POST-TRAUMATIC SYRINGOMYELIA IN A CASE OF CRANIOCEREBRAL TRAUMA

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ABSTRACT

The authors report a case of a 52-year-old man, with a history of craniocerebral trauma, followed by left parietal cranial epidural abscess and parietal bone craniectomy, who was hospitalized for loss of consciousness and secondary generalized tonic- clonic seizures. The case particularities are the old craniocerebral trauma, acute ischemic stroke, appearance of pain at the scapulohumeral joint with atrophy of interosseous muscle of hands; the cervical spine MRI showed syringomyelia syrinx C2-C4.
Key words: syringomyelia; craniocerebral trauma; parietal cranial epidural abscess; parietal bone craniectomy

We report a case of 52-year-old male patient who was referred to the emergency room, Emergency Hospital Bucharest for loss of consciousness and secondary generalized tonic-clonic seizures.

Medical history: Craniocerebral trauma in 1972, followed at 2 weeks by left parietal cranial epidural abscess. Parietal bone craniectomy was performed during the surgical procedure. Three weeks after craniectomy he developed right hemiparesis and dysarthria, he was reoperated and completely recovered. In 1983 the patient presented multiple episodes of loss of consciousness (diagnosed as syncope!?). In 1990 right hemiparesis (fingers, forearm, arm and secondary the inferior limb) developed insidiously. The brain CT performed in 1998 showed left parietal craniectomy. Under this level it was found encephalic post operator status without any compressive hydric liquid (Fig.1). In 2001 the brain CT was repeated but no major changes had been found. (Fig.2). In 2007 patient developed disturbance of equilibrium, disturbance of deglutition, hyperphagia, disturbances of speech, hyperhidrosis of cervical region (frequent during in day). Also he was diagnosed with hepatitis virus C.

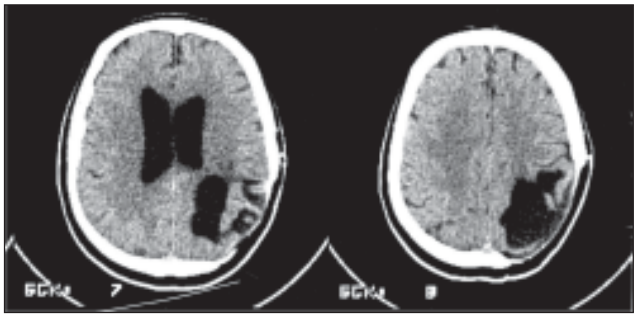

Figure 1. *Brain CT from 1998*

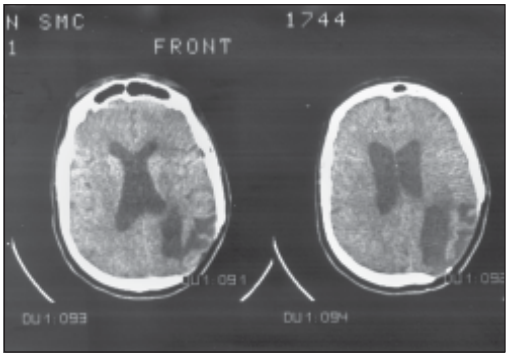

Figure 2. *Brain CT from 2001*

On hospital admission clinical examination revealed no fever, normal skin and mucous membrane, blood pressure = 140/80 mmHg. The neurological examination showed no meningeal signs, normal cranial nerves, impossible orthostatic position and walking due to motor disorder, spastic right hemiparesis, left onset hemiparesis (superior > inferior), dysarthria, bilateral Babinski, jerk osteotendinous reflexes on left hemicorp and

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diminished osteotendinous reflexes on right hemicorp, no sensibility disturbance, no sphincter disturbance, no syringomyelic dissociation, bilateral interosseous muscle atrophy, bilateral scapulo-humeral atrophy.

The brain CT revealed: 56/34 mm hypoattenuation aria in left parietal zone, with missing parietal bone; old cerebral deterioration 3/0.88 mm situated right temporal; no high density of hemorrhage intra or extra cerebral. The brain CT repeated at 48 hours did not show any changes (Fig. 3-4).

