rhabdomyolysis associated with the drug have been reported to date: in two cases the patients were taking 10 mg of rosuvastatin per day, and in five the dose was 40 mg per day; in one case the dose was not specified.

Minor muscle complaints without elevated creatinine kinase levels may not necessitate discontinuation of the drug. However, patients, particularly those with risk factors for statin-induced myopathy, should be warned of the potential for rhabdomyolysis and told to report immediately any muscle pain, muscle weakness or cramps, or dark urine. If rhabdomyolysis is suspected, the drug should be stopped immediately, and appropriate medical management should be instituted as well as a work-up of predisposing risk factors. In our patient as his risk for future coronary events was very high it was essential to exhibit combination therapy to control his dyslipidemia. As he had evidence of rhabdomyolysis early on exhibiting combination treatment with Rosuvastatin and Fenofibrate, the therapy had to be stopped immediately.

Therefore, in high risk patients with atherosclerosis, on combination therapy with fibrates and statins it is essential to instruct patients regarding the possible adverse effects and need for regular follow up. Myopathy is reversible on withdrawal of the combination therapy.

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REFERENCES

Infectious Causes of Peripheral Autonomic Neuropathy

Sir,

I read with interest an article “approach to a case of autonomic peripheral neuropathy” by Chowdhury and Patel. The authors have given a long list of various causes of autonomic peripheral neuropathy. Of course the main theme of the article was an approach towards this entity, but I would like to add two important infections as the cause of autonomic peripheral neuropathy, that is P. falciparum malaria and brucellosis which are quite pertinent in tropics. I myself have seen autonomic involvement in patients of falciparum malaria.

As the authors have said in the last “treatment aims to treat specific cause of the neuropathy” our patient improved with specific therapy i.e. anti-malarial drugs.

The aim of my this correspondence is to make aware the primary care physicians in general and neurologists in particular that as India is witnessing outbreaks of falciparum malaria at one or the other part from time to time, we should look for P. falciparum malaria in a patient with fever and autonomic involvement in a particular setting.

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Received : 31.10.2006; Accepted : 2.12.2006

REFERENCES

Reply from Author
Sir,

We appreciate the valuable addition to the long list of causes of autonomic peripheral neuropathy mentioned in our article. Falciparum malaria and neuro-brucellosis can occasionally cause autonomic manifestations associated with radiculoneuropathy. However, our main emphasis in the article was to highlight conditions in which the autonomic neuropathy is the major or sole manifestation of the disease. Treating the underlying causes of autonomic neuropathy including the infectious causes can be quite rewarding.

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Effect of Valsalva Maneuver on QT Interval, QT Dispersion and Rate Pressure Product

Sir,

Autonomic tone modulates the ventricular recovery time dispersion indices (QTc and QTd). Valsalva maneuver (VM) modifies the autonomic tone and thus employed is a test of cardiac function. However, effect
of VM on QT interval has not got proper attention. So the present study was conducted on 25 young healthy male subjects (to avoid gender differences) in the age group of 20-40 years (mean age 25.62 ± 10.30) with mean Quetlet index 24.2 ± 0.34 to investigate the effect of VM on QTc interval (QT interval corrected for heart rate by using Bazzette’s formula), QTd (difference between maximum and minimum QT values) calculated from Lend II of electrocardiogram (ECG) and rate pressure product (RPP = heart rate (HR) systolic BP/100 and is an index of myocardial oxygen consumption).

Subjects had normal haemoglobin and blood pressure (BP). Presence of congenital, valvular or organic heart diseases were excluded. The procedure for recording of VM (a deeper than normal inspiration was followed by a forceful exhalation into mouthpiece connected to sphygmanometer and maintained the pressure to 40 mmHg for about 15 seconds) was explained to the subjects. Parameters were analysed in:
1. Control period before initiation of strain.
2. Strain period (phase II).
3. Post release period (phase IV).

Statistical analysis was done by paired ‘t’ test.

