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Sir,

Recent events in the country have once again brought into sharp focus the urgent need for the medical fraternity to address issues of medical ethics in clinical medical practice. There have been warning signals earlier also but these have failed to elicit an appropriate response from the professional medical community. This situation cannot continue for long. We cannot persist with an ostrich-like attitude that assumes that all is well because public concerns seldom reach explosive dimensions, or that, public memory being short, such events as do come to public view will soon be forgotten and leave the profession untouched in the long run. Both assumptions are rapidly losing relevance. Media smoke cannot be dismissed as an aberration here or a vested interest there; surely there must be some embers if not muted fires. Indeed private conversations point to a widespread belief that issues of major concern do indeed exist. Female foeticide, caesarian operations on demand, organ transplantation, clinical use of new drugs, use of new devices, and skills necessary for clinical application of new technologies are but some of the issues.

It is the view of this writer that time is running out and that the medical fraternity must act fast and decisively if the present concerns of the community are to be addressed and the situation retrieved for the profession. Already there is considerable erosion of public trust in medical professionals and our status in society has been on a continuous downslide for sometime. Today the general perception is that commercial considerations override the professional and social commitment of medical professionals. Alas! medical professionals have fallen off the pedestal. The pointers are all there for even the blind to see. What better evidence of the erosion of public trust than the common scenario of patients trying to cross-check and recheck the advice given to them even when they are in no position to evaluate specific advice that they may obtain from various quarters. This was not the scenario in the past, and is not the scenario in other parts of the world even today. Lest my statement be misconstrued as an assault on patient autonomy, let me hasten to state, quite clearly and unambiguously, that there is nothing wrong with a second opinion provided the patient is informed choice is, at best, self-defeating. Without digging further in the matter, which could be a subject of a separate discourse, it seems to me quite clear that the common spectacle of shopping around by the patient in our scenario is a manifestation of lack of trust in professionals and that should be a matter of serious concern for all of us.

Further, it is generally recognised that the preference shown by students setting out to seek a career for themselves is indicative, amongst other things, of the social standing of the profession. Once again there has been a general decline in the number of brilliant students opting for a career in medicine in this country. One of the reasons for this change in student preference is the decline in the social standing of a doctor in the community.

The need to bring the medical profession in the purview of the Consumer Protection Act was perhaps the first indication of a change in public perception regarding our profession. Alarm bells should have rung then. They did not. Instead we chose to look elsewhere and transfer blame to collateral, less important, issues. Consequential developments have lead to a predictable erosion of the self-confidence of medical professionals with serious repercussions on day-to-day patient management and avoidable cost escalation that impacts most adversely patients with limited financial muscle. If the designated professional regulatory authority had taken timely steps to inspire public confidence such a step may not have been necessary. That was not to be then and it hasn’t happened since.

Contrary to what all of us would like to believe, all is not honky dory with the medical profession. Clearly there is an urgent need for action, not a pat on the back for a quid pro quo. All professional bodies, especially the Indian Medical Association, have to wake up and come up with a credible plan of action to uphold and promote ethical standards in the medical profession. They must lay down their own code of conduct, define clearly the minimum ethical standards that would be acceptable, and develop a monitoring system that will reward good ethical practices while punishing deviant behaviour. It is far too easy to shirk responsibility and hide under the excuse that professional organisations in the country do not have any teeth. Sound ethical standards pursued unflinchingly and honestly are a power in themselves and do not need any legislative support. Professional solidarity built on an edifice of moral and ethical values is a formidable force that no one dare challenge, whether a professional colleague, a community member or even the executive. Even if legislative support is considered necessary, there already exists a mechanism in the form of the Medical Council of India. That Khumbhkaran could be stirred into action by collective persuasion by the medical fraternity, there are a number of ways to tackle the issues provided there is a will to take cognizance of the problems and act. If we don’t do so on our own, and soon, someone else will do it for us. That is for sure. We can no longer wish away the problem without further diminishing our standing in society. There is nothing wrong with striving for a secure, even lavish and flamboyant, lifestyle provided the means are honest and ethical, and that the means protect the best interests of the patient, and thereby also the best interests of the doctors.