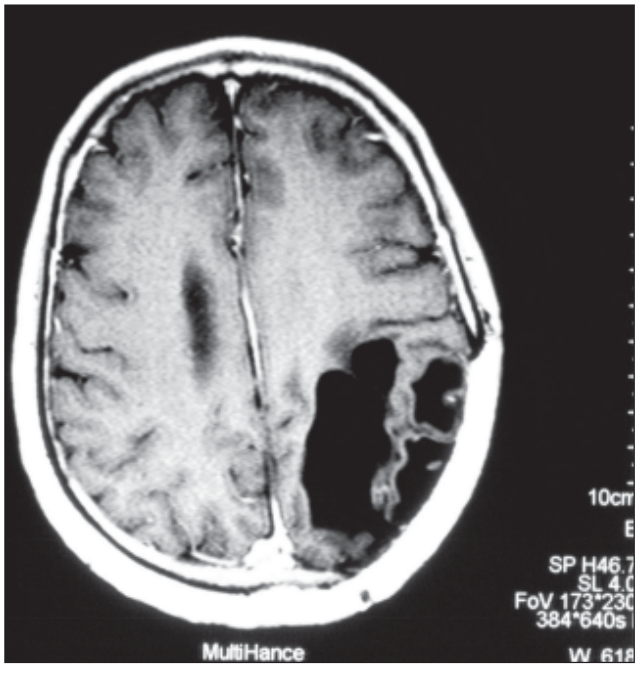


Figure 3. *Brain CT from 2008*

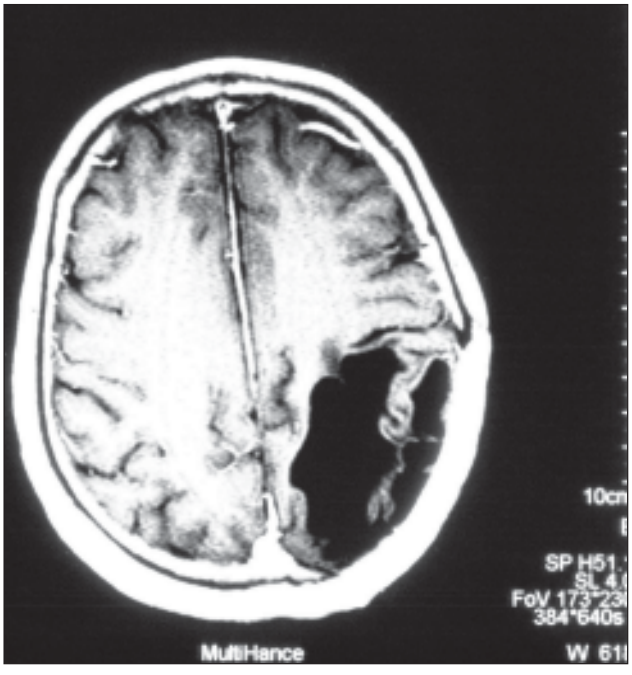


Figure 4. *Brain CT from 2008*

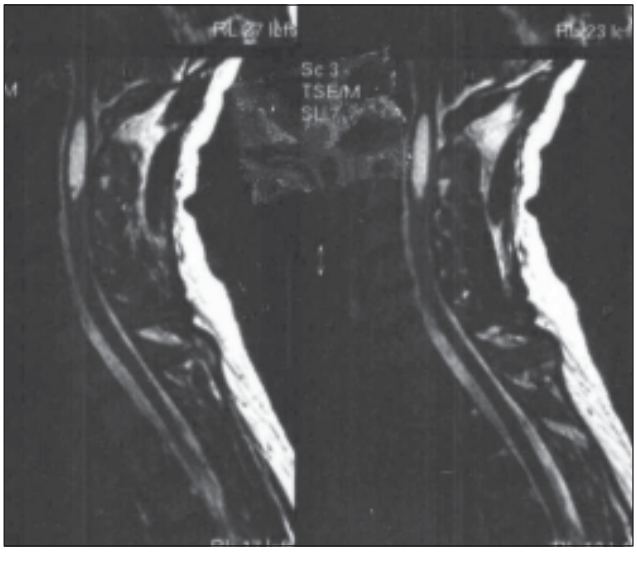


Figure 5. *MRI of cervical spine T2*

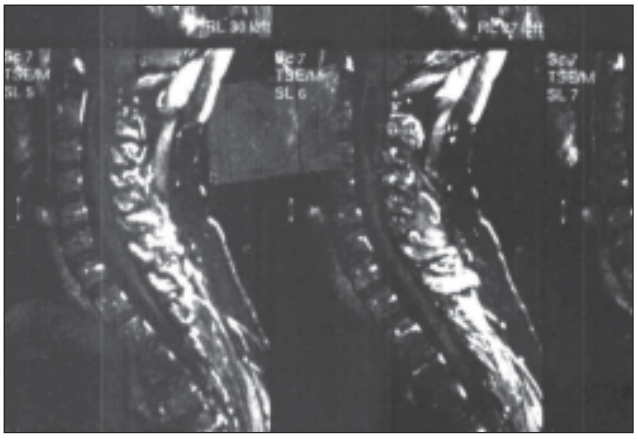


Figure 6. *MRI of cervical spine T1*

Cervical radiography was normal.

The patient complains of paresthesias in superior limbs and pains in cervical spinal column, which exacerbated from the hospitalization moment.

MRI of cervical spine was performed, and this examination revealed syringomyelic cavitation with axial diameter of 11mm and craniocaudal diameter of 4,4 mm who extended between