CHAPTER

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Approach to a Patient with Hemiplegia and Monoplegia

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INTRODUCTION

Monoplegia and hemiplegia are common neurological symptoms in patients presenting to the emergency department as well as outpatient department.

Monoplegia refers to weakness of one limb (either arm or leg) and hemiplegia refers to weakness of one arm and leg on the same side of body (either left or right side).

There are a variety of underlying causes for monoplegia and hemiplegia. The causes differ in different age groups. The causes also differ depending on the onset, progression and duration of weakness. Therefore, one needs to adopt a systematic approach during history taking and examination in order to arrive at the correct diagnosis. Appropriate investigations after these would confirm the diagnosis.

The aim of this chapter is to systematically look at the differential diagnosis of monoplegia and hemiplegia and outline the approach needed to pinpoint the exact underlying cause.

APPROACH TO THE DIAGNOSIS OF MONOPLEGIA

Causes of Monoplegia

The causes can be classified on the basis of onset- acute or chronic; and involvement of the limb (lower or upper).

Acute onset monoplegia affecting the lower limb can be caused by one of the following conditions:

1. Stroke- affecting anterior cerebral artery territory.
2. Cerebral venous sinus thrombosis, affecting superior sagittal sinus.
3. Trauma-head injury, with contusion in the frontal lobe.
4. Infection, such as granuloma affecting frontal lobe.
5. Trauma to the lumbo-sacral plexus, diabetic lumbo-sacral plexopathy.
6. Functional or psychogenic.

Acute onset monoplegia affecting upper limb can be caused by the following conditions:

1. Stroke, affecting superior division of contralateral middle cerebral artery territory, affecting parietal lobe, or unpaired anterior cerebral artery.
2. Head injury, with contusion in the parietal lobe.
3. Trauma to the brachial plexus.
4. Injury to multiple cervical nerve roots.
5. Functional or psychogenic.

Insidious onset, gradually progressive monoplegia affecting lower limb can be caused by the following conditions:

1. Tumor of the contralateral frontal lobe.
2. Tumor of spinal cord at thoracic or lumbar level.
3. Chronic infection of brain (frontal lobe) or spinal cord (thoracic or lumbar level), such as tuberculous.
4. Lumbosacral-plexopathy, due to diabetes mellitus.

Insidious onset, gradually progressive monoplegia, affecting upper limb, can be caused by one of the following conditions:

1. Tumor of the contralateral parietal lobe.
2. Compressive lesion (tumor, large disc, etc) in cervical cord region.
3. Chronic infection of the brain (parietal lobe) or spinal cord (cervical region), such as tuberculous.
4. Tumor of the brachial plexus.

Causes of hemiplegia

Acute onset hemiplegia can be caused by one of the following conditions:

1. Ischemic or hemorrhagic stroke, affecting contralateral cerebral hemisphere, internal capsule, brainstem or ipsilateral upper cervical cord.
2. Cerebral venous sinus thrombosis with venous infarction of contralateral cerebral hemisphere.
3. Acute central nervous system infection, such as meningitis or encephalitis, brain abscess, granulomatous infections.
4. Head injury, causing contusion/bleeding in the contralateral cerebral hemisphere, internal capsule, basal ganglia, or brainstem.
5. Bleeding into a brain tumor on the contralateral side.
6. Demyelinating illness, such as ADEM (acute disseminated encephalomyelitis) or MS (multiple sclerosis).
7. Todd's paresis.

8. Metabolic derangements, such as hypoglycemia, hyperglycemia or hyponatremia.
9. Functional or psychogenic.

Insidious onset, gradually progressive hemiplegia can be caused by one of the following conditions:

1. Brain tumor, affecting cerebral hemisphere, internal capsule, basal ganglia or brainstem.
2. Tumor of the spinal cord in cervical region.
3. Chronic infections of the brain, such as tuberculosis, hydatid cysts, etc.
4. Mill's hemiplegic variant of motor neuron disease (MND).

Above, we have seen the list of differential diagnosis for acute onset as well as insidious onset, gradually progressive conditions causing monoplegia and hemiplegia. The list mentions the most common causes encountered in routine clinical practice, and rare conditions have not been listed. Still, there are a lot of conditions/diseases to be considered while evaluating a case. Therefore, it is important to tailor the history and clinical examination to arrive at the correct diagnosis. A thorough history taking and examination would also lead us to order appropriate investigations, in order to clinch the diagnosis.

In the subsequent sections, we would discuss in detail about the approach to monoplegia and hemiplegia. For ease of discussion, we would separately consider the acute onset and chronic conditions.

Acute onset monoplegia affecting lower limb

In this section, we would consider the conditions that cause acute onset weakness of the lower limb, evolving over hours to days. The site of lesion in a patient with lower limb weakness can vary from peripheral to central nervous system.

Lesions in the central nervous system more often cause acute onset monoplegia affecting the lower limb. Among these, the most common are the vascular syndromes. Leg-predominant weakness with stroke is due to contralateral anterior cerebral artery (ACA) infarction in only 25% of cases. More often, it is related to lesions in the contralateral corona radiata or internal capsule, in the territory of the anterior choroidal artery or perforators (30%), or in the brainstem (25%) and can occur with lesions in the middle cerebral artery territory or with thalamic hemorrhage.[1] Regarding lesions of the medial aspect of the frontal lobe, those restricted to precentral gyrus of the paracentral lobule cause contralateral leg weakness. In rare cases, lacunar infarction of the corona radiata can cause ipsilateral weakness of the leg.[2] This can happen due to anomalous pyramidal fibers with ipsilateral innervation or due to reorganization of pyramidal fibers due to old stroke. Ipsilateral pure motor monoparesis of the leg can also rarely occur with lateral medullary infarction.[3] This occurs due to involvement of corticospinal tract fibers innervating the lower limb caudal to pyramidal decussation.

The clue towards a likely vascular cause is the sudden onset of weakness, and the exact time of onset of weakness can be obtained from the history. The exception includes "wake up strokes", where the patient goes to sleep without any deficits and wakes up with a new onset weakness of the leg.

The diagnosis of stroke can be confirmed by doing a computerized tomography (CT) scan. Hemorrhage or bleeding is easily picked up on the CT scan. Acute infarction may be missed on the CT scan during initial few hours. Therefore, magnetic resonance imaging (MRI) with diffusion-weighted imaging is the modality of choice for diagnosis of acute ischemic stroke causing isolated monoparesis.[4]

Acute onset weakness of contralateral leg can also occur with cerebral venous sinus thrombosis (CVST) with venous infarction affecting medial frontal lobe. Weakness of contralateral leg can also rarely occur due to granulomatous infections affecting the medial frontal lobe and head injury causing contusion/hemorrhage of the precentral gyrus.[5]

In peripheral nervous system, involvement of lumbosacral plexus (lumbo-sacral plexopathy) is the commonest cause of acute onset unilateral lower limb weakness. The most characteristic feature of lumbo-sacral plexopathy is the presence of pain along with weakness of the lower limb. Pain may be severe and poorly localized. It is described as lancinating, aching or burning pain. It may involve thigh, leg and gluteal regions. There is predominant involvement of proximal group of muscles, as compared to the distal group of muscles. In addition, there may be loss of sensations too. Weight loss is noted in most patients. Clinical examination would confirm the weakness of proximal group of muscles. If patients present after a few weeks, there may be wasting of thigh and leg muscles. Knee jerk is absent. Ankle jerk may be present, however it may be sluggish or absent, if the patient has associated peripheral neuropathy. The diagnosis of lumbo-sacral plexopathy can be confirmed by nerve conduction studies and needle electromyography. A clinical clue for a plexus lesion (as against multiple peripheral nerves and nerve roots) is the motor and sensory involvement in the distribution of two or more peripheral nerves and two or more nerve root territories in the same limb. The common causes for acute onset lumbo-sacral plexopathy are diabetes mellitus (also called as Bruns Garland syndrome), viral infections, hemorrhage, vasculitis and trauma. In diabetic lumbo-sacral plexopathy, both lower limbs may be affected, however, one side is more affected than the other side.

Acute onset monoplegia affecting upper limb

Lesions in the central as well as peripheral nervous system can cause acute onset monoplegia affecting upper limb.

The most common cause of acute onset isolated arm weakness on one side is stroke. However, it should be noted that it is uncommon for stroke to present with isolated arm weakness without associated face

or leg weakness. Stroke presenting with isolated arm monoparesis may be misdiagnosed as a peripheral nerve disorder, because of absence of pyramidal tract signs or the involvement of the speech, face or lower limbs. Distal arm monoparesis is an unusual form of cortical infarct, which occurs in the parietal lobe or central sulcus region, comprising less than 1% of stroke cases.[6]

Again, the acute onset is the key to suspicion of stroke. The diagnosis can be confirmed by diffusion-weighted imaging on MRI brain.

Acute onset monoplegia of upper limb can also occur due to peripheral nervous system involvement. The most common cause in this category is brachial plexopathy. Brachial plexus lesions that can result in acute onset weakness include injury/trauma, infections, hemorrhage, etc. The classical features include arm and shoulder pain, weakness and sensory disturbances. Weakness affects proximal group of muscles more than the distal group. Most patients also have wasting of muscles. Deep tendon reflexes of the affected upper limb may be sluggish or absent. A clinical involvement in the distribution of more than two spinal nerves and more than two peripheral nerves is a strong indicator of brachial plexus lesion. The diagnosis of brachial plexopathy can be confirmed by doing nerve conduction studies with Erb's point stimulation and needle EMG. MRI is also valuable in diagnosing brachial plexus lesion. An abnormal hyperintense signal on MRI suggestive of inflammation may be seen in brachial neuritis caused by varicella zoster virus.[7]

While evaluating a case of acute onset weakness of leg or arm, we must also consider a diagnosis of psychogenic or functional basis for the same. Psychogenic monoplegia is common in younger people, and is more common in women. Detailed history may reveal stress factors related to personal relationships, job or studies in the affected individual. Psychogenic weakness may be associated with primary gain (deriving attention towards illness) and secondary gain (avoidance of stress factor). Examination of a patient with psychogenic monoplegia may reveal inconsistent findings. Power of the affected limb may change, when repeatedly tested. Also, if a person with psychogenic weakness of leg were made to stand by self, she would avoid injury while falling. Hoover's sign is very important while examining a person suspected to have psychogenic leg weakness. Palm of the examiner's hand is placed under the heel of patient's normal leg; and the patient is asked to raise the weak leg. In a person with true weakness, pressure on the palm would be felt due to downward movement of the normal leg, however, in a person with psychogenic weakness, no such mechanical pressure is felt on the palm.

Slowly progressive monoplegia affecting lower limb

A variety of conditions can cause insidious onset, slowly progressive weakness of one leg. The site of lesion causing such isolated progressive weakness of one leg can extend from brain, spinal cord to nerve roots and plexus. Therefore, a systematic approach is needed while evaluating such a case in order to correctly localize and diagnose.

Tumors of the brain or spinal cord are the common causes of slowly progressive isolated monoparesis of lower limb. The person presents with weakness of one leg, which often starts distally in the foot, and over a few weeks to months, spreads to involve the proximal group of muscles. This is because the lower limb fibers are located laterally in the spinal cord. Presence of back pain and radicular pain may point towards a spinal cord lesion, most probably a tumor. Examination, in addition to motor weakness, may reveal loss of sensations in the affected leg. Bladder symptoms are unusual with unilateral leg weakness. Diagnosis is almost always delayed in a case of neoplastic spinal cord compression, when the patient presents with unilateral leg weakness.[8] However, we need to suspect a spinal cord compression even in the absence of sensory level, as 23% of cases may present with unilateral weakness.[9] An MRI of the spine with contrast is valuable in confirming the diagnosis of spinal cord tumor. The common spinal cord tumors include meningioma & schwannoma (benign tumors) and metastases & glioma (malignant tumors).

Tumors of the brain affecting precentral gyrus, medial frontal lobe and paracentral lobule may present with monoparesis of contralateral leg.

Tuberculosis of spine and tuberculous meningitis (TBM) can also present with subacute or chronic weakness of one leg. A case of 37-year old woman is reported where she presented with spastic weakness of right leg of six years duration.[10] The cause was found to be tuberculous syringomyelia, as she had a past history of TBM and had received anti-tuberculous therapy for that.

Among the peripheral nervous system causes, the most common cause for unilateral leg weakness would be lumbo-sacral plexopathy. The clinical features of lumbo-sacral plexopathy have already been discussed above. The underlying etiology for slowly progressive lumbo-sacral plexopathy could be metastases, neurofibroma & nerve sheath tumors, infections and diabetes mellitus.

Slowly progressive monoplegia affecting upper limb

The causes for slowly progressive weakness affecting upper limb would be similar to that of lower limb. This would include spinal cord tumors affecting the cervical cord region, and tumors affecting the parietal lobe of brain. Similarly, infections such as tuberculosis of these areas could also result in unilateral arm weakness.

Among the peripheral nervous system causes, involvement of brachial plexus would present with unilateral arm weakness. Causes of slowly progressive brachial plexopathy include tumors, metastases, infections, etc. Diabetes could also cause brachial plexopathy, though the involvement of lumbo-sacral plexus is more common with diabetes.[11] In this condition, patients complain of shoulder and arm pain, and there is weakness of hand and forearm muscles.

Another cause to be considered in a patient presenting

with slowly progressive weakness of one limb (upper or lower limb) associated with wasting is monomelic amyotrophy.[12] The characteristic clinical features are insidious onset in the second and third decades, male preponderance, sporadic occurrence, wasting and weakness confined to one limb, and absence of involvement of the cranial nerves, cerebrum, brain stem, and sensory system. The electromyographic features, along with histologic features of neurogenic atrophy, are suggestive of an anterior horn cell lesion. The slow progression of illness for two to four years followed by a stationary phase is observed. There is no clinical evidence of involvement of the other three limbs even in patients with long-standing illness of ten to 15 years' duration. Monomelic amyotrophy affecting upper limb is also called as Hirayama disease. MRI cervical spine in these cases show asymmetrical lower cervical cord atrophy in about half the cases.[13]

Acute onset hemiplegia

Acute onset hemiplegia is among the commonest presentations in the emergency department. The underlying cause of acute hemiplegia can be varied, however, brain stroke remains the commonest cause. Also, it is of utmost importance to make a quick diagnosis of acute ischemic stroke, as the only approved therapy for acute stroke (thrombolytic therapy with tissue plasminogen activator) can be given only within the first four and half hours after stroke onset.

The most important consideration in the diagnosis of stroke is the abruptness of onset of symptoms. The typical history of a patient with stroke could be as per the described case here: "Mr KS went for a morning walk as usual and returned in half an hour. He sat down in balcony reading a newspaper and having a cup of tea. All of a sudden, the newspaper fell off his right hand, and he could not hold the cup of tea. He slumped off the chair to the ground. He could not use his right hand or leg. He tried calling for his wife but could not." This is the typical history of a patient with stroke in left middle cerebral artery (MCA) territory. In fact, the commonest site of arterial occlusion in a patient with hemiplegia is MCA territory. It is also important to enquire about the history of transient ischemic attacks (TIAs), as several patients would have had one or more TIA in previous 30 days. The risk factors for stroke such as diabetes, hypertension, dyslipidemia, hyperhomocystinemia, cardiac disease and smoking should also be looked into. An enquiry about any accompanying symptoms should also be made. For example, presence of aphasia localizes the site of occlusion to left MCA territory.

When upper and lower limbs are equally affected, it is called as dense hemiplegia, and is typical of infarcts in internal capsule. Motor power is usually grade 0 or 1. On the other hand, unequal involvement of limbs point to a lesion in cerebral cortex. Upper limbs are more affected than the lower limbs in a MCA territory infarction, whereas lower limbs are more affected than the upper limbs in ACA territory infarction.

In the initial period after stroke, upper motor neuron signs are absent. There is hypotonia of affected limbs and deep tendon reflexes are absent. This state is called as cerebral shock. This state should not be confused with a lower motor neuron lesion. Presence of aphasia, visual field defects, cortical sensory loss and extensor plantar response may help in localizing the lesion to cerebral cortex. The typical upper motor neuron signs develop after a few days of stroke onset.

A note should be made of the cranial nerve involvement. Cranial nerve involvement on the same side of hemiplegia localizes the lesion to the cerebral cortex or internal capsule. Whereas, involvement of cranial nerves on the side opposite to that of hemiplegia (crossed hemiplegia), localizes the lesion to the brainstem.[14] In brainstem, the localization depends on the cranial nerve involvement. Third cranial nerve involvement along with contralateral hemiplegia localizes the lesion to the midbrain (Weber's syndrome). Involvement of sixth and seventh cranial nerves along with contralateral hemiplegia localizes the lesion to pons. Ipsilateral hypoglossal palsy and contralateral hemiplegia localizes the lesion to medial medulla.[15]

Acute onset hemiplegia may also occur due to spinal cord small infarctions in the upper cervical cord. Hemiplegia in these cases is on the side of lesion. Sensory examination may also reveal ipsilateral loss of joint position and vibratory sensations, and pain and temperature impairment on the contralateral side.[16] The artery affected is usually anterior spinal artery.

In a patient suspected to have stroke, MRI brain or spinal cord (as per the clinical findings) is the imaging modality of choice for confirming the diagnosis. Diffusion weighted sequences are the most preferred and valuable in detecting acute infarcts.

Bleeding into a tumor in brain is another cause of acute hemiplegia. History of headache for a few weeks to months may point towards a neoplastic etiology. The tumor may be located in cerebral cortex, basal ganglia, thalamus or brainstem. CT or MRI scan of brain would confirm the diagnosis.

In addition, we also need to consider demyelinating illnesses and infections as the alternative possible causes for acute onset hemiplegia. Among demyelinating illnesses, acute disseminated encephalomyelitis (ADEM) is more common. ADEM is known to present as acute onset hemiplegia, and may mimic stroke.[17] Patients with ADEM are relatively younger & healthy, and may give a history of fever two weeks prior to the onset of hemiplegia. MRI brain is helpful in differentiating ADEM from MS and brain stroke.

In our country, cerebral venous sinus thrombosis (CVST) is another important diagnostic consideration in patients with acute onset hemiplegia. Almost always, patients complain of headache for a few days preceding the hemiplegia. Most patients are young and the risk factors may be present. These include pregnancy, post-partum

period, use of hormonal pills, nephrotic syndrome, hypercoagulable states, etc. MRI with MR venogram of brain would confirm the diagnosis. Hemiplegia in CVST usually occurs due to hemorrhagic infarctions in the cerebral cortex.

Infections such as herpes encephalitis and tuberculous meningitis (TBM) can also rarely cause acute-onset hemiplegia mimicking stroke. In patients with herpes encephalitis, sudden onset stroke-like hemiparesis has been reported.[18] In suspected case of TBM, patient may complain of sudden onset hemiplegia, often due to vasculitis affecting TBM. The commonest artery affected in TBM is MCA. Hemiplegia has also been reported with dengue and falciparum malaria. An infective cause should be suspected when the patient with hemiplegia has a fever. Appropriate investigations should be done to confirm or exclude these infections in a patient presenting with hemiplegia.

Enquiry should be made about any episode of seizure prior to the onset of hemiplegia. The most common cause of hemiplegia after a seizure could be Todd's paresis. Todd's paresis may occur after a generalized or partial seizure, however, it is more common after a generalized seizure. The weakness usually lasts for a few hours, however, it may last for upto 36 hours in some cases.[19] Weakness may occur after the first seizure or after many years of seizures and does not appear after every seizure. It should be noted that seizure might also occur in the setting of brain stroke, where it is more common with embolic strokes and venous sinus thrombosis.

Metabolic derangements should also be kept in mind while dealing with acute hemiplegia. Hypoglycemia (low blood glucose) is a well known, but uncommon cause of hemiplegia. Patients with hypoglycemia can present with acute hemiplegia associated with other signs such as aphasia, which can mimic strokes.[20] MRI brain shows infarctions. However, administration of glucose and correction of glucose rapidly reverses the hemiplegia and other neurological deficits. Hemiparesis has also been reported in cases of hyponatremia (low serum sodium) apparently due to central pontine myelinolysis.[21]

Functional or psychogenic cause of hemiplegia is also fairly common. The typical scenario is a young woman brought to the hospital with acute onset hemiplegia, with seemingly no risk factors for stroke. History may reveal a stressful mental state. Examination is very important in confirming a psychogenic cause of hemiplegia. When we raise the arm and let it fall, the person typically avoids letting it fall over the face to avoid getting hurt. Similarly, when we make the patient stand and leave, the patient falls "carefully" to avoid getting hurt. While doing these maneuvers, we should be cautious to avoid injury to the patient, if it were not due to a psychogenic cause. Another useful clinical sign is Hoover sign, as described above in the section on monoplegia. Motor-evoked potentials may be done, which are normal.[22] As expected, MRI of brain would also be normal.

Slowly progressive hemiplegia

In a patient presenting with insidious onset and slowly progressive hemiplegia, brain tumors are the most important consideration. In a series on brain tumors presenting to ER, about 25% of them had hemiparesis.[23] Patients may complain of headache of raised intracranial tension type. Headaches are worse on awakening and are associated with projectile vomiting. There may be visual obscuration at the peak of headaches. CT or MRI brain scan with contrast should be performed in suspected cases to confirm the diagnosis. The commonest brain tumors are metastases and gliomas. Other tumors that can cause hemiparesis are lymphoma, meningioma, neurofibroma, etc. The location of tumor can be in cerebral cortex, basal ganglia, thalamus, and brainstem.

We must also consider infections such as tuberculoma in a patient with slowly progressive hemiplegia. Hemiplegia may also be an initial manifestation of CNS tuberculosis.[24] It is important to suspect a diagnosis of tuberculosis, if the patient has fever, headache and weight loss as the symptoms. Institution of anti-tuberculous treatment may reverse the hemiparesis. Neurosarcoidosis can also present with various neurological manifestations, including hemiparesis.[25]

We should also consider a diagnosis of Mill's variant of amyotrophic lateral sclerosis in a person presenting with slowly progressive hemiplegia.[26] Pure motor system is affected, with sparing of sensory and autonomic symptoms. Clinical examination would reveal a combination of lower and upper motor neuron signs. Needle EMG would confirm the diagnosis.

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