A case of communicating rami between the median and musculocutaneous nerves

Sir,

One read with great interest the article “A case of communicating rami between the median and musculocutaneous nerves passing through the substance of an accessory head of biceps brachii” by Indrasingh et al.\[1\] This cadaveric study details an anatomical variation of the musculocutaneous nerve in the arm and adds to the known variations of the musculocutaneous and median nerves in the human body.\[2\] As the authors clearly state, this has repercussions in the diagnosis and treatment of peripheral nerve injuries. The authors are to be congratulated for their work.

One is compelled to comment on in the original article.\[1\] The structure marked as the ulnar nerve (UN), on very critical inspection, appears to be arborizing in the axilla superficial to the axillary vein. This probably implies that the structure may actually be the medial cutaneous nerve of the arm or forearm, and the axillary branch an intercostobrachial nerve contribution.\[3\] A deeper unlabeled structure that lies medial to the neurovascular bundle in the arm may actually be the ulnar nerve.

This mimicry is not atypical, but has significant implications. For peripheral nerve surgeons, both the ulnar nerve and the musculocutaneous nerves carry significant importance in the management of upper trunk injuries. The former is a critical donor nerve, whereas the latter has two recipients (nerve to biceps and nerve to brachialis) that may be targeted by the Oberlin procedure and the double fascicular transfer, respectively.\[4,5\] As this paper points out, the nerve to brachialis may come off the median nerve. As this comment indicates, any confusion regarding the ulnar nerve, which is a mixed nerve, and the medial brachial/antebrachial cutaneous nerve, which is a sensory nerve, will lead to a catastrophic failure of the Oberlin procedure (ulnar nerve fascicle to nerve to biceps transfer). To avoid this, one has to be constantly aware of the fact that the ulnar nerve ordinarily gives off no major branches in the axilla or
in the arm. At surgery it must be stimulable using an intraoperative nerve stimulator.

If any doubt exists, the entire plexus should be dissected out to account for the several variations that may exist, as the authors have so clearly pointed out. This gains additional importance as operative exposures for peripheral nerve reanimation become more minimal.

This article has several teaching points and I would like to commend the authors for allowing these to come forth.

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References


Primary amoebic meningoencephalitis

Sir,

Primary amoebic meningoencephalitis (PAM) is an acute, fulminant and rapidly fatal central nervous system (CNS) infection caused by a thermophilic ameboflagellate Naegleria fowleri.[1] Infection commonly occurs in healthy children and young adults with a history of recent exposure to warm fresh water in ponds, swimming pools and man-made lakes, but older people are also affected. Infection is usually acquired during swimming or nasal irrigation by contaminated water. PAM is clinically indistinguishable from bacterial meningitis. Diagnosis is made by examination of cerebro-spinal fluid (CSF) wet mounts for motile trophozoites.[2] In recent years, the number of cases reported have increased, probably due to greater awareness of disease or due to development of more rapid, highly sensitive and specific diagnostic tests.[3] About 300 cases have been reported so far with high case fatality rate of around 95%.[4] There has been advances in understanding of pathophysiology of this grave disease. Many therapeutic options have been tried, but with variable success and evidences are limited to small case series.[5‑9]

For the first time, we are reporting a case of PAM in Nepal. The patient died despite intensive multi-drug therapy. A 51-year-old gentleman weighing around 50 kg, fisherman by occupation, presented with headache, vomiting, abnormal behavior and irrelevant talking of 4 days duration. Cerebrospinal fluid (CSF) opening pressure was 380 mmH2O.

On CSF analysis, total cell count was 800/mm3 with 82% neutrophils, sugar was 20.5 mg/dL and protein was 171 mg/dL. Corresponding serum sugar was 156 mg/dL. There were no red blood cells (RBCs) seen. With the provisional diagnosis of bacterial meningitis, intravenous Ceftriaxone 2 gm twice daily was started. Gram staining and culture of CSF were negative for bacteria. There was progressive deterioration in neurological status and the patient was intubated for fall in Glasgow Coma Scale (GCS) score to 7/15. CT scan of head was unremarkable. Detail review of the case revealed his hobby of swimming and his recent history of exposure to fresh water of river about a week before his illness. Lumbar puncture was repeated on day 4 of presentation to hospital. CSF was cloudy and slightly hemorrhagic with 300 RBCs/mm3. Wet mount direct microscopic examination of the centrifuged sample revealed motile amoebae [Figure 1]. The trophozoites were approximately 10‑14 µm in size and contained a nucleus with a large karyosome. Enflagellation test was performed by adding 0.5 mL CSF to 2 mL distilled water on a sterile flat-bottom plate and was incubated at 37°C. Microscopy done at 15 minute intervals revealed amoebae moving in a distinctive flagellar pattern.

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