C2-C3 vertebra till C3-C4 vertebra with homogeneous liquid similar to CSF, no gadolinium enhancement- the image is suggestive for syringomyelia (Fig.5, 6, 7).

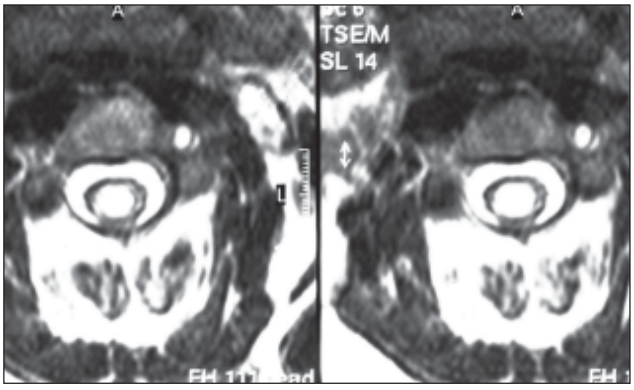


Figure 7. *MRI of cervical spine – transversal section*

EKG - SR without ST-T elevation.

The Lab tests where normal.

In hospital patient received treatment with Ringer solution, Manitol 20%(Mannitolum), Furosemid (Furosemidum), Clexane 0.6 mg(Enoxoparinum), Aspenter 75 mg(Acidum acetylsalycilicum), Omeprazol(Omeprazolum), Algocalmin (Metamizolum Natricum).

The patient was discharged from hospital and presented to a neurosurgery clinic for surgical intervention.

