Cerebral blood flow assessed with color Doppler sonography during sedation with dexmedetomidine versus propofol in critically ill patients: a prospective case-crossover study

Dott.ssa GIULIA BONATTI (1), Dott. FABIO TARANTINO (2), Dott. ANGELO INSORSI (2), Dott. ALESSANDRA DE FERRARI (1), Dott. TAREK SENUSSI TESTA (1), Dott. GABRIELE ZUCCO (1), Dott. ANDREA GALANTI (1), Dott. ANDREA MONTEVECCHI (1), Dott. DENISE BATTAGLINI (1), Dott. ALEXANDRE MOLIN (2), Dott. VALERIA PINASCO (2), Dott. IOLE BRUNETTI (2), Dott. CHIARA ROBBA (2), Dott. LORENZO BALL (1), Dott. NICOLÒ PATRONITI (1)(2), Dott. PAOLO PELOSI (1)(2)

 (1) Dipartimento di Scienze Chirurgiche e Diagnostiche Integrate, Università degli Studi di Genova, Largo Rosanna Benzi 10, Genova, Liguria, Italia.
(2) Dipartimento di Emergenza e Accettazione, U.O. Anestesia e Terapia Intensiva, Ospedale Policlinico San Martino, IRCCS per l'oncologia, Largo Rosanna Benzi 10, Genova, Liguria, Italia.

Argomento: Neuroanestesia e neurorianimazione

Introduction: Dexmedetomidine is increasingly used to achieve a 'light sedation', corresponding to a Richmond Agitation-Sedation Scale (RASS) between 0 and -2. However, has been suggested that dexmedetomidine could decrease cerebral blood flow, and its use in patients with impaired cerebral perfusion is not recommended. We aimed to investigate the changes in cerebral blood flow in non-brain-injured patients in which sedation was switched from propofol to dexmedetomidine at comparable RASS scores. We hypothesized that cerebral blood flow decreased with dexmedetomidine compared to propofol.

Methods: Ventilated adult patients without acute brain injury sedated with propofol at RASS 0 to -2 were considered for inclusion if the clinician decided a switch to sedation with dexmedetomidine. Propofol infusion rate was halved when dexmedetomidine infusion was initiated and then stopped after 30m. We measured the mean flow velocity (MFV) in the middle cerebral artery with transcranial color Doppler at the following time-points: before dexmedetomidine infusion (propofol alone), at 3 and 6 h (dexmedetomidine alone). We also recorded the S100- β levels at baseline and after 24 h from switching to dexmedetomidine. Differences were sought with Wilcoxon or Friedman test with Dunn's post-hoc, as appropriate.

Results: Five patients were included in the analysis, aged 62 ± 10 , 60% male. RASS scores were in the target range in all patients and time-points and similar during propofol and dexmedetomidine sedation (p=0.22). MFV was 75 ± 24 cm/s, 62 ± 11 cm/s, 69 ± 21 cm/s, during sedation with propofol, at 3h and 6h, respectively. Compared to propofol sedation, there were no differences at 3h (p>0.99) nor at 6h (p>0.99). The levels of S100- β were 0,19 \pm 0,21 µg/L and 0,13 \pm 0,17µg/L at baseline and 24h (p=0.13).

Conclusions: These preliminary data suggest that dexmedetomidine does not decrease cerebral blood flow in non-brain-injured critically ill patients compared to propofol at the same depth of sedation.