creatinine, lipid profile and thyroid functions were normal. CT scan of head was normal. A provisional diagnosis of Flx - induced tremors was made and she was advised to discontinue Flx and to start a tricyclic antidepressant i.e. amitriptyline. She showed remarkable improvement within 7 days of discontinuation of Flx and had no tremors of both hands. The patient however, again showed up 2 months later with the previous complaints. On enquiry she confided that she had restarted Flx one month following its discontinuation. She was again switched over to amitriptyline and she continued to remain symptom-free on subsequent follow up.

In the differential diagnosis of tremor, drug induced tremors (DIT) must be considered since tremor is a common side effect of a wide range of drugs. DIT is most characteristically a postural or action tremor, causing a variable degree of disability. However, classification of individual tremor into resting, postural or kinetic tremor is difficult because it often appears as irregular tremulousness associated with more complex involuntary movements such as chorea, dystonia or myoclonus.

Flx is the most frequently used antidepressant in clinical practice. Flx is a specific serotonin reuptake inhibitor (SSRI), presumed to be working by desensitising both inhibitory somatodendritic and terminal 5-HT autoreceptors, thus increasing central nervous system 5-HT synaptic transmission. It has also been reported to be effective in premenstural syndrome, fibromyalgia, hypochondriasis, obesity, chronic pain etc.[1] Although considered by many, to be the drug of first choice in depression for its favourable side effect profile, Flx unfortunately has been associated with many side effects. It is reported to cause akathisia like manifestation characterised by agitation, restless motor movements, dysphoria, pacing and internal sense of desperation and suicidal ideations. High dose may cause a syndrome resembling a frontal lobe syndrome characterized by apathy, indifference, inattention and perseveration. It has also been proposed to be a possible cause of syndrome of inappropriate secretion of antidiuretic hormone (SIADH). Tremor is an uncommonly reported side effect during Flx therapy[2] and following its withdrawal.[3]

A number of drugs stimulate the peripheral adrenergic system directly and produce an enhanced physiologic tremor. Physiologic tremors can be increased in amplitude by exogenous catacholeamines. This action also could be induced by exogenous adrenergic drugs such as epinephrine, norepinephrine, theophylline, beta adrenergic agonists, levodopa, amphetamines, caffeine and steroids. These drugs are believed to act directly on peripheral beta receptors. Neuroleptics can produce exacerbation of physiologic and essential tremors and can cause tardive tremor in addition to parkinsonian tremor.

Flx acts predominantly on central serotonergic system in addition to cholinergic and, catecholaminergic systems in generation of tremors. Hence in clinical practice it is essential to obtain history of Flx intake as a possible cause of tremors in a depressed individual as Flx - induced tremors are reversible and rarely present as a life threatening situation.

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Fibrocalculous Pancreatic Diabetes in a Patient from Bihar

Sir,

Fibrocalculous pancreatic diabetes (FCPD) is a form of diabetes prevalent in tropical countries which is secondary to chronic, non-alcoholic, calcific pancreatitis. FCPD has been reported to be much more frequent in southern states of India and is less common in northern India.[1] We here report on a case of classical fibrocalculous pancreatic diabetes from Bihar State, which is probably the first case identified from this state.

Mr. DK, a 20 year old male student from Ranchi, Bihar, presented to our centre, in April 1998 for control and assessment of diabetes. He had been diagnosed to have insulin dependent diabetes mellitus (IDDM) in 1995 at Bihar at the age of 17 years and had been treated with insulin for all the 3 years. He gave a history of recurrent abdominal pain first noted in 1993. He lost more than 10 kgs in weight. He also gave a history of steatorrhoea (passing of greasy/oil stools) especially after intake of fatty food. He did not smoke or drink alcohol. He was a vegetarian and consumed chappatis and rice. He did not take jowar, sorghum or tapioca. His body weight was 51 kg and body mass index 18.7 kg/m^2. On admission, his random blood sugar was 315 mgs% with a glycosylated haemoglobin (HbA1c) value of 15% indicating poor glycaemic control for the past 2-3 months. He did not have any diabetes related complications like retinopathy, nephropathy or neuropathy.

Plain abdominal x-ray showed a multiple pancreatic calculi. Ultrasound abdomen showed evidence of dilated pancreatic ducts. CT scan also corroborated the ultrasound findings. An endoscopic retrograde cholangio pancreatography (ERCP) was done which showed a dilated main pancreatic duct, dilated side branches and pseudocyst formation. Faecal chymotrypsin assay, an index of pancreatic exocrine dysfunction, was done and this showed a value of 1.5 units/gm indicating evidence of impaired pancreatic exocrine function.

Our patient thus fits all the criteria for FCPD as laid down by Mohan *et al*[2] and hence there is no doubt of the diagnosis in this patient. To the best of our knowledge there is no report of FCPD from the state of Bihar. The presence of a case of FCPD from Bihar picked up at Chennai, points to the possible presence of many more cases occuring in Bihar. It is obvious that once one is aware of the condition and has a high index of suspicion, the diagnosis can be easily made.

Making a diagnosis of FCPD is essential for several reasons. The cause of abdominal pain being known, proper treatment can be offered for the same if the pain recurs. Addition of pancreatic enzymes may help to alleviate symptoms of steatorrhoea and promote weight gain and improve general health. Finally, since FCPD is a pre-malignant condition,[3] one should be aware of possibility of occurrence of adenocarcinoma of the pancreas in FCPD patients.

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The Dengue Fever Epidemic in Delhi

Sir,

I read with keen interest the article entitled 'Experience in adult population in dengue outbreak in Delhi' by Dr. Tripathi *et al* published in the March 1998 issue of JAPI, which was indeed enlightening. I wish to compliment them for highlighting the fact that the panicky situation regarding the requirement and availability of platelet concentrates, created in the media due to irresponsible statements made by certain section of the medical fraternity, was an unnecessary and avoidable complication. It only created problems for those managing such cases first hand. Indeed, platelet concentrates were rarely required and it is doubtful if such concentrates alone would be able to control bleeding, which, as the authors mention could result from numerous other factors. Besides, the life of transfused platelets under such circumstances is extremely short.[1]

In the aforementioned context, I would like to briefly mention about 10 patients of dengue fever, from over 100 who received treatment at our hospital. These cases can be classified into three broad groups. The first group consisted of 4 patients, who presented with an initial platelet count of 15,000/mm^3 or less, a positive tourniquet test, no overt bleeding manifestations and with hemodynamic stability. This group was monitored closely for overt or internal bleeding and hemodynamic instability. No active management was required. The platelet counts started increasing on the second or third day to over 100,000/mm^3 by day 6. The second group consisted of three patients, who presented with overt bleeding from multiple sites. Curiously, all these patients had platelet counts of over 60,000/mm^3 at admission. These patients were managed actively with replacement therapy. A third group of patients (3 in number) presented in shock without overt bleeding. Of these, only one had a platelet count of less than 100,000/mm^3, but each one of them had a history of sudden defervescence of fever on the 4th or 5th day of a febrile illness.

One other case that I would like to mention is of a young male who presented during the epidemic with fever, purpura, bleeding and thrombocytopenia. In this patient, smears from purpuric skin lesions demonstrated the presence of intracellular Gram negative diplococci. It is therefore important that in a tropical country like India, even during epidemic like the one we had, one needs to keep an open mind regarding other diseases with similar clinical presentation, and that are curable with effective and appropriate chemotherapy.

The authors have mentioned about disseminated intravascular coagulopathy (DIC) as the cause of death in 3 out of 11 patients who died. This is an interesting observation that has been described earlier also.[2] This finding is interesting and important on two counts. Firstly, it could be a mechanism of thrombocytopenia, in addition to immune destruction and marrow suppression.[3] Were all 560 patients investigated for the presence of DIC with a complete coagulation profile and FDP assay or was such investigation limited only to patients with overt bleeding? The second point of importance pertains to this fact. If DIC is demonstrable in cases of dengue without bleeding also, it could be of value in developing an index, combining results of coagulation profile, FDP assays and platelet counts in prediction the occurrence of hemorrhage in patients with dengue fever.

One last thing I would like to mention is regarding the methodology of platelet counts. An optimal manual count should always supplement counts from automatic or semi-automatic counters, especially in patients with reportedly very low platelet counts.

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Reply from the Authors

Sir,

We thankfully acknowledge the interest shown by Dr. Atul Goel in our article "Experience in adult population in dengue outbreak in Delhi", (JAPI, 1998; 46 (3) : 273-76) where he shared our view and laid stress that platelet transfusion is rarely required in managing patients of dengue fever with minor haemorrhages. Since the incidence of dengue virus infection is rising gradually, it is mandatory on our part that we must learn to differentiate between dengue fever (with or without haemorrhage) and dengue haemorrhagic fever (DHF). Largely, these manifestations are age/immune status dependent.[1] Dengue fever, by and large, runs a benign course with minor haemorrhagic phenomenon like epistaxis, gingival bleeding and GI tract haemorrhage. Platelet counts are usually normal. On the other hand, DHF is a medical emergency which manifests as fever, haemorrhagic diathesis and varying degree of circulatory disturbances sometimes leading to more serious complications like shock, intracranial haemorrhage etc. Thrombocytopenia and haemoconcentration are consistent findings. The outbreak which we had experienced was of dengue fever with haemorrhage and we could manage our patients successfully by simple supportive measures. Disseminated intravascular coagulation (DIC) is a complication of DHF. Coagulation profile was studied in only a few patients in our study where we suspected DIC on clinical ground i.e. bleeding from multiple sites. Thrombocytopenia, increased fibrin and fibrinogen degrada-