DISCUSSION

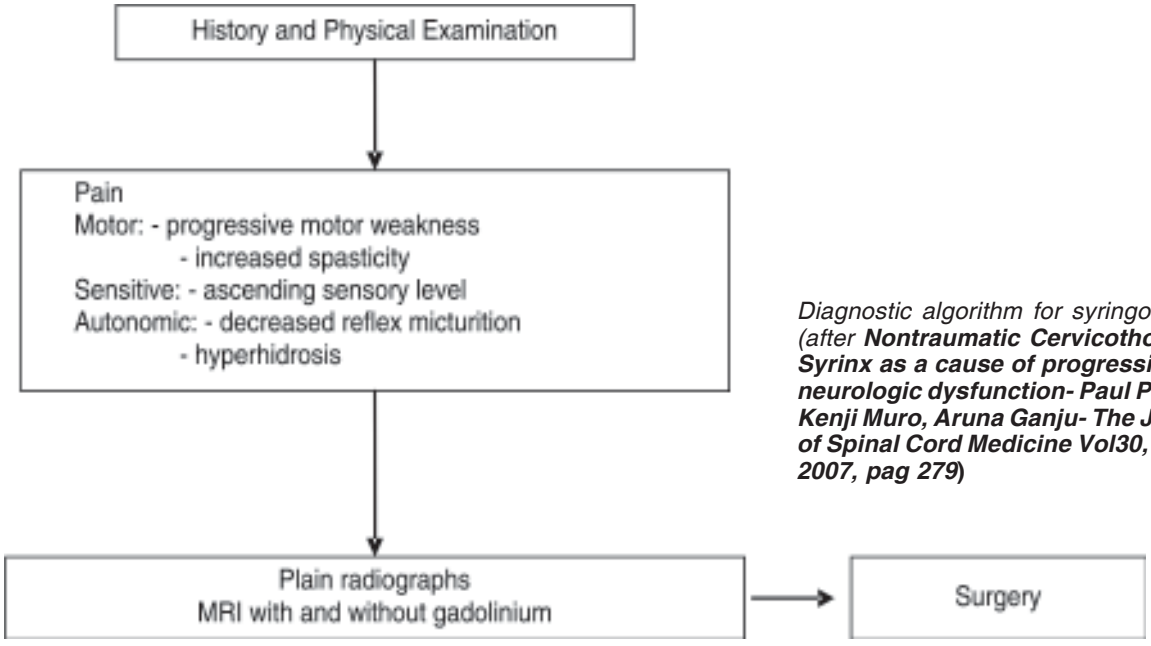
First described by Bastian in 1867, posttraumatic syringomyelia (PTS) refers to the development and progression of a cavity, filled with cerebrospinal fluid (CSF), situated within the spinal cord. Spinal cord cavitation after traumatic spinal cord injury (SCI) is relatively infrequent, but potentially devastating. Later, some reports showed the clinical appearance of syringomyelia, many years after traumatic SCI, often insidious progression of pain and loss of sensorimotor function. If left untreated, the natural evolution of PTS is loss of function, chronic pain, respiratory failure, or death (1, 2).

Syringomyelia is characterized clinically by painless weakness and wasting of the hands and arms (brachial amyotrophy) and segmental sensory loss of dissociated type (loss of thermal and painful sensation with sparing of tactile, joint position, and vibratory sense) (3).

Classic clinical elements are:

- pain, which is the most commonly reported symptom;
- motor:
- increased spasticity;
- segmental weakness and atrophy of the hands and arms;
- loss of some or all tendon reflexes in the arms;
- sensitive:
- segmental anesthesia of a dissociated type over the neck, shoulders, and arms (loss of pain and thermal sense with preservation of the sense of touch);
- autonomic;
- decreased reflex micturition;
- hyperhidrosis (1,2,3,4,7).

The patient had pain in his arms, right spastic hemiparesis and left hemiparesis recently onset, decreased tendon reflexes, progressive weakness, hyperhidrosis, decreased reflex micturition, but without segmental anesthesia. Pain was localized to the syrinx site like a radicular pain. Posture change or Valsava-like maneuver exacerbated the symptoms. This phenomena is in line with the hydrodynamic theories of syringomyelia genesis (4).



Diagnostic algorithm for syringomyelia (after ***Nontraumatic Cervicothoracic Syrinx as a cause of progressive neurologic dysfunction- Paul Porensky, Kenji Muro, Aruna Ganju- The Journal of Spinal Cord Medicine Vol30, 3 Nov 2007, pag 279****)*

Diagnostic algorithm for syringomyelia (after Nontraumatic Cervicothoracic Syrinx as a cause of progressive neurologic dysfunction – Paul Porensky, Kenji Muro, Aruna Ganju – *The Journal of Spinal Cord Medicine* Vol. 30, 3 Nov 2007, pag 279)

