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Van die Redaksie/Editorial

Reisigers-diarree

Die nabyheid van Mexiko aan die VSA, die feit dat ongeveer 3 miljoen Amerikaners Mexiko jaarliks besoek, en die feit dat enigiets van 25% tot 50% van hulle as gevolg van hulle reis aan diarree ly, het Mexiko 'n sentrum vir die studie van daardie universele teistering, reisigers-diarree, gemaak. Soos Gorbach[1] dit pittig stel, 'travel expands the mind and loosens the bowels'. Ons weet nou dat die hoofoorsaak vir reisigers-diarree 'n enterotoksigeniese *Escherichia coli* is wat 'n warmte-labiele of -stabiele toksien veroorsaak. Hierdie organisme is in 40 - 70% van gevalle geïsoleer, afhangende van die plek en die tegniek wat gebruik is. Die volgende mees algemene organisme wat geïsoleer word, is *Shigella,* met 'n voorkomssyfer van 5 - 20%. In baie gevalle word daar egter geen organisme geïsoleer nie, wat ook al die tegniek.

Baie reisigers het al probeer om hulleself teen hierdie siekte, wat 'n toeris in die bed kan hou of ten minste sy aktiwiteite in 'n groot mate kan beperk, te beskerm. 'n Hele aantal antibiotika soos ko-trimoksasool en die tetrasikliene is al vir hierdie doel aangewend. Dat hulle redelik effektief is, is in verskeie proefneminge aangetoon, maar daar is besware teen die wydverspreide gebruik van hierdie middels. Die mees voor die hand liggende een is die ontwikkeling van weerstandige koliforme, en in sekere dele van die wêreld is daar alreeds 'n hoë weerstandsvoorkoms wat die gebruik van hierdie middels bevraagteken. Die tweede beswaar is die newe-effekte wat hierdie middels mag veroorsaak — bismut-subsalisaat in groot dosisse 4 keer per dag is al as 'n alternatief voorgestel. Ongelukkig is die voorgeskrewe dosis baie groot en ongemaklik om te neem asook om saam te dra.

Die alternatief skyn om vroeë behandeling van enige diarree wat ontwikkel te wees, en die mees onlangse verslag deur Du Pont *et al.*[2] stel voor dat ko-trimoksasool (Bactrim, Septran, ens.) of selfs slegs sy komponent trimetoprim vir hierdie doel gepas is. Hulle het 'n bevolking van volwasse manlike en vroulike Amerikaanse studente in Mexiko bestudeer, en enige student wat binne 48 uur na die aanvang van die siekte aangemeld het, in 'n kliniese proefneming opgeneem. Dié studente het òf ko-trimoksasool in 'n dosis van 2 tablette 2 keer per dag vir 5 dae òf 200 mg trimetoprim òf 'n plasebo ontvang. Die proefneming het duidelik die voordelige effek van die middels bo die plasebo aangetoon. Die behandeling het in slegs 5% van die pasiënte wat ko-trimoksasool ontvang het, en in 8% van dié wat trimetoprim ontvang het, misluk, in vergelyking met 49% in diegene wat 'n plasebo ontvang het. Die middel was net so effektief hetsy 'n stoelgangmonster *E. coli, Shigella* of geen enteropatogeen getoon het. Aangesien die kliniese reaksie binne 48 uur waarneembaar was, meen die outeurs dat 3 dae van behandeling voldoende is en voeg hulle by dat, indien die middel self toegedien word en dit misluk, mediese en verdere ondersoek noodsaaklik is aangesien die diarree aan *Campylobacter* of 'n intestinale parasiet te wyte mag wees.