Any coffee room discussion on the subject usually ends up with the remark that we are part of a society most segments
of which do not give a farthing for ethical standards. That may well be true but it is a weak argument in support of the status quo. One robbery cannot justify another. The medical profession has been a torchbearer for ethical values through the ages, which is what glorifies the profession. That is what constitutes the only basis for trust in the doctor-patient relationship; that is why the doctor-patient relationship is not based on cold numbers or algebraic equations but blossoms on a warm commitment of trust. It is time to reinforce that trust and restore the profession to its rightful glory and ourselves to peace of mind.

This presentation is not driven by any sense of altruism, only by cold logic to preserve and promote the best interests of the profession. The present global movement towards transparency in all walks of life cannot leave the medical profession untouched for long. The only question for debate is whether we, in the profession, act in time to put our house in order, or procrastinate till others do it for us. That is the only choice, and what a choice! Mere cosmetic conformity with legal provisions will not provide a lasting solution; the code of ethics must permeate an awakened conscience of the profession. Historical or logistic reasons do not mitigate the calling of the present as well as the future.

The author would urge readers to write to the editor of this journal, or to the author, in support, or otherwise, of the proposed agenda. That would help to persuade any faltering minds on the subject. Even one-liners on a postcard would do. It is time to stand up and be counted. The future is not for the mind.

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### Brucellosis as a Cause of Prolonged Fever in an Era of Pasteurized Milk

**Sir,**

A 40 years male presented with a history of low grade fever (Maximum 100.6°F), occurring between 12 noon to 11 pm without any chills or rigors of two months duration along with profuse sweating, headache, joint pain, muscle pain, weakness, weight loss (5 kg), mild abdominal pain and feeling of depression. Physical examination did not reveal anything except mild epigastric tenderness. Drug history revealed that the patient had been treated empirically with ofloxacin for 10 days without any positive response, along with paracetamol, nimesulide and aspirin individually on various days for fever.

Investigations revealed - Hb 13.6 gm%, TLC 5000 cells/mm³, DLC P54 L41 M1 E4, ESR (Westergren) 9 mm/1st hour, malarial parasite not seen, widal test negative, urine-NAD, stool microscopic examination was normal but occult blood was positive. Liver and kidney function tests were normal. Blood and urine cultures were sterile after 48 hours of incubation. X-ray chest PA view was normal. Montaux test was negative. Serum amylase was normal. Upper GI endoscopy showed petechial erosive spots in body and antrum of stomach possibly NSAIDs induced. Histopathology of gastric antral biopsy showed normal gastric mucosa. There were nonspecific mononuclear infiltrates in the lamina propria. No *Helicobacter pylori* were seen. ASO titres and C-Reactive protein were within normal range.

Patient’s dietary history revealed that he was a vegetarian. He had been consuming flavoured chilled milk daily in the past two months preceding the illness. In view of this history, Brucella antibody test was suggested. Standard tube agglutination (STA) test was positive in the titres of 1:320. Brucella total antibodies against *Brucella abortus* and *Brucella mellitensis* were positive. By ELISA, *Brucella abortus* IgM antibodies were 33 U/ml (Normal less than 15 U/ml), while IgG was less than 5 U/ml (Normal less than 20 U/ml) thus making a diagnosis of acute brucellosis. Patient was treated as per recommendation of WHO expert committee on brucellosis,¹ with a 45 days course of Doxycycline 200 mg daily plus Rifampicin 900 mg daily along with supportive treatment.

By the 7th day, patient became afebrile along with the disappearance of associated symptoms. In the last one year of follow-up patient has no recurrence of symptoms.

This case highlights the fact that brucellosis should be kept in the differential diagnosis of any patient with fever with paucity of findings. In an interesting study from the Department of Clinical Microbiology, CMC, Vellore, Brucella serology using ELISA and STA was performed on 23 patients with prolonged fever where the test was requested, on 26 randomly chosen patients with prolonged fever where it was not requested and on 17 controls. ELISA was positive in 39%, 27% and 0% respectively in these groups hence concluding that brucellosis may often be unsuspected because of its varied clinical manifestations and may be a more important cause of fever than previously considered.² Even in developed nations the true incidence of brucellosis may be up to 26 times higher than official figures suggested. Only 4% of cases are recognised and reported in USA.³

Brucellosis does not cross the mind of a treating physician in city bred vegetarian patients who have access to only packed dairy pasteurized milk and milk products.

