

Abstracts from the Journals of the East

Pages with reference to book, From 163 To 164

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Effect of Smoking on Serum Selenium, Zinc and Cholesterol Khurshid, r., Sbcikh, A.S., Akbari, M.Z.A., Ash rat, M. Pak.J.Med.Res., 1995;34:88-90.

The effect of smoking on serum selenium, zinc and cholesterol was studied in 50 heavy smokers, smoking 20 cigarettes daily for five years. A similar estimation was made on 50 non-smokers. The subjects studied were non-diabetic and normotensive. All were examined physically. A fasting blood sample of 5 ml was collected from the ante-cubital vein, serum was separated and stored in metal free plastic tubes for estimation of glucose, cholesterol, selenium and zinc.

The results showed an insignificant difference in the mean cholesterol levels of the two groups. The serum selenium levels were much higher (34.2ug/L) in the nonsmokers compared to the smokers (18.18ug/L). The mean serum zinc of the non-smoker group was again significantly raised (103 . 58ug/100 ml) than the smokers (84. 14ug/100 ml). The values of serum selenium and zinc were found to be very much lower in the heavy smokers group. Zinc and selenium are trace metals playing a protective role in coronary heart disease. After absorption they accumulate in body tissues, mainly in liver and kidneys. These essential trace metals could easily be displaced by toxic trace metals like lead and cadmium. A pack of 20 cigarettes contains 30mg of cadmium of which 70% passes in smoke. This extra cadmium accumulates in the liver, lungs and kidneys. It displaces the zinc and selenium and accounts for their low levels in smokers.

Selenium deficiency causes cell damage leading to a raw vessel lining, plaque formation and enhanced risk for a heart attack. Cigarette smoking thus becomes an important risk factor for coronary heart disease.

Significance of Isolated Gastric Varices among Chinese Patients. Tong, K.J., Chao, Y., Wang, S.S., Hwang, J.I., Lee, S.H. Chin.Med.J. (Taipei), 1995;56: 166-172,

An extensive review of the endoscopic records at the Veterans General Hospital, Taipei, from 1984 to 1993, revealed 36 cases of isolated gastric varices. Gastric neoplasms had been excluded. Also gastric varices occurring along with oesophageal varices or secondary to sclerotherapy were also not included. Medical records were analysed in detail to determine the etiology.

The location of the gastric varices was classified as fundal found in 30 cases and junctional, located below the gastroesophageal junction in 6 cases. The morphology showed prominent mucosal and tortuous elevations of mucosa in 12 patients, nodular swellings in 19 and tumour like mass in 5 individuals. Of the 36 patients, 26 had liver cirrhosis. The diagnosis was made in 16 by liver biopsy and in 10 by clinical, radiological and laboratory findings. HbsAg was positive in 14 patients, anti HCV positive in 6, both positive in one, alcoholic in 2 and undetermined in 2 individuals. None of the patients had evidence of pancreatic disease.

Seven of the cases were associated with pancreatic disease. Chronic pancreatitis was found in 4 and 3 had pancreatic cancer. They all had splenomegaly, gastric varices and splenic vein occlusion. None had liver cirrhosis. Of the three remaining cases of the total, one was diagnosed as myelofibrosis by bone marrow biopsy and the other could not have a confirmed diagnosis.

Active massive bleeding was the presenting symptom in 8 patients (7 with liver cirrhosis and one with pancreatic cancer). Three of these cases died without any intervention. Three of the 5 subjects undergoing splenectomy and fundectomy also died. Two patients remained well to follow-up with no evidence of gastric varices.

Isolated gastric varices is a rare occurrence manifesting usually as a sign of portal hypertension. Endoscopic diagnosis may present difficulties whereas computed tomography usually provides a

confirmed diagnosis. Angiography gives definite diagnosis too. The etiology is based on obstruction to the blood supply of the stomach. Liver cirrhosis or pancreatic cancers can present as bleeding from these dilated vessels. The two pathologies should first be considered in the differential diagnosis.

Skull Tuberculosis - A Case Report. karim, KE., Hossain, A., Haq, S.A., Barua, KK, Mustafa, M.R.. Bangladesh Med.J., 1993;22:45- 46.

The case of a 10 years old boy with skull tuberculosis is presented. He was brought in with complaints of fever and painful swellings on the scalp, left elbow and left heel. All the symptoms were noted after an injury and except for the scalp nodules, disappeared after a course of antibiotics. The fever alongwith fluctuant swellings on the scalp, elbow and heel reappeared after sustaining another trauma. On examination the child was pale, febrile and malnourished. The cervical lymph nodes were enlarged but not matted. Blood examination showed a total white cell count to be 12×10^8 /cmm and an ESR of 150mm in the first hour. All the other blood chemistry was in the normal range. Radiological survey revealed patchy destruction of the entire vault of the skull and sphenoid bone, fracture in the right frontal bone and sutural diastasis. The upper end of the ulna showed bone destruction and periostitis and absorption of trabecules in the upper end of the radius. Localised bone destruction was found in the left calcaneum. A skull biopsy was performed which confirmed the diagnosis of bone tuberculosis. Anti-tubercular therapy with rifampicin, isoniazid and ethambutol was initiated and the fever subsided in two weeks whereas the swellings disappeared in 6 months.

Tuberculosis of the flat bones is rarely encountered. Tuberculous osteitis of the skull usually occurs in the younger age group. It may be the only bony lesion or it may be a part of disseminated skeletal tuberculosis. Trauma is a recognised predisposing factor. It is thus important to consider tuberculosis if there is a persistent swelling of the skull. — -

Use of Cefixime in Treatment of Enteric Fever in Children. Ali, S.M.I. Specialist, Pak.J.Med.Sci., 1996;12:185-189.

A prospective study on 50 consecutive children upto 15 years age (24 males and 26 females) with uncomplicated salmonella infection was conducted to evaluate the usefulness of cefixime in these cases. The study also highlighted the increasing growing resistance in cases of enteric fever. All the patients were febrile and 25 presented between 5 and 10 days of the illness. None of the patients had been immunized. Complicated cases with ileus, perforation, shock and disseminated intravascular coagulopathy were excluded. Investigations included blood picture with malarial parasite, blood culture with sensitivity, Widal test, serum creatinine and urine analysis. These were repeated at the end of the therapy too. Blood culture was performed aerobically and anaerobically. Sub-cultures were done subsequently.

Therapy with cefixime was started with 10mg/kg as an oral dose at 12 hourly intervals for either 14 days or 7 days after defervescence, whichever was earlier. Patients not showing a positive response till 10 days were changed to other antibiotics. Temperature charts were maintained on 4 hourly scales and out-patient visits were made on day 1, 3, and then every 4th day till cure.

A good clinical response with cefixime was observed in 39 cases. Two cases were cured with amoxicillin. There were 9 resistant cases of which two again responded well to cefixime. Relapse occurred in 3 cases on 14th, 17th and 23rd day. They responded well to cefixime. Side effects of cefixime were noted as diarrhoea in 2 patients, vomiting in 2 and nausea in one case. Resistance of salmonella was observed against chloramphenicol 54%, co-trimoxazole 56% and amoxicillin 50%. Cefixime showed a good sensitivity though it was less than the injectable third generation cephalosporins. Cefixime proved to be economical and safe drug providing a defervescence period of 6.7 days in multi- drug resistant, uncomplicated salmonella infection. Severe cases of enteric fever with life threatening complications should be treated promptly with parenteral third generation cephalosporins.