Without any change in QTc and RPP in phase IV of VM compared to baseline level. There were significant changes in R-R interval, HR and BP in both phases of VM (Table 1).

In VM, sympathetic and parasympathetic activity increase in phase II and phase IV respectively. Prolongation of QTc interval produced by valsalva strain is comparable to that produced by exercise and isoproterenal infusion. Both are associated with increased adrenergic activity. Catastrophic rise in blood pressure is known to be a precipitating factor for vascular events particularly in the morning hours. This time is thought to be associated with adrenergic system activation. Higher incidence of ST segment elevation myocardial infarction (STEMI) is also reported in the morning hours.

Moreover, significant rise in blood pressure on straining in squatting (a common posture adopted by most Indians during defecation) may mimic with the rise in blood pressure in VM. Although not reported in the literature, an increase in QTc and QTd might contribute to an increased incidence of vascular events and sudden deaths in the morning hours, STEMI, squatting posture particularly in uncontrolled hypertension and aged person, as RPP is also elevated in sympathetic phase of VM in current study. Squatting posture during defecation and primary angioplasty in STEMI in morning hours should be avoided, although additional studies are needed to confirm the findings.

### Table 1: Parameters recorded during valsalva maneuver (VM) (Mean ± SD)

<table>
<thead>
<tr>
<th>Sr. No.</th>
<th>Parameters</th>
<th>Before VM</th>
<th>VM (Phase II)</th>
<th>VM (Phase IV)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Heart Rate (Beats/Min)</td>
<td>74.62 ± 11.03</td>
<td>89.84 ± 17.50**</td>
<td>63.22 ± 9.7***</td>
</tr>
<tr>
<td>2.</td>
<td>Blood Pressure (mm Hg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Systolic</td>
<td>116.84 ± 10.02</td>
<td>100.21 ± 9.46**</td>
<td>138.90 ± 9.27***</td>
</tr>
<tr>
<td></td>
<td>- Diastolic</td>
<td>76.83 ± 10.47</td>
<td>68.72 ± 8.52**</td>
<td>91.06 ± 10.57***</td>
</tr>
<tr>
<td>3.</td>
<td>R-R interval (msec)</td>
<td>827.20 ± 114.40</td>
<td>680.0 ± 126.88**</td>
<td>980.0 ± 35.66**</td>
</tr>
<tr>
<td>4.</td>
<td>QTc (msec)</td>
<td>400.0 ± 150.6</td>
<td>440 ± 78.7**</td>
<td>380.0 ± 59.5 NS</td>
</tr>
<tr>
<td>5.</td>
<td>QTd (msec)</td>
<td>28.0 ± 3.9</td>
<td>30.0 ± 2.72**</td>
<td>21.4 ± 1.7**</td>
</tr>
<tr>
<td>6.</td>
<td>RPP</td>
<td>88.18 ± 6.8</td>
<td>91.02 ± 11.3**</td>
<td>87.81 ± 9.6NS</td>
</tr>
</tbody>
</table>

Comparison between - before and after VM (Phase II): * p<0.01, ** p<0.001, NS - Non significant; Comparison between before and after VM (phase IV): * p< 0.01, ** p<0.001, NS - Non significant; RPP - Rate pressure product

REFERENCES


Utility of K39 Strip Test in Visceral Leishmaniasis (VL) and HIV Co-infected Patients: An Early Report from Eastern India

Sir,

Since 1986 visceral leishmaniasis (VL) has emerged as an AIDS associated opportunistic infection where VL is thought to represent reactivation of latent infection or the failure to express cellular immunity to a new infection. However, very few co-infected cases have been reported from India. The typical clinical presentation of VL in this situation may be masked by the presence of other opportunistic infections in HIV/AIDS. There is also widespread dissemination of *L. donovani* in multiple viscera causing multitude of different symptoms.

The diagnosis is difficult as demonstration of parasites is accomplished by splenic or bone-marrow aspirations. Recently a promising ready to use strip test has been developed using leishmanial antigen K39 - a...