In this case blood culture was sterile, *Brucella* is slow and fastidious to grow in vitro, cultures must be kept and examined for four to six weeks.⁴ It should also be kept in mind that ofloxacin and other quinolones have fairly good activity against *Brucella*.⁴

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**A Case of Sensory Guillain Barre’ Syndrome**

Sir,

Guillain-Barre’ syndrome typically presents as an acute onset of bilateral, symmetrical, progressive weakness of limbs and sometimes of the trunk and cranial musculature, associated with absent or decreased deep tendon reflexes and CSF albuminocytological dissociation. GBS presenting as an acute monophasic sensory neuropathy is rare. Only few case reports are found in literature. We report a patient with sensory neuropathy, normal motor power, absent deep tendon reflexes, high CSF protein and nerve conduction studies showing features of demyelination.

A 41 years male presented with acute onset of numbness and tingling sensation in both hands and feet. This ascended up to involve the whole body sparing the face, before it stabilized within three weeks of onset. He did not have any weakness of limbs, diplopia, dysphagia, dryness of mouth, postural giddiness, bladder or bowel symptoms or arthralgia. There was no history of any antecedent illness, loss of weight or appetite, exposure to drugs or toxins. Neurological examination revealed, normal cranial nerves and muscle strength. He had total areflexia. Sensory examination revealed loss of vibratory and proprioceptive sense with preserved light touch and pinprick sensations in all four limbs and trunk below neck. He had a wide based stamping gait. Romberg’s sign was positive. Laboratory tests with normal or negative results included: complete blood count, blood glucose, liver and renal function tests, HIV, VDRL, ANA, rheumatoid factor chest X-ray, ultrasound abdomen and stool occult blood. CSF study revealed albuminocytological dissociation. Electrodiagnostic study revealed bilateral demyelinating sensory motor neuropathy. After refusing IV immunoglobulin treatment, the patient was treated with a course of prednisone (50 mg/day PO for two weeks and tapered off in two weeks) and he had a complete resolution of signs and symptoms by two months.

Progressive motor weakness of more than one limb is one of the features of the classic diagnostic criteria of GBS. Sensory variant of GBS will not meet these criteria. Hence, the following diagnostic criteria for GBS variant with sensory loss and areflexia have been described. The onset must be rapid; peak deficit achieved within four weeks; diminished or absent tendon reflexes; normal motor power; nerve conduction evidence of demyelination in at least two nerves; monophasic course; no other known cause for neuropathy; elevated CSF protein during the acute phase of disease; and no family history of neuropathy. Our patient presented with symmetric sensory loss with areflexia and normal strength. The CSF revealed albuminocytological dissociation. The symptoms stabilized within three weeks of onset and the patient began to show improvement within a month of stabilization. Our case satisfies all the criteria for sensory GBS. Recently, Oh SJ et al reported eight cases of sensory GBS and his study confirms the existence of sensory GBS. From his study, it is evident that acute sensory neuropathy represents two clinical syndromes: Acute sensory neuropathy involving the dorsal root ganglia and Sensory GBS - an acute demyelinating neuropathy with only peripheral sensory nerve involvement. Acute sensory neuropathy is characterized by absent SNAPs and normal motor nerve conduction, whereas sensory GBS is characterized by a demyelinating pattern in both motor and sensory nerves. CSF protein does not help to distinguish between the two, because it may be elevated in both. The diagnosis of sensory GBS is important because it helps the physician in planning potential immunotherapies, provides useful prognostic information and permits reassurance to the patient and family.

**REFERENCES**


**New Frontiers of Therapy in Hemato-Oncology**

Sir,

We read with interest the article “New frontiers of therapy in hemato-oncology” (J Assoc Physicians India, Vol. 50, July 2002:950-61).

