Clinical judgment and diagnostic procedures – role reversion?

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Abstract

In a high tech era, like ours, doctors may tend to rely too much on diagnostic procedures, not using adequately our most precious tool, the clinical judgment. Such an approach can be hazardous, as illustrated in the presented case.

Key words: Diagnostic procedures, clinical judgment.

INTRODUCTION

Not many years ago, Medical practice was focused on the detailed collection of the patient's clinical history and the precise execution of the physical examination. The data obtained were then *analyzed,* in order to form diagnostic hypotheses and establish a diagnostic and therapeutic strategy, Nowadays, faced with the enormous range of complementary exams available, there is a tendency not to *waste* so much time obtaining the anamnetic data and formulating a well-founded clinical reasoning, resorting instead to complementary exams right from the start. This strategy often results in undue costs, and can even be harmful for the patient.

CLINICAL CASE

Woman aged 50 years, Caucasian, married, a business owner. Transferred from another hospital with coma of unclarified etiology.

The patient was apparently well until the morning of the day of admission, when she was found in a coma in her home, with evidence of having vomited food. She was known to have type 2 diabetes mellitus, osteoarthroses and depression, and was medicated with glimepiride, nabumetone, venlafaxine, trazodone and alprazolam.

On arrival at the Hospital, she was in a deep coma. The only factors highlighted from the remainder of the physical examination are obesity, noisy but effective breathing, and miotic pupils. Subsequently, pulmonary auscultation revealed rhonchi and bilateral rales.

Of the analyses carried out, the following are highlighted: leukocytosis of 12.100/mm^3, neutrophilia of 85%, remainder of hemogram normal, glycemia 226mg/dL, renal and hepatic functions normal, cardiac enzymes normal, reactive protein C negative, benzodiazepine (BZD) levels normal and triclicic antidepressants (TCAs) positive, and barbituric, amphetamine, cannabinoid, opiates, cocaine and methadone levels negative.

The electrocardiogram was normal.

Chest teleradiography showed heterogenous hypotransparency throughout the right lung field (more in the lower 2/3) and left lung base.

An intravenous formula of flumazenil was administered, after which the patient awakened from the coma. This was followed by another deterioration of sensation.

The hypothesis of brainstem lesion was considered, and cranioencephalic axial computerized tomography (CE CT) was performed. This showed "very slight loss of definition of the sulci and decrease of the lateral ventricles, probably related to age group, which may, however, suggest cerebral edema?" At this point, intravenous dexamethasone and mannitol were initiated. Given the hypotheses of drug intoxication and aspiration pneumonia, the patient also received nasogastric intubation and activated charcoal and amoxicillin-clavulanate 2.2 grams were initiated.

During the second day of hospitalization, the

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Received for publication on the 16th March 2007
Accepted for publication on the 15th May 2010

patient was transferred to our hospital with suspected diagnoses of "brain stem lesion/anoxic cerebral edema?" Aspiration of vomit in the home? Drug medication?" for "clarification of the diagnosis". In the telephone contact, it was suggested that craneoencephalic magnetic resonance (CE (NMR) be carried out.

On entry to our Emergency Service, she had a score of 3 on the Glasgow coma scale. The pupils were miotic and reactive to light, and the remaining brain stem reflexes were present; muscle tone was maintained and symmetrical, and the cutaneous plantar reflexes were apparent on flexion. The patient was hemodynamically stable. She had a clamped nasogastric probe, and vomited activated charcoal on entry to our Service (the bedhead was positioned at 0º and the patient arrived with a dental prosthesis in place). Noisy, spontaneous breathing with wheezing, pulse oxymetries were 94% (supplementary oxygen at 60%) and lung auscultation showed subcrepitant rails at all times, in both lung fields.

Intravenous flumazenil was administered (1+1 formula), after which the patient became alert and collaborative. When questioned about what had happened, she reported having been involved in an interpersonal conflict, followed by voluntary ingestion of alprazolam and trazodone pills, as well as other drugs that she had in the home. She had subsequently provoked vomiting.

Faced with a hypothesis of aspiration of activated charcoal, fibrobronchoscopy was carried out, aspirating only mucopurulent secretions.

Urinary doses of BZDs (above 1000UI) and TCAs (unmeasureable) were repeated. It was decided to maintain the antibiotherapy instituted, and to administer intravenous perfusion of flumazenil, which was necessary for around 24 hours. Subsequently, and after psychiatric evaluation, the patient was transferred to the Hospital of origin.

DISCUSSION

The case is reported of a middle aged woman in a state of coma. Diabetic and suffering from depression, she was medicated with sulphonylurea, benzodiazepines and antidepressants. In the observation, deep coma, miotic pupils and noisy breathing were observed, although with normal lung auscultation. Glycemia was slightly elevated. Flumazenil was administered, with regression of the coma. BZD and TCA levels were positive.

These data are sufficient to admit a diagnosis of benzodiazepine overdose. Although the ingestion of TCA was also possible, the severity of the clinical state did not appear to be related to this drug, since the pupils were miotic and the coma regressed with the administration of flumazenil. A correct diagnosis, at this juncture, may have avoided the aspiration pneumonia (assuming that this occurred in the hospital environment – lung auscultation normal on admission), through the continued administration of flumazenil or orotracheal intubation.

The determination of amphetamine, cannabinoid, opioid, cocaine and methadone levels was not justified.

Neither was the CE CT justified, given that the coma regressed with flumazenil. This drug blocks the effects of the BZD on the central nervous system by competitive action in the receptors,[1,2,3] and has no action in cases of organic lesions, particularly cerebral vascular accidents (CVA). In fact, the only documented role of flumazenil in CVA is that it determines the reversibility of neurological lesions, through proton emission tomography (PET) with flumazenil (areas with less flumazenil uptake correspond to irreversible lesion).[4,5] The CE CT did not present any significant alterations, but was reported "in defensive form". As a result, the possibility of a brainstem lesion with anoxic cerebral edema was considered, with unnecessary administration of dexamethasone and mannitol. Given that there was no diagnostic certainty, the decision was made to transfer the patient to a Central Hospital, and CE NMI was suggested. This decision may have resulted in foreign body aspiration pneumonia (dental prosthesis) or of activated charcoal and chemical pneumonitis, according to the way in which the patient was transported.

CONCLUSIONS

Nowadays, there is easy access to all types of complementary exams, and there may be a tendency to *relax* the clinical reasoning and rely too heavily on the exams for obtaining a diagnosis. Furthermore, it has the added compensation of additional security, in the event of a complaint of clinical malpractice. This case illustrates the importance of correctly weighing the patient against the danger of relying too much on complementary exams. In summary, it is still worth *wasting time* thinking about the patients, and the

complementary diagnostic exams should continue to be seen as just that - complementary. ■

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