In the most advanced forms of medicine (regenerative, substitutive, genetic, targeted) two problems deserve reflection:

(i) Can medicine today claim the status of a proper science?
(ii) Are the results properly ranked by the impact factor (i.e. the Thomson Scientific Citability Index)?

The first question runs up against an immediate difficulty that science is always changing itself, unerringly shifting the confines of the various disciplines, plumbing the depths of human ignorance and reviewing the benchmarks of our knowledge. Again, what does scientific mean? Is there any property that would allow some, but not all, research to be termed scientific? In what sense can scientific propositions be attributed the value of truth to nature?

One thing to be said for Medicine is that, unlike many other disciplines, it neither simply describes, nor searches for a calculus; it possesses a highly important basis for assessment: it judges by the facts, which can then be tested by their correspondence with reality as seen from medicine’s multidisciplinary angles.

Much of the merit for this can be traced to Nephrology, whose great feats of application (especially dialysis and transplantation) are recognized among the major milestones in the evolution of medicine from Hippocrates to today.

Up to 30–40 years ago, the fact that death of a vital organ no longer spelled death for the individual was ‘miraculous’. Organ replacement (dialysis and/or transplantation) ensured survival. Survival might be good enough (good or spectacular), depending on the replacement programme.

- In terms of clinical and social rehabilitation, transplantation provides the best results, but can be performed in no more than 30% of patients, which creates class I and class II discrimination.
- Dialysis is being applied more and more widely, but the long-term cost/benefit verdict is far from satisfactory.

To reach the status of a proper science, any branch of knowledge must involve a combination of theory and application (the conceptual and procedural knowledge of the present day). Such simultaneous development has only occurred in a few disciplines. One is bacteriology/virology and its clinical pharmacology, which has led to the disappearance of many infectious diseases. This is ‘true progress in medicine’: a winning solution, striking at the cause and completely rehabilitating the patient, at minimal cost to society.

Unfortunately, in the last 30 years the applicative evolution of most medical disciplines, including nephrology, has not always been matched by a parallel evolution in cognitive theory, and the consequences of this gap are quite clear today.

As a direct result, dialysis has witnessed a progressive inbreeding of cognitive science, a progressive recurrence of the drawbacks of applied technology: a satisfactory survival rate in dialysis patients; but in general, rather disappointing in all other expectations (five times higher hospitalization than normal, 60% unemployment and 40% disability rates).

Transplantation is still performed via protocols fixed by the clinician according to some alleged pharmacodynamics of the drugs and not to the specific immunological needs of the patient. Other disappointing long-term results may occur: progressive patient–doctor detachment; patient discrimination, social and family difficulties, present frustration and future anxiety.

This mismatch occurring in the evolution of medical knowledge is hardly surprising if one reflects on the grounds on which nephro-thinking has evolved in the last 20 years. In ancient Greece, classical medicine developed in a simultaneous cosmological vision (health and disease as part of a philosophical and environmental whole). From the Renaissance on, the vision became more anthropological (the patient as the prime object or subject). In contrast, modern medicine (including nephrology and its application) has evolved in largely technological stages, in which the gap between theory and application has grown even more evident due to the tendency to advance applicative proposals without any precise cognitive foundation.

And thus, from the reality of 40 years back—survival guaranteed by renal replacement therapy—we come to our present reality: what counts is no longer survival but the quality of life.

How can the scientific standing of nephrology be assessed on its applicative performance? One certainly has reason to doubt whether the results are being properly graded by the impact factor (i.e. the Thomson Scientific Citability Index).

This is not the place to discuss the merits (or ethics) of Thomson Scientific citability. Like all forms of snobbery, it penalizes the outsider, newcomer or plain modest and favours the self-advertising, and the ‘in’ set. It is statistics applied to name-dropping—an unsavory combination, most people might think, but promotion, careers, grants and the funding of whole institutions are coming to depend on it, from Far East to Far West. Equally serious, whereas the catch phrase ‘impact factor’ is on the lips of the majority, how many know how it really works, or who Thomson Scientific are; what their formula is based on, or the fact that a subjective Thomson selection decides both elements of that formula (what journals deserve to be ‘indexed’, and what articles are deemed to be ‘citable’).

Perhaps we would do better, in judging a new medical product, to stick to consideration of its scientific standing, if we can agree on how scientific medicine actually is. Adequate professional, impartial assessment becomes especially important in disciplines—renal replacement is one—where the real impact is on millions of patients worldwide.

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**Acute renal failure and chronic lymphocytic leukaemia**

**Sir,**

Renal infiltration is often associated with chronic lymphocytic leukaemia (CLL). However, it is infrequently the direct cause of kidney failure. In CLL, post-renal obstruction with intra-abdominal [1–5] lymphadenopathy and/or increased incidence of uric stones, Bence–Jones proteinuria and/or cryoglobulinaemia can cause kidney failure indirectly [1,5].

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Case

A 69-year-old man with a 2-year history of lymphoma had CT thorax/abdomen/pelvis from 2 years prior to our evaluation that revealed extensive lymphadenopathy consistent with a diagnosis of CLL. The patient had no known drug allergies and was not on medications. The patient had had a kidney biopsy 1 year prior to our evaluation that revealed severe tubulointerstitial nephritis and mild interstitial fibrosis. His creatinine had progressed from 214 to 279 over 6 months, until 3 months prior to this presentation. At this time, he presented with dysphagia, without haematemesis, fever or diarrhoea and his serum creatinine was noted to be greater than 900 mmol/l.

Investigations

Revealed a haemoglobin of 99, platelets $287 \times 10^9/l$, white cells $79.0 \times 10^9/l$, lymphocytes $56.2 \times 10^9/l$ and presence of smudge cells. A CT of the abdomen and pelvis revealed extensive lymphadenopathy with bilaterally enlarged kidneys and no evidence of hydronephrosis. The patient did not demonstrate Bence–Jones proteinuria or cryoglobulinaemia and thus a kidney biopsy was performed (Figures 1 and 2).

The pathology results indicated diffuse lymphocytic infiltrate consistent with B-cell lymphomproliferative, small cell type, in keeping with the diagnosis of CLL. Immunohistochemical study confirmed the diagnosis with positive staining of the neoplastic cells for CD20, CD79, CD5 and CD23.

Unfortunately, the patient adamantly declined chemotherapy and haemodialysis was initiated. At present time, he remains dialysis-dependent.

Discussion

In the absence of other obvious explanations for this patient’s acute renal failure, we presume that it is secondary to malignant infiltration by his CLL. However, we could not confirm this by treatment with chemotherapy, due to the patient’s adamant refusal. In hind-sight, it is presumed that his previously diagnosed tubulointerstitial nephritis was the early manifestation of his lymphocytic renal infiltration due to his CLL.

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Idiopathic tubulointerstitial nephritis and uveitis—‘TINU syndrome’

Sir,

Acute interstitial nephritis (AIN) is an important cause of acute renal failure (ARF). Kidney biopsies for evaluation of causes of ARF reveal AIN in 15% of the lesions [1]. Acute renal failure due to idiopathic tubulointerstitial nephritis and uveitis (TINU) syndrome is an uncommon clinical entity [2],