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La Voix

Clinical Medicine in the Era of Advanced Technology: Is It Relevant ?

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Advances in medical technology have completely transformed the way health care is delivered in the present day. The last two to three decades have seen rapid strides in technology in all fields of medicine including imaging (CT,MRI ,functional imaging), laboratory services (advanced serological tests, IHC, molecular biology), endoscopic techniques (advanced ERCP, EUS techniques, diagnostic and therapeutic), surgical techniques (minimally invasive, robotic), therapeutics (antibiotics, antivirals, biologicals, anti-cancer) and medical information (electronic records, teleconsultation, internet). There is no doubt that technology is indispensable for current medical practice and for best patient outcomes. However, what is alarming is that, instead of serving as a useful adjunct, it is increasingly replacing good clinical medicine and therein lies the rub.

First, what do we mean by clinical medicine? As defined in the Medical Dictionary, it is "the study and practice of medicine in relation to the care of patients; the art of medicine as distinguished from laboratory science." It comprises several steps starting with taking a detailed history followed by a thorough physical examination, which will enable one to come to a diagnosis after excluding other differential diagnoses, so that only relevant investigations need be recommended Of course, all this is based on the assumption that the physician has a good understanding and knowledge of medicine.

The following narratives represent actual case scenarios encountered in my outpatient department, which underscore the ill effects of the lack of application of clinical medicine, leading to delayed or erroneous diagnoses .

Case 1

A 38 year old lady presented with longstanding (12 yrs) symptoms of dyspepsia, anorexia, gradual weight loss and episodic nonbilious vomiting occurring once or twice a month and lasting several days. There was no abdominal pain at any time. and there was no headache or giddiness accompanying the vomiting. Her bowels were variable in frequency and consistency but there was no blood or mucus, and no nocturnal stool. She was also very anxious and had in fact been diagnosed to have irritable bowel syndrome and treated for the same. In the past, she was diagnosed to be hypothyroid 5 yrs ago and was on supplemental therapy and had undergone a cholecystectomy 2 yrs ago for gallstones detected incidentally during evaluation of her GI complaints. 2 months prior to her visit here, she had developed varicella infection, during which time she became "unconscious" and required hospitalization. She was the mother of two children, both full term normal delivery. She had been evaluated at multiple centers and laboratory tests including hemogram, tests of liver and renal function, chest X-ray and ultrasound abdomen were repeatedly normal. An upper GI endoscopy was reported to show a hiatus hernia, for which she was placed on proton pump inhibitors and prokinetics, along with anxiolytics.

When asked about her menstrual history as a routine, she said that she had been amenorrhoeic since her last childbirth 17 years ago. This immediately raised a red flag, and on further probing she admitted to severe postpartum bleeding necessitating blood transfusion, as well as total lactational failure.

Clinical examination was unremarkable except for the obvious evidence of weight loss in the form of a BMI of 19.5, and routine laboratory investigations were essentially normal except for mild anemia. A the history favoured Sheehan's syndrome, LH, FSH and prolactin levels were estimated which were found to be low ,as were T3 and T4 with low normal TSH level. She was referred to the endocrinologist , who asked for an MRI brain at which time she recalled that an MRI was done at the time of her varicella infection. The report was reviewed and found to reveal an empty sella. So there you have it: the diagnosis of Sheehan's syndrome which had been missed for so many years, despite the pointers in history and imaging.

Case 2

A 33-year old male presented to the OPD because he was worried about the diagnosis of fatty liver on ultrasound coupled with abnormal "liver function tests "noted since the last 2 years. He had mild GE reflux symptoms and constipation and occasional mild right loin pain but was otherwise asymptomatic. There was no history of jaundice, surgery, blood transfusion, alcohol abuse, and there was no family history of liver disease. A perusal of his previous reports showed that he had fatty liver and significant alkaline phosphatase elevation on numerous occasions, including his recent evaluation at a hepatology unit elsewhere, where fatty liver was again confirmed on ultrasound, and LFT showed an elevated alkaline phosphatase of 540 IU /ml. The bilirubin /transaminases and GGTP were normal and fibroscan showed S2 steatosis and no fibrosis. An MRCP was then recommended which was normal and the patient was reassured and advised liver supportives, dietary restriction and exercise. However, the patient was anxious and wanted a second opinion.

If you look for the approach to elevated ALP, most algorithms require you to first eliminate nonhepatic causes, which is done by heat fractionation of alkaline phosphatase or more simply, by estimating GGTP, which if elevated suggests a hepatic origin. If GGTP is normal ,the elevated alkaline phosphates is possibly of nonhepatic origin, the most common being from bone (Paget's disease osteomalacia, vitamin D deficiency or hyperparathyroidism).

This patient had an isolated elevation of alkaline phosphatase, suggesting a nonhepatic origin. On further evaluation, he was found to have hypercalcemia with elevated PTH ,and ultrasound and radionuclide evidence of a functioning left parathyroid adenoma. Again a simple diagnosis which was missed all the same for 2 years, because basic algorithms were overlooked and an unnecessary and expensive technological intervention carried out.

