Letter to the Editor

Transient neurological deficit immediately after central venous line removal: a poorly explained finding

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The sudden onset of a major neurological deficit in concomitance with the removal of a central venous line (CVL) is an unexpected finding to our knowledge not previously described. We recently observed this phenomenon in 3 of 460 consecutive patients in whom a CVL line (usually inserted in the right internal jugular vein) was used during routine cardiac surgical procedures. All 3 patients had an uneventful post-operative period and manifested major neurological symptoms (confusion and right hemiparesis in the first case, paroxystic dispnea, confusion and coma in the second and right barchio-cephalic paralysis in the third) immediately after removal of the CVL. In all patients complete relief of all the neurological symptoms occurred within 24 h and a TC scan of the brain and EEG were negative. The patients were then discharged two or three days after the neurological episode.

The exact pathophysiology of what we observed is not clear. Although a direct correlation between CVL removal and neurological symptoms could not be demonstrated, the fact that in all cases the deficit began immediately after CVL removal makes this correlation extremely plausible. In absence of a definitive explanation, several different aetiologies can be hypothesised.

Direct compression of the carotid artery is unlikely to be the causative factor, only slight external compression was performed using a gauze applied at the point of insertion of the CVL. Moreover preoperative echo-Doppler demonstrated normal carotid arteries in all patients.

Activation of a neuro-vegetative reflex (either sympathetic or parasympathetic), due to the compression on the carotid region, could be a possible explanation. In this case hypotension and bradycardia (from vagal activation) or cerebral vasospasm (from a cathecolaminergic drive) could have led to cerebral hypoperfusion. However, in 2 of our 3 cases the ECG and the systemic pressure were monitored and modifications in the heart rate and blood pressure should have been noted.

Another possibility, at least in the case in whom the neurological deficit was limited to the upper arm, is a stunning of the brachial plexus, due to the central venous line insertion and permanence in place. Nevertheless, this hypothesis does not explain why the neurological deficit appeared only after CVL removal.

A paradox embolism from the CVL to the systemic circulation through a patent foramen ovale could also be hypothesized. However, in all patients preoperative echo-Doppler and intraoperative central venous saturations excluded the possibility of an interatrial communication.

Our last hypothesis regards a possible pulmonary micro-embolisation of platelets (presumably adherent to the catheter). Platelets micro emboli could have caused a reactive arterial and bronchiolar constriction (histamine and serotonin mediated) in the pulmonary bed, with consequent abrupt hypoxemia (explaining the paroxystic dispnea observed in one case) and reduction in oxygen availability at brain level.

In conclusion, the sudden onset of a major neurological deficit in concomitance with CVL removal in an otherwise healthy patient is an undescribed phenomenon with a low but not negligible incidence.

The exact pathophysiology of this phenomenon, as well as the full spectrum of its possible consequences, remain to be determined.