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Phaeochromocytoma — diagnosis and localization

Phaeochromocytomas are tumours of neuro-ectodermal origin arising from the chromaffin cells of the sympatho-adrenal system. The clinical expression of the tumour is very variable, and it has therefore earned the title of 'the great mimic'.[1] Current data suggest that 0,1 - 1% of patients with persistent diastolic hypertension harbour a phaeochromocytoma.[2] Of greater concern is the fact that over 90% of these tumours are surgically remediable,[3] and one should therefore not miss the diagnosis of an otherwise potentially lethal condition. It is significant that 54 of 40 078 autopsies performed at the Mayo Clinic during the 50-year period 1928 - 1977 revealed phaeochromocytoma, representing an autopsy incidence of 0,13%.[4] The finding that in only 24% of cases had the diagnosis of phaeochromocytoma been made during life was not surprising. Retrospective analysis revealed that 91% of the patients with phaeochromocytoma (diagnosed and undiagnosed) had had symptoms attributable to excess circulating catecholamines, while 61% had been shown to be hypertensive on at least three separate

occasions; in two-thirds of these patients the hypertension was persistent and in one-third paroxysmal. Phaeochromocytoma should therefore be suspected in any patient, with or without hypertension, with any of the kaleidoscope of symptoms suggestive of the lesion.[2]

The normal adrenal medulla secretes both adrenaline and noradrenaline, but predominantly adrenaline, whereas phaeochromocytomas tend to secrete larger amounts of noradrenaline. Both adrenaline and noradrenaline are metabolized to their methylated derivatives and to 4-hydroxy-3-methoxymandelic acid, more commonly known as vanillylmandelic acid (VMA). Definitive diagnosis rests on biochemical confirmation of the presence of excess catecholamines or their metabolites. The simplest screening test for a phaeochromocytoma is estimation of urinary total metanephrines,[2,5] which is said to offer close to 95% sensitivity and 97% specificity. The total metanephrine value is less subject to false-positive and false-negative results and drug interference than the urinary VMA value.[2] If equivocal results are obtained, estimation of total metanephrines should be repeated on three further 24-hour urine collections or by measuring the urinary VMA value. Unfortunately urine collections over 24 hours are often incomplete, but this may be partly corrected by expressing the results in terms of the urinary creatinine value.

With the advent of specific and sensitive radio-enzymatic and high-performance liquid chromatographic methods for determination of the individual catecholamines, estimation of catecholamines in small volumes of blood taken by selective venous sampling has made the biochemical localization of phaeochromocytoma possible.[5-7] The supine plasma noradrenaline level is not a reliable screening method for the detection of phaeochromocytoma, as it overlaps considerably with levels in the normal population and there is paroxysmal release of noradrenaline from the tumour.[2,5] The peripheral plasma noradrenaline level may be increased by anxiety and by various illnesses; the results may therefore be incorrectly interpreted unless the patient's clinical condition is known.[2,7]

Once a firm clinical and biochemical diagnosis of phaeochromocytoma has been made, pre-operative localization of the tumour is mandatory. Because phaeochromocytomas obey the well-known 'rule of ten' — 10% extra-adrenal (i.e. 90% adrenal), 10% multiple and 10% malignant — and because the vast majority (95%) are intra-abdominal, non-invasive techniques such as computed tomography (CT) are now preferred initially for localization of phaeochromocytomas. Since most of these tumours measure more than 2 cm in diameter, CT will localize over 90%.[8] In our own experience CT has successfully localized all of 6 intra-adrenal phaeochromocytomas, and although an intravesical phaeochromocytoma was missed initially it was clearly evident on repeat CT. When CT fails, selective venous sampling for determination of the plasma catecholamine levels has proved useful; this is less invasive than selective arteriography.[6] What appears promising is the report by the Ann Arbor Group of scintigraphic localization by means of ^{131}I-meta-iodobenzyl-guanidine (MIBG).[9] Predictably, with the commercial availability of ^{131}I-MIBG or related compounds, non-invasive methods of radiological examination (CT) and nuclear medicine (^{131}I-MIBG) will be the mainstay of successful localization.

Because of the protean presentation of phaeochromocytoma and the high incidence of fatal complications in an otherwise curable disease, the tumour should be suspected whenever a patient exhibits any symptom or sign vaguely suggestive of excess circulating catecholamines. It behoves us as clinicians to screen for this disorder in these patients.

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