Our diagnosis was:
- Cervical Syringomyelia at C2-C4 vertebra secondary to Craniocerebral trauma
- Craniocerebral trauma with operated left epidural cerebral abscess
- Acute ischemic stroke of right sylvian artery
- Spastic right hemiparesis
- New onset of left hemiparesis

Difficulties in diagnosis: on old known craniocerebral trauma, the patient was presented with an acute ischemic stroke on vascularization territory of right middle cerebral artery. The motors signs are from ischemic stroke. The patient didn't have any cervical trauma, only with craniocerebral trauma being known since 1972. The neurological examination did not reveal thermalgesic dissociation. The appearance of pains at the scapulohumeral joint with atrophy of interosseous muscles of hands could suggest cervical spine pathology. The cervical spine MRI showed syringomyelia syrinx C2-C4. The symptomatology aggravated in recent years being augmented by the syringomyelic lesions. MRI and complete neurological examination enabled us to diagnose cervical syringomyelia C2- C4. The patient has motor disturbance due to accentuated upper motor signs (ischemic stroke) and high cervical suffer.

Clinical differential diagnosis for this case:

Spinal Epidural Abscess: The patient had pains in his arms, recently onset of right spastic hemiparesis and left hemiparesis, bilateral Babinski, jerk osteotendinous reflexes on left hemicorp and diminished osteotendinous reflexes on right hemicorp, but he did not manifest nuchal rigidity, sphincter dysfunction, fever or localized pain to percussion or palpation of spinal cord.(5)

Acute Inflammatory Demyelinating Polyradiculoneuropathy: the patient had progressive weekness, hyperhidrosis of cervical region, pains in scapulohumeral joints, jerk osteotendinous reflexes on left hemicorp and diminished osteotendinous reflexes on right hemicorp, but he did not show paresthesias in the distal limbs, urinary retention or fluctuations in heart rate (6).

MRI examination differential diagnosis was made with myelomalacia, which is hypointense on T1 and hyperintense on T2 compared with normal cord. Also post traumatic hygroma was another possibility of diagnosis, but the MRI findings are hyperintense on T1, T2, FLAIR. Multiple sclerosis was another differential diagnosis, but it needs multiple lesions hyperintense on T2.

CONCLUSION

The proper neurological examination with careful assessing of the clinical history and imagistic examination helped us to diagnose both the sechelar cerebral lesion, acute ischemic stroke lesion and the cervical syringomyelia. The onset through the years of new neurological signs (pains, hypotonia, modification of reflexes) and the results of imageries (brain CT and cervical spine MRI) helped us to complete the diagnosis (Cervical Syringomyelia at C2-C4 vertebra secondary to craniocerebral trauma; craniocerebral trauma with operated left epidural cerebral abscess; acute ischemic stroke of right sylvian artery; spastic right hemiparesis; new onset of left hemiparesis).

REFERENCES

1. **Lance Goetz** – Posttraumatic Syringomyelia – Jan 26, 2007
2. **B. Schurch, W. Wichamann, A. B. Rossier** – Post-traumatic syringomyelia(cystic myelopathy): a prospective study of 449 patients with spinal cord injury – *Journal of Neurology, Neurosurgery and Psychiatry* 1996; 60-67
3. **Adams and Victor's** – Principles of neurology – 8th edition; 1084
4. **Paul Porensky, Kenji Muro, Aruna Ganju** – Nontraumatic Cervicothoracic Syrinx as a Cause of Progresseive Neurologic Dysfunction – *The Journal of Spinal Cord Medicine* vol 30, no.3, 2007; 279
5. **J Stephen Huff** – Spinal Epidural Abscess – Jan 17, 2007
6. **Tarakad S Ramachandran** – Acute Inflammatory Demyelinating Polyradiculoneuropathy – Jul 10, 2006
7. **Hassan Ahmad Hassan Al-Shatoury** – Syringomyelia- Sep 24, 2008
8. **Ligia Opris** – Cerebral imaging by magnetic resonance imaging – 2004