Ambulatory blood pressure is associated with subclinical atherosclerosis in spinal cord injury subjects ☆

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ARTICLE INFO

Article history:
Received 26 September 2011
Accepted 18 October 2011
Available online 5 November 2011

Keywords:
Spinal cord injury
Paraplegia
Tetraplegia
Intima-media thickness
Blood pressure

Subjects with chronic spinal cord injury (SCI) exhibit increased subclinical atherosclerosis and worse left ventricular diastolic function in comparison to able-bodied individuals, independent of traditional cardiovascular risk factors [1-4]. SCI may impair the descending spinal sympathetic tract and promote alterations in blood pressure (BP) profile, such as loss of nocturnal dipping due to orthostatic hypotension, and labile hypertension secondary to uncontrolled sympathetic output as a consequence of autonomic dysreflexia [5]. These alterations, in turn, could potentially explain the increased cardiovascular risk attributed to chronic SCI [5,6]. This study evaluated the relationship between carotid and echocardiographic features and Ambulatory BP monitoring (ABPM) in SCI patients.

Thirty-two nondiabetic, nonhypertensive, nonsmoker, normotensive and normolipemic men attended at a university hospital outpatient clinic with at least 1 year of SCI were evaluated. SCI level ranged from C4 to T12 and only individuals without any preserved motor function below the injury level were included. Fasting serum glucose, lipids and C-reactive protein were measured using standard laboratory techniques. Office blood pressure was measured in the sitting position using validated digital oscillometric device (Omron HEM-705CP, Omron Corp.). ABPM was carried out using a Spacelabs 90207 device. Nocturnal dipping was defined as a reduction in the average systolic and diastolic BP at night greater than 10% compared to daytime values. Carotid ultrasonography and echocardiography studies were performed on each subject in the sitting position with a Vivid 3 Pro apparatus as previously described [2,3]. The study was approved by the Ethics Committee of the State University of Campinas and informed consent was obtained from all participants. The authors certify that they comply with the Principles of Ethical Publishing in the International Journal of Cardiology [7]. All values were expressed as mean ± standard error and median (interquartile range). Univariate and linear regression analyses were used to evaluate the association between cardiovascular parameters and studied variables. A p-value <0.05 was considered significant.

The features of enrolled subjects are shown in Table 1. No subject exhibited plaques at carotid ultrasound or presented complaints of autonomic dysreflexia or BP peaks at ABPM. ABPM values correlated with carotid IMT but not with cardiac parameters. In this regard, 24-h diastolic BP was the measurement showing major correlation with IMT ($r = 0.54$; $p = 0.002$) (Fig. 1). Conversely, office BP, nocturnal dipping status, clinical and laboratory variables exhibited no significant relationship with carotid or cardiac measurements. At last, linear regression analysis revealed that 24-h diastolic BP was the only variable independently associated with carotid IMT ($\beta = 0.417 \pm 0.193$; $p = 0.018$) after adjustment for injury level (tetraplegia or paraplegia) and nocturnal dipping status.

This report provided novel knowledge regarding the impact of BP profile in cardiovascular alterations following chronic SCI. First, it demonstrated that the loss of nocturnal dipping, although highly prevalent, was not related to carotid or echocardiographic parameters, which differs from data reported in able-bodied subjects [6]. Second, none of enrolled subjects exhibited hemodynamic or clinical features of autonomic dysreflexia, suggesting that such condition does not play a major role in SCI-related cardiovascular alterations. Third, 24 h BP measurements were significantly associated with carotid IMT but not with cardiac parameters in normotensive SCI subjects. This finding indicates that remodeling of SCI arteries is sensitive to hemodynamic load even within normal BP range and supports the notion that ABPM might be more adequate to evaluate the effects of BP on vascular but not in cardiac phenotype in injured individuals. Interestingly, 24-h diastolic BP was the best predictor of carotid IMT, suggesting that SCI-related atherogenesis is more influenced by baseline rather than

Table 1
Features of enrolled subjects.

Variable	$n = 32$
Age, years	31.1 ± 1.3
Time of injury, years	6.5 ± 0.7
Body mass index, kg/m^2	22.8 ± 0.7
Paraplegic, *n* (%)	16(50)
Glucose, mg/dL	80.0 ± 2.4
LDL-cholesterol, mg/dL	99.7 ± 6.1
HDL-cholesterol, mg/dL	39.3 ± 1.4
Triglycerides, mg/dL	110.4 ± 9.7
C-reactive protein, ng/mL	6.6(9.7)
Office blood pressure, mm Hg	103.5 ± 2.9/65.9 ± 1.9
24 h blood pressure, mm Hg	108.6 ± 1.3/64.0 ± 1.2
Daytime ambulatory blood pressure, mm Hg	108.8 ± 1.6/65.2 ± 1.4
Night-time ambulatory blood pressure, mm Hg	106.5 ± 1.6/60.4 ± 1.5
Nocturnal dipping, *n* (%)	12(38)
Left ventricular end-diastolic diameter, mm	45.3 ± 0.7
Interventricular septum, mm	8.5 ± 0.2
Posterior wall thickness, mm	8.1 ± 0.1
Left ventricular mass index, g/m^2	81.5 ± 2.9
Ejection fraction, %	68.0 ± 0.9
E/A Ratio	1.43 ± 0.05
Sm, cm/s	10.7 ± 0.5
Em, cm/s	9.4 ± 0.5
Am, cm/s	8.7 ± 0.6
Carotid intima-media thickness, mm	0.70 ± 0.02

