function of the cingulate.

References