Hypertension and reduced renal function: a rebuttal.

LETTERS TO THE EDITOR

More on hypertension and reduced renal function

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First of all, compliments for the new and wider "dress and mission" of the journal of our society. As it is destined for a double audience, it is no surprise that each paper will naturally be seen from two different points of view. Even though many of the Italian emergency physicians (EP), at the beginning of Italian speciality courses in emergency medicine (EM), started out in Internal Medicine, as they have been working in EM they have changed quickly their clinical style because of unstable clinical conditions of some patients, and more pressing rhythms that have to be faced. One must develop a quicker, simpler and more pragmatic way of thinking and acting: does the patient have a life-threatening disease or not? Does the patient need hospitalisation or is it safe for a primary care physician (PCP) to manage the problem on an outpatient basis? The introduction of cost/benefit ratios in medicine is creating another pressure: not to admit the patient.

As internists we read with great interest the case report in the first issue [1], but as physicians working in EM, we immediately transferred its contents to our practice. How many patients with increased blood pressure (BP) can we "appropriately" hospitalise? Surely, the ones with a complicated hypertensive crisis, like the patient in the case report. What about the others? Is it enough to reduce the BP at presentation, re-educate them on diet and treatment compliance, and then send them to the PCP with a recommendation to keep a diary of their BP for 10–14 days? Maybe diagnosing secondary hypertension is not the responsibility of the EP, especially during the first presentation and in asymptomatic cases, but can the EP prevent, in some way, deterioration requiring further visits to the emergency department (ED) for the same level of hypertension as well as more complicated deteriorations?

The patient in this case report had already been evaluated for mild nocturnal dyspnoea and palpitations associated with an increased BP, and treated with an addition of diuretics. Blood tests and ultrasound scan 1 month before could have prevented the pulmonary oedema and the need for hospitalisation, even though it would have been more expensive than a simple introduction of diuretics.

It is obvious we cannot screen all hypertensive and dyslipidaemic patients coming to the emergency room with duplex ultrasonographic scanning, as this is not a readily available resource, nor can we undertake other examinations for other secondary kinds of hypertension, nor can we hospitalise all of them. It is true that we would not miss any diagnoses of secondary hypertension if we were able to do these things.

What, then, would be the best alternative to achieve an efficacious, safe and cost-effective practice for an EP facing an asymptomatic hypertensive patient who exhibits strong compliance with diet and treatment?

Should we test the serum creatinine and potassium levels, and hospitalise the patients with altered values? Should we hospitalise patients whose clinical presentation suggests secondary hypertension? Most hypertensive patients take diuretics, and will routinely have slightly elevated creatinine and potassium. Moreover, in many rural and peripheral small EDs, it is impossible to obtain blood tests at night!

Should we refer the patients to the PCP for the blood tests, including creatinine clearance and close follow-up? The reality is that patients are sent to us by the PCP, who often encounters obstacles in having early appointments for diagnostic and imaging procedures.

Should we refer the patients to EM outpatient clinics?

Should we refer the patients to Internal Medicine or Cardiology for same-day hospital evaluations? Formerly we used to hospitalise asymptomatic hypertensive patients with a strong possibility of secondary hypertension (a BP that remains elevated in spite of different treatments in the ED).

Should we have the patient return to the ED for brief follow-up over a short time period, especially if they have altered blood tests where available, and refer all the others to the PCP? At present, we are just starting a complex assistance package project, sustained by our regional government. It would be interesting to know about similar experiences regarding this and other diagnoses.

Reference

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We agree with Drs. La Regina and del Prato that problems in medicine can be approached from a variety of perspectives, although some basic directions should be shared by physicians working in different fields. There is no doubt that arterial hypertension is today a diagnosis that rarely leads to hospitalisation. There are however occasions characterised by a high risk for the patient, when this will be necessary, as in cases of pulmonary oedema or transient ischaemic cerebral attack. Concerning the patients without life-threatening hypertensive crises, the current Guidelines for the management of high blood pressure provide clear indications, suggesting the need for repeated visits to confirm the presence and the severity of an elevation of blood pressure, and to assess the global risk profile of the patient and the presence of target organ damage. In most cases there is no need to hasten the start of antihypertensive treatment until the diagnostic process has been completed. The time interval before starting treatment is obviously a function of the patient's risk level. An appropriate diagnostic work-up should also lead to identification of cases with secondary hypertension. Primary Care Physicians (PCP) have the duty to start management of patients with hypertension, in cooperation with specialised centres that provide all the necessary support for definition of the patient's risk profile and organ damage. This approach, accompanied by a good relationship between the patient and the physician, will also reduce patients' utilisation of the emergency service for false hypertensive crisis, often due to anxiety and sympathetic hyper-reactivity.

The diagnostic work-up of the patient should not be carried out in the Emergency Department, but should be part of the routine interaction between PCP and specialists.

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Bilateral posterior shoulder dislocations following seizure

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Anterior shoulder dislocations are a common complaint in emergency medicine. Most emergency physicians can recognise an anterior shoulder dislocation based on physical examination alone, and the diagnosis is quickly confirmed with appropriate radiographs. Reduction can be accomplished with any number of techniques; a standard textbook of emergency medicine procedures lists nine ways, each with variations, to reduce anterior shoulder dislocations [1].

Posterior shoulder dislocations are rare, and account for less than 5% of all shoulder dislocations [1]. Although classical physical examination findings are described, they are frequently not appreciated by the initial examiner. The anteroposterior (AP) radiograph of the shoulder may appear normal, which contributes to a high misdiagnosis rate.

We report a case of bilateral posterior shoulder dislocations after seizure in a young man with no orthopaedic history.

A 25-year-old man with type I diabetes was at a local sporting event when he became hypoglycaemic and suffered a tonic-clonic seizure. Nearby fans caught him before he fell and helped him to the ground. He was taken to the stadium first aid station, where a fingerstick blood glucose level was too low to register on the glucometer. He received IV glucose and was then transported to our Emergency Department (ED) for further evaluation.

On arrival at the ED, he was post-ictal and unable to offer either a complaint or a history of present illness. On physical examination, his vital signs were: pulse, 77 beats/min; blood pressure, 144/87 mmHg; respirations, 21/min; temperature, 35.9°C. General assessment revealed a well developed, muscular young man who was confused but otherwise in no significant distress. Initial examination of the heart, lungs and abdomen were unremarkable. A fingerstick glucose obtained on arrival in the ED was 66 mg/dl (4 mmol/l).

The patient's mental status improved as his post-ictal state resolved. He reported a history of type I diabetes for approximately 1 year with a prior history of hypoglycaemia from poor compliance with his insulin regimen. He denied any other medical or surgical history. He complained that his shoulders hurt and that he could not move them.

More detailed examination of the extremities revealed shoulders that were symmetric but deformed, with empty glenoid fossae bilaterally and loss of the normal superior contour on each side. The humeral heads were palpable posteriorly on each side. The shoulders were adducted bilaterally. The patient was unable to lift or externally rotate either arm. Neurovascular status of the upper extremities was intact bilaterally.

AP radiographs of each shoulder (Fig. 1) appeared normal. Axillary radiographs of the shoulders (Fig. 2) revealed bilateral posterior dislocations without fractures. Procedural