LDL—low-density-lipoprotein; HDL—high-density-lipoprotein.

☆ Funding: This work was supported by grants from Fundação de Amparo à Pesquisa do Estado de São Paulo (2010/16252-0) and Conselho Nacional de Desenvolvimento Científico e Tecnológico (474966/2010-0 and 303539/2010-0).

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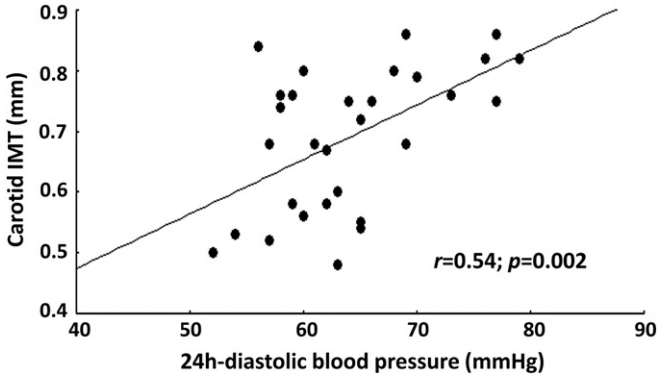

Fig. 1. Univariate correlation between carotid intima-media thickness (IMT) and 24 h-diastolic blood pressure.

cyclic peak vascular stretch. Nevertheless, further studies are necessary to address this issue.

A limitation of the study was the relatively small sample size. For this reason, these findings might not be generalized to other SCI populations. However, it was noteworthy that our sample was very homogeneous, since we excluded individuals with confounding risk factors commonly seen after chronic SCI [8], such as obesity, dyslipidemias, diabetes mellitus and hypertension. Conversely, our sample presented a thicker IMT compared to that reported by Wang et al. [1]. The reason for this discrepancy is not clear, but it is possible that the fact that we only included subjects without any preserved motor function below the injury level, which is a feature consistently associated with increased prevalence of cardiovascular disease [4,9], might have played a role in this regard.

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0167-5273/$ – see front matter
doi:10.1016/j.ijcard.2011.10.055

Novel markers of cerebral embolism in the course of infective endocarditis

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ARTICLE INFO

Article history:
Received 26 September 2011
Accepted 18 October 2011
Available online 5 November 2011

Keywords:
Protein S-100B
Neuronal specific enolase
Infective ednocarditis
Stroke
Silent stroke

Infective endocarditis (IE) is associated with a high mortality rate (16–25%) [1,2], and high prevalence of embolic complications 13–49% [3], 65% of which affect the central nervous system (CNS) [4] with the mortality rate around 21–83% [5]. CNS stroke diagnosis is based on clinical picture and neuroimaging with MRI or CT. The markers associated with CNS stroke which are measured most commonly are S-100B protein and neuron-specific enolase (NSE) [6,7], which are released from neural tissue when it is damaged. Most cerebral complications are symptomatic CNS strokes. Recently there have been clinically silent CNS strokes described, which could be diagnosed only with MRI/CT. The influence of clinically silent CNS embolism on the prognosis is not reliably defined. Neither has the usefulness of biomarkers been evaluated in this diagnostics.

For this purpose, we have performed the study on IE patients. In our first manuscript [8] have described epidemiological data on overt and silent CNS embolism, presently we assess the usefulness of S-100B protein and NSE as biological markers of CNS lesions in the diagnosis of CNS embolism in the course of IE.

The study group consists of 65 patients with IE hospitalized at the Institute of Cardiology, Warsaw, Poland. The inclusion and exclusion criteria, examinations and tests, neurological examination and neuroimaging with MRI or CT were described in our previous manuscript [8]. Each patient gave their informed consent to participate in the study.

As in the previous paper [8], based on the neurological examination and MRI/CT picture we divided study group as described below:

- Group I — with clinically overt CNS embolism (cOCNSe)
- Group II — with clinically silent CNS embolism (cSCNSe)
- Group III — with no CNS embolism (NCNSe)

The groups are characterized by the criteria given in the previous article [8].

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