Case 3

A 54-year old male consulted a physician for complaints of pedal edema and abdominal distension since 6 months with anorexia, weight loss of 6 kg, lethargy, fatigueability, mild shortness of breath He gave a history of significant alcohol intake in the past but was abstinent since 11 year. He had been diagnosed elsewhere to have cirrhosis liver and was on dietary salt and fluid restriction and diuretics ,and had been advised to undergo orthotopic liver transplantation Clinical examination showed significant pedal edema and gross ascites. The investigations advised by the physician revealed normal blood counts, an elevated, normal renal function tests , normal HbA1c and mild dyslipidemia. Liver function tests were as follows: Bilirubin 0.7 mg/dL, AST 93IU/L, ALT 57IU/L, GGTP: 762 IU/L, ALP :572 IU/L, albumin 2 gm/DL globulin 2.8 gm /dL Chest Xray was normal, USG abdomen revealed coarse liver,ascites, prostatomegaly, and urine R/E: Protein +++, no RBCs, no casts. The physician had gone on to do additional tests: an echocardiogram: which showed Grade 3 diastolic dysfunction, asymmetrical septal hypertrophy with speckling of IVS, a normal LVEF, suggesting possible restrictive cardiomyopathy. 24 hr urine protein estimation was 7 gm/dl and a CECT abdomen had been done which was reported as showing hepatic venous outflow obstruction, thrombus in splenic vein, and hypoattenuation of spleen.

He was referred to me by the physician in view of the CECT abdomen report.

If one considers the combination of fatigue, anorexia, nephrotic range proteinuria, infiltrative pattern of LFT abnormality and a restrictive cardiomyopathy,

the clinical possibility that immediately springs to mind is amyloidosis but the CECT findings do not fit in. On reviewing the literature on abdominal CT findings in hepatic and splenic amyloidosis, one learns that the liver findings can be quite nonspecific: heterogenous attenuation of the liver due to involvement of vessel walls, and what may help to differentiate is the marked hypoattenuation of the spleen due to hypoperfusion.[1] So, the next step was to request the radiologist to review the CT again to see if the findings would fit in, and she concurred that it was a possibility. After that the diagnosis was confirmed by serum inmmunofiaxation electrophoresis which showed a lambda monoclonoal gammopathy, elevated free lambda light chains ,a bone marrow biopsy showing a plasma cell dyscrasia with 7% plasma cells. On OGD there were no varices / there was antral gastritis with small ulcers in duodenal bulb: gastric and duodenal biopsies were done and were reported to show pale eosinophilic deposits with Congo red staining, showing applegreen birefringence to polarised light,resistant to potassium permanganate pretreatment, consistent with nonAA amyloidosis.

So how did technology fail in these patients ?

- Case 1: Technology (MRI brain showing empty sella) was useful for diagnosis, but not given importance because not enough attention was given to the patient's history of secondary amenorrhoea and lactational failure.
- Case 2: Wrong, expensive technology (MRCP for a nonhepatic origin of elevated ALP) was utilised in patient workup .
- Case 3: Technology (CT abdomen) was not essential for making a diagnosis,and actually misleading when taken out of context .

Lessons Learnt

1. The importance of the history and physical examination should never be underestimated To quote Sir William Osler: "Listen to your patient; he is telling you the diagnosis" Leading questions should be asked whenever appropriate. In a 1975 study by Hampton *et al*, of patients attending an outpatient medicine department, a correct diagnosis was made after taking the history in 66 out of 80 new patients; the physical examination was useful in only seven patients, and the laboratory investigations in a further seven.[2] A later study by Paley *et al* showed that 20 % of diagnoses can be predicted by history alone, physical examination increases the diagnostic accuracy by another 40% and basic investigations add a further 33%.[3]
2. It is good to follow well-established algorithms while trying to make a diagnosis.
3. It is important to form a provisional diagnosis and then ask for investigations to confirm this, and not the other way round .
4. It would be prudent as, Dr Herbert L Fred, MD, put it," to avoid the maladies of modern medicine, namely-technologic tenesmus: the uncontrollable urge to rely on the latest medical gadgetry for diagnoses and hyposkillia: a deficiency of clinical skills."[4]
   Unnecessary expensive tests should be avoided when simpler, cheaper tests can provide the diagnosis.
5. If a test does not fit in with a diagnosis, one should never hesitate to reexamine it, to request colleagues to review their findings when required.
6. We should never be afraid to admit if we are wrong, and should not hesitate to acknowledge that we may not always get the right answer .

In the end, we would be wise to remember Atul Gawande's words:

"We look for medicine to be an orderly field of knowledge and procedure. But it is not. It is an imperfect science, an enterprise of constantly changing knowledge, uncertain information, fallible individuals, and at the same time lives on the line. There is science in what we do, yes, but also habit, intuition, and sometimes plain old guessing. The gap between what we know and what we aim for persists. And this gap complicates everything we do."[5]

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