

RIGHT HEMIPARESIS (Side CC11-2)

CASE 7: Answers:

1. **Speech slurred, dysarthric** - It is crucial to distinguish between aphasia and dysarthria. Aphasia is impairment of *language* functions such as comprehension, naming, fluency, etc. In contrast, dysarthria is simply impaired pronunciation with normal language skills. Thus, our patient has dysarthria but not aphasia. Speech articulation depends on the coordinated functioning of CN V (jaw movement), CN VII (lips and cheeks), and CN XII (tongue). Both LMN lesions of these nerves, and UMN lesions involving the appropriate somatotopic region of the cerebral cortex or corticobulbar pathways can produce dysarthria.

slight flattening of R nasolabial fold - Facial weakness due to UMN and LMN lesions can usually be distinguished by clinical exam. The forehead and orbicularis oculi are supplied by neurons in CN VII which receive *bilateral* input from the motor cortex. In contrast, the lower face is innervated by CN VII neurons that receive predominantly *crossed unilateral* input from the motor cortex. Therefore, UMN lesions cause weakness of the contralateral lower face with sparing of the forehead. This is usually more subtle than the effect of LMN damage to CN VII in which the entire ipsilateral face is often weak. Thus, the mild weakness of the R lower face seen in our patient suggest an UMN lesion involving the L motor cortex or corticobulbar tracts.

R arm and leg weakness and R hyperreflexia - This is strongly suggestive of an UMN lesion in the L motor cortex or corticospinal tract (*review the differences between upper and lower motor neuron damage*).

RAM and FNF slowed on R - Recall that meaningful testing of cerebellar function depends on intact pyramidal, basal ganglia and peripheral nerve pathways. Thus, since our patient has R sided weakness and hyperreflexia, impaired RAM and FNF is most likely due to damage to pyramidal pathways and *not* due to a cerebellar lesion.

2. The patient has evidence of UMN damage affecting the entire R side of the body. If this were a L cortical infarct, it would have to be very large including both middle cerebral artery territory (face/hand) and anterior cerebral artery territory (leg). This should produce sensory as well as motor deficits, and aphasia. It is, thus, more likely that our patient has a lesion, either in the L internal capsule, e.g. a lacune, or of paramedian branches of the basilar (or superior cerebellar) arteries involving the corticobulbar and corticospinal pathways of the L upper pons, 7. Paramedian Upper Pontine Syndrome). If the lesion were in the pons, it would have to be large enough to affect the corticospinal and corticobulbar tracts, yet small enough to preserve the medial lemniscus, MLF and ponto-cerebellar fibers.

Head CT (see slide):

Identify the frontal lobes, temporal lobes, cerebellum, fourth ventricle and pons. Identify the approximate level of this "slice" using a figure showing a mid-sagittal view of the brain. Note the hypodense area in the L pons. This represents edema and cell death produced by an acute infarct.

Clinical Course:

The patient was treated with anticoagulation. With rehabilitation and time his speech improved and he could ambulate with a cane.