We are impressed on the academic aspects of the article new frontiers of therapy in hemato-oncology. The classification and the probable clinical applications are very well covered. In our opinion some of the practical applications are missing, which we will like to share.

FDA has approved Rituximab for CD-20+ve low grade non-Hodgkin’s lymphoma (NHL) relapsed or refractory after standard chemotherapy. This has not been approved for other types of non-Hodgkin’s lymphoma.

Only three approved new biologic agents are mentioned in the article. There are few more biologic agents approved by Food and Drug Association (FDA) and commonly used in hemato-oncology.

Mylotarg, is a humanized anti CD-33 Ig G4 antibody bound to the potent antitumour antibiotic calicheamicin. In phase II trial, 142 relapsed acute myeloid leukemia (AML) patients
were treated with 9 mg/m² of Mylotarg on day 1 and 14. Overall 30% of patient achieved complete remission. This molecule has been approved by FDA for the treatment of CD-33+ve AML patients in first relapse who cannot tolerate intensive treatment.¹

Campath-1H (Alemtuzumab) is a humanized IgG1K monoclonal antibody that targets the CD 52 antigen, expressed on normal and malignant B and T lymphocytes, as well as NK cell, monocytes and macrophages. Chronic lymphocytic leukemia (CLL) has been most extensively studied with this agent. In fludarabine resistant CLL, response rate of 33% has been achieved. First dose generally consists of 3 mg administered over 2 hrs intravenous infusion on day 1, followed by 10 mg on day 2 with subsequent dose of 30 mg/day three times per week up to 12 weeks. FDA has approved this compound for refractory CLL.²

STI 571 (Imatinab mesylate) (Gleevec) inhibits Bcr-Abl tyrosine kinase. In phase II trials STI 571 induced complete hematological response in 91% patients with Interferon failure chronic phase chronic myeloid leukemia (CML), 34% in accelerated phase and 7% in myeloid blast crisis. Major cytogenetic response was observed in 55% of patients with interferon failure chronic phase CML, 24% with accelerated phase CML and 16% patient with myeloid blast crisis CML. The dose used is 400-600 mg/day. FDA has approved this for interferon failure chronic phase CML patients, accelerated phase CML and myeloid blast crisis CML.³

Thalidomide is an antiangiogenic agent. This also acts by inhibiting TNF α production. Thadiomide, at a dose of 200 mg/day induced response in 44% of patients with relapsed multiple myeloma. Remission is maintained in 100% of patients at six months and 22% of patients at two years. This dose is commonly used in refractory or relapsed multiple myeloma patient.¹

REFERENCES

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Sir,

I wish to point a few new advances in response to the Update entitled “New Frontiers of Therapy in Haematological Oncology”. I would like to make the following comments.

STI 571 has got one of the most rapid approvals from US FDA. An article comparing STI 571 and Interpritation Cytarabine (the IRIS trial) was published in the New England Journal of Medicine. An editorial in the same issue entitled “Imatinib Mesylate - The New Gold Standard for Treatment of Chronic Myeloid Leukemia” mentions “Imatinib (STI 571) now seems to be the initial treatment of choice for patients with CML who do not have a suitable bone marrow donor or who are not candidates for transplantation.” Anticancer therapy is toxic. Haematologists are most concerned about severe (grade III and grade IV) toxicity. The toxicity figures quoted by the author are way off the figure published in abovementioned article.

<table>
<thead>
<tr>
<th>Event</th>
<th>Author's Claim</th>
<th>Published data</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haematological and Cytogenetic response</td>
<td>97.7%</td>
<td>95.3% (93.2-96.9%)</td>
</tr>
<tr>
<td>Hepatic toxicity</td>
<td>50%</td>
<td>85.2% (81.9-88.0%)</td>
</tr>
<tr>
<td>Hepatic toxicity</td>
<td>24.5%</td>
<td>43.2% with elevated transaminases; only 5.2% has grade III/IV toxicity; Number of patients who discontinued therapy because of all forms of drug toxicity : 12/553</td>
</tr>
<tr>
<td>Major haematological toxicity</td>
<td>77.6%</td>
<td>Anaemia 3.1%; neutropenia 14.3%; thrombocytopenia 7.8% (grade III/IV toxicity, which is the only toxicity of clinical significance).</td>
</tr>
<tr>
<td>Cardiac failure</td>
<td>Occurs</td>
<td>Not mentioned</td>
</tr>
<tr>
<td>AMI</td>
<td>Occurs</td>
<td>Not mentioned</td>
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</tbody>
</table>

Everyone with an interest in haematology has been keenly following the development of STI 571. Publication of the IRIS trial was an eagerly awaited culmination of events which started with the discovery of the Philadelphia chromosome in 1960. The STI 571 alongwith rutiximab are the two success stories of the “New frontiers of haemato-oncology” (many more like Mylotarg, and Campath-1H are “just behind the frontier”).

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Quality of Life in Type 2 Diabetes

Sir,

The April 2003 issue of the Journal carries two articles that show the directions to research and clinical care in this millennium. On the one hand is an Update on Human Genome Project;¹ what is remarkable is the previous Update on HGP in the Journal was published less than two years ago.² In this short time, so much clinically relevant information has accumulated that the 2003 Update could not by any account be faulted for repetition from its predecessor. I cannot imagine
there are areas of biological science where such rapid progress has occurred. And the pace is torrential, with proteomics on the heels of genomics.

On the other end is a paper on quality of life in diabetes.1 Whereas the former can be considered the glamorous face of science, the latter is the foot soldier. Neither is redundant. Diabetes mellitus is an increasingly common prototype chronic condition in which the focus is on care, rather than cure.4 Enabling individuals and their families to live with it is a pivotal though difficult aspect of management. Neither psychological well-being nor metabolic control is to be pursued in isolation. Rather, a balance between the two must be struck. Using the same psychological instruments, Indian women were shown to have poorer quality of life, well-being and adjustment to diabetes.3 Even using a composite ‘living with diabetes score’ from quality of life, well-being and psychological adjustment to diabetes, men were shown to be living more effectively with diabetes.6 The gender difference is unlikely to be disease-specific.4 Given the importance of this crucial information, we need to ‘recognize the extra burden she already carries when the next women with diabetes presents to us’.

A variety of validated diabetes-specific psychological instruments are available. If data can be generated from different geographical, social and cultural regions, they can be universally applied in patient care and in medical audit.

The two articles have together, shown fascinating glimpses into the future, while highlighting what can be readily applied, starting now.

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REFERENCES


Comment

Sir,

Rapid expansion of clinical information provided by researches as told by Dr Sridhar is eminently true. Dr Sridhar cites his studies indicating that focus is on care rather than cure. It is my opinion that the two are interlinked and thus interactional.

Regarding gender differences found by him and us point out that men live more effectively with diabetes than their women counterparts. We agree that differences are unlikely to be disease-specific. Gender differences encompass a whole gamut of sociological and psychological factors. We and others usually report studies based on ex-post facto research endeavour.1 It may be desirable, while not eschewing ex-post-facto design to utilize factorial research designs2 which could partition various sources of variance on dependent variable. In addition we have to have collaborative research endeavours with disease specific measures in different samples of subjects from different segments of our society using multistage sampling procedures. Post-present future being interlinked, the future possibilities unfold themselves based on research out-put.

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Is Sildenafil Safe with Alcohol?

Sir,

With the introduction of effective oral agents for treatment of erectile dysfunction, more men are seeking treatment. The underlying cause of the erectile dysfunction is usually a chronic medical illness like hypertension or diabetes.1 In the literature the most commonly reported cardiovascular adverse effects in patients on sildenafil are headache (16%), flushing (10%) and dizziness (2%). These side effects are due to the vasodilatation caused by sildenafil. The incidence of hypotension, orthostatic hypotension, and syncope and rate of discontinuation of this drug is < 2%.2 The concomitant use of other vasodilators like nitrates is contraindicated with sildenafil as both increase cGMP level in the systemic circulation but at different points along the NO-cGMP pathway and result in potent vasodilatation and may cause significant reduction in blood pressure and side effects.3

We observed potentiation of the side effect of sildenafil in a patient consuming alcohol with it.

A 36 years male hypertensive was on regular treatment with calcium channel blocker (Amlodpine 5 mg/day) since three years with good blood pressure control. He used to consume alcohol occasionally at parties but never more than two drinks of 30 ml each. He started complaining of erectile dysfunction since six months and was prescribed sildenafil citrate in the dose of 25 mg/day. He tolerated the drug well
and achieved normal sexual function. He was using sildenafil regularly three times a week without any side effects. One night after a party where he had two regular drinks of whiskey (120 ml of Royal Challenge : 55.2 gm of alcohol) he took the prescribed drug within an hour of having consumed alcohol. He experienced severe headache and flushing after about 15 minutes of taking the drug. Headache subsided after three hours on taking nimesulide. He reported this to us in the OPD and was asked to take 25 mg sildenafil the next day without consuming any alcohol. This time he did not develop headache or flushing. A challenge of sildenafil in the same dose after a single drink of 30 ml whiskey was given after one week to establish the correlation between the two. A similar kind of severe headache and flushing was reported, confirming that concomitant ingestion of alcohol and sildenafil resulted in this side effect. He was advised not to consume sildenafil when he had had alcohol and since then he was not developed this side effect.

Alcohol though is a central nervous system depressant it also results in peripheral vasodilatation. The concomitant use of alcohol and sildenafil may result in significant vasodilatation resulting in headache and flushing. However this side effect was not seen when either of them (alcohol or sildenafil) was consumed alone in our case. This interaction between alcohol and sildenafil has not been studied extensively. The product information inserts of sildenafil provided along with drugs do not supply sufficient information regarding its interaction with other agents like antihypertensive drugs, protease inhibitors, alcohol and drugs inhibiting cytochrome P450. The package insert state that in healthy adult volunteers the hypotensive effects of alcohol were not potentiated when co-administered with sildenafil, when the mean maximum level of alcohol was 0.08%. However this information is grossly insufficient as we do not know the age of these volunteers and its interaction in presence of co-morbid conditions. This information is a must before prescribing these drugs to diverse population so as to avoid potential adverse events. In this case we have seen that concomitant use of sildenafil with alcohol may result in distressing side effects and it should be avoided. More information is needed about these potential interactions. The assumption that sildenafil is safe with alcohol need sufficient studies to establish its safety.

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Inverted Biceps Jerk

Sir,

Clinical signs in neurology are best known for their reproducibility. They help in localizing the pathological lesions both during clinical practice and during examinations. The skill of the student is often assessed in the clinical examination of neurology by eliciting these signs and their proper interpretation. But sometimes interpretation of a physical sign may be different in different books. Inverted biceps jerk is one such example.

One book describes it as absence of biceps contraction but brisk flexion of fingers on eliciting the biceps jerk. Whereas another book describes it as tapping of biceps tendon may fail to elicit the biceps jerk but gives contraction of triceps. Still another book writes that if tapping the biceps tendon causes a contraction of finger flexors it is a sign of upper motor neuron lesion. Inverted biceps reflex, the biceps does not contract at all, but tapping its tendon smartly causes a contraction of the triceps and hence extension of elbow. This results from the unopposed response of the triceps as a slight lengthening of its tendon is sensed.

Inverted biceps jerk is considered pathognomonic for a C-5/C-6 disc herniation with a C-6 radiculopathy. It is useful in localizing the pathological lesion in the spinal cord. Lesion at C-5 interrupts the reflex arc for biceps reflex but associated compression of the corticospinal tract produces increased response in other reflexes supplied by cervical segments below C-5. Triceps muscle is supplied by C 6-7 and finger flexors by C 7-8. Elicitation of biceps jerk is one such example.

One book describes it as absence of biceps contraction but brisk flexion of fingers on eliciting the biceps jerk. Whereas another book describes it as tapping of biceps tendon may fail to elicit the biceps jerk but gives contraction of triceps. Still another book writes that if tapping the biceps tendon causes a contraction of finger flexors it is a sign of upper motor neuron lesion. Inverted biceps reflex, the biceps does not contract at all, but tapping its tendon smartly causes a contraction of the triceps and hence extension of elbow. This results from the unopposed response of the triceps as a slight lengthening of its tendon is sensed.

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