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Aga Khan University Hospital, Karachi

Newsletter of Departments of Pathology and Microbiology, and Radiology

LABRAD

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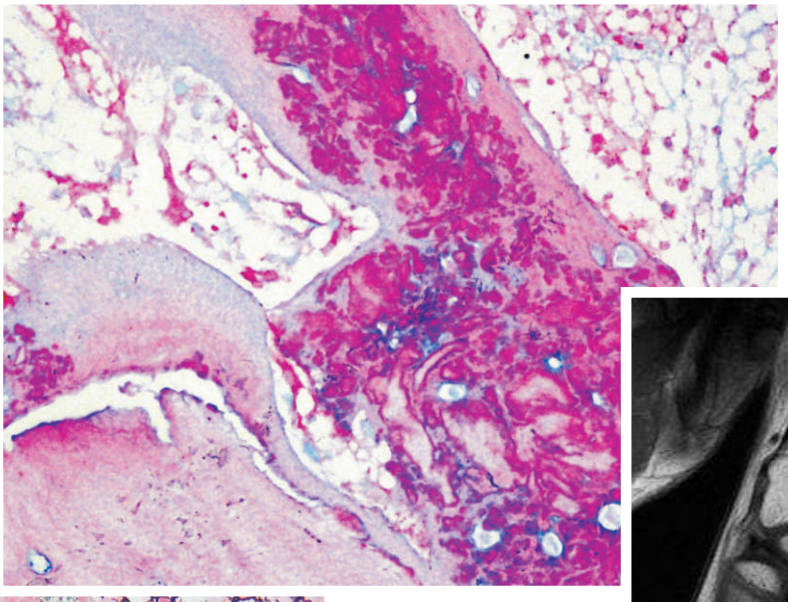

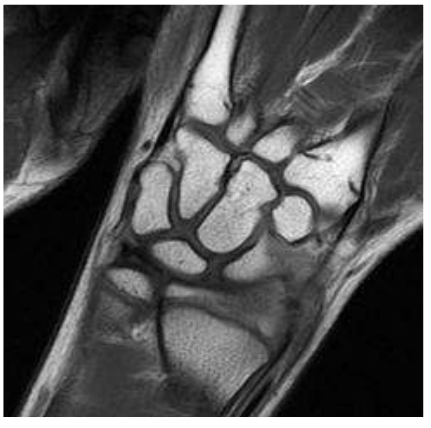

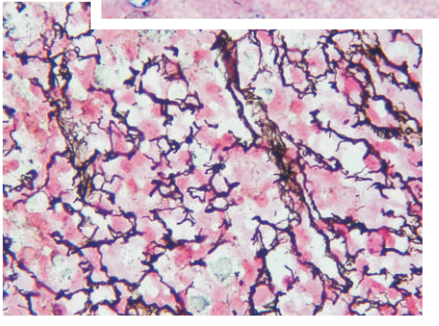

In this issue

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N-Telopeptide (NTx) of Type I Collagen: A Biochemical Tool for Assessing Bone Turnover

Drs Aysha Habib Khan, Farhan Javed Dar
Chemical Pathology

Bone undergoes continuous remodeling as it is a dynamic structure. Bone remodeling can be assessed by the use of bone turnover markers. Depending upon the state of bone they can be categorized into bone formation and bone resorption markers. Markers of bone resorption seemed to be stronger predictors of future bone loss than markers of bone formation. Out of these, a bone resorption marker, cross-linked N-Telopeptides of type I collagen (NTx) provides a valuable tool for assessing Bone Turnover.

Approximately 90% of the organic matrix of mammalian bone consists of type I collagen that is cross-linked at the N-terminal and C-terminal ends. This highly cross-linked structure provides the basic fabric and tensile strength to the bone tissue. NTx represents the N-terminal cross link and is specific to bone and is found in serum and urine as a stable end product of bone degradation. An elevated NTx level correlates with high bone turnover.

Experimental and clinical studies have demonstrated that markers of bone turnover may reflect changes in bone metabolism induced by oophorectomy, hyperparathyroidism, Paget's disease, physical exercise, immobilization, alcoholism, smoking, vitamin D deficiency, chronic inflammatory bowel disease, chronic starvation, thyroid disorders as well as pharmacological effects of glucocorticoids, androgens, gonadotropin releasing hormone agonists, warfarin, growth hormone or insulin like growth factors. However, in most of these conditions there role has not been rigorously examined.

Using biochemical markers like NTx, it may be possible to identify those subjects who lose bone rapidly and are likely to develop osteoporosis and future fractures. However, its main role is in monitoring the therapeutic response. Although DEXA scan is still the best way to judge the changes in bone mineral density in osteoporosis, the bone mass changes takes 1-2 years to become apparent on DEXA scan. With NTx patients can be monitored as early as 3 to 6 months after starting therapy for osteoporosis. A baseline NTx level should be measured in all patients before beginning anti-resorptive therapy. If treatment is stopped, NTx levels tend to return slowly to the pre-treatment baseline, which makes it a useful monitoring tool for poor compliance. For monitoring purpose it is advised to collect sequential samples at approximately the same time of day.

Sources of variability in bone resorption markers include circadian rhythm, fluctuations in renal function, and dietary calcium intake. Serum based markers of bone turnover tend to show less variability as compared with urine based markers. Measurement of NTx in serum offer bone resorption testing with decreased intrasubject variability.

NTx levels are now measured in the Clinical Laboratory of Aga Khan University Hospital using serum by ELISA technique.

Automated Blood Grouping

Dr Nausheen Kamran
Resident, Haematology

Blood banks and transfusion services are the last areas of the clinical laboratory to move to automation. Chemistry, Haematology and Immunology have been using automation for many years, but blood services have been hampered by the complexity of testing and subjectivity of the test interpretation.

In recent years, new pressure has been applied to this area of laboratory. Personnel shortages, turnaround time and the need for cost containment produced by increased managed care have provided the incentive for blood services to seek automation.

By using walk-away automation, lab personnel are able to perform multiple tasks simultaneously.

Automated equipment also provides the level of quality assurance required by new regulatory standards. Standardized techniques reduce testing errors.

In 1985 the gel test was developed by Dr Yves Lapierre of Lyon, France. Various media including gelatin, acrylamide gel and glass beads were investigated in an attempt to trap agglutinates during a standardized sedimentation or centrifugation step.

The gel test, which is performed in a specially designed micro tube, is based on the controlled centrifugation of RBCs through a dextranacrylamide gel that contains predispensed reagents. Each micro tube is composed of an upper reaction chamber that is wider than the tube itself and a long, narrow portion referred to as the column. In the gel test plastic cards with micro tubes are used instead of test tubes. The gel particles are porous and they serve as a reaction medium and filter. Large agglutinates are trapped at the top of the gel and are not allowed to travel through the gel during the centrifugation of the card. The unagglutinated RBCs travel unimpeded through the length of the micro tube forming a pellet at the bottom following centrifugation.

Blood samples should be drawn in citrate, EDTA or CPD-A anticoagulant. A gel card is approximately 5x7 centimeters and consists of six micro tubes (Figure 1). The ABO blood grouping cards contains gel that include blood group specific reagent, e.g., anti-A, anti- B, and anti-A, B for forward grouping. Microtubes with buffered gel are used for ABO reverse grouping. Pipette 25 µl of the patient's red cell suspension to the first 3 micro tubes of the ID card. Centrifuge the ID cards for minutes in the centrifuge.

Read and record the result. Agglutination reaction in the gel is graded from 1+ to 4+ (including mixed field). The reactions are read as:

A 4 + reaction → solid band of agglutinated RBCs at the top of the gel.
A 3 + reaction → agglutinated RBCs near the top of the gel column, with few agglutinates staggered below thicker band.
A 2+ reaction → agglutinated RBCs dispersed throughout the gel column.
A 1+ reaction → agglutinated RBCs predominantly in the lower half of the gel column.
Negative reaction → RBCs form a well delineated pellet at the bottom of the micro tube.

Known positive and negative samples should be included as controls.

Figure 1: Diamed ID Gel Station

Advantages of automated red cell typing:

- Standardization: there is no tube shaking to re-suspend the RBC button
- It provides stable end point of the agglutination reaction
- Results can be interpreted even after hours
- Results can be photographed
- Decreased sample volume required for testing
- One card can be used for two samples

Disadvantages:

- Purchase of special incubators and centrifuges to accommodate micro tube cards
- Special pipettes are required

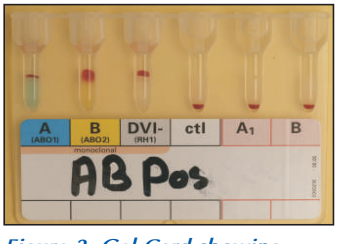

Figure 2: Gel Card showing AB positive blood group

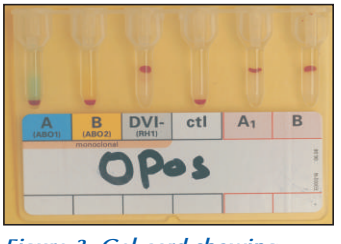

Figure 3: Gel card showing O positive blood group

Leukocyte Alkaline Phosphatase (LAP Score)

Ms Mashhooda Rasool Hashmi
Senior Technologist, Haematology

Alkaline phosphatase activity is found predominantly in mature neutrophils, with some activity in metamyelocytes. Normally about 2% of the mature granulocytes give a positive alkaline phosphatase reaction. Although demonstrated as a granular reaction product in the cytoplasm, enzyme activity is associated with a poorly characterized intra cytoplasmic membranous component distinct from primary and secondary granules. Other leukocytes are generally negative, but rare cases of lymphoid malignancies show cytochemical demonstrable activity. Bone marrow macrophages are positive.

In 1929, Kay first suggested the presence of alkaline phosphatase in leukocytes. However, not until many years later did Kaplow introduce a practical staining method for demonstrating the leukocyte enzyme.

The substrate, naphthol AS-B1 phosphate is hydrolysed to phosphate and aryl naphtholamide by alkaline phosphatase. The aryl naphtholamide is coupled to the diazonium salt, fast blue RR forming an insoluble dye.

Blood films prepared from capillary blood or heparinized venous blood may be used. Blood smears should be stained for enzyme activity within eight hours after preparation. It is preferable to stain the smears immediately, however, fixed smears may be held at freezing temperature for two to three weeks with loss of only approximately 10% of activity. Films should be dried one hour prior to fixation and three hours post-fixation before freezing.

The reaction product is blue/brown and granular (Figure 1). The intensity of reaction product in neutrophils varies from negative to strongly positive, with coarse granules filling the cytoplasm and overlying the nucleus. An overall score is obtained by assessing the staining intensity in 100 consecutive neutrophils, with each neutrophil scored on a scale of 1 to 4 as follows:

0 = negative, no granules
1 = occasional granules scattered in the cytoplasm
2 = moderate number of granules
3 = numerous granules
4 = heavy positivity with numerous coarse granules crowding the cytoplasm, frequently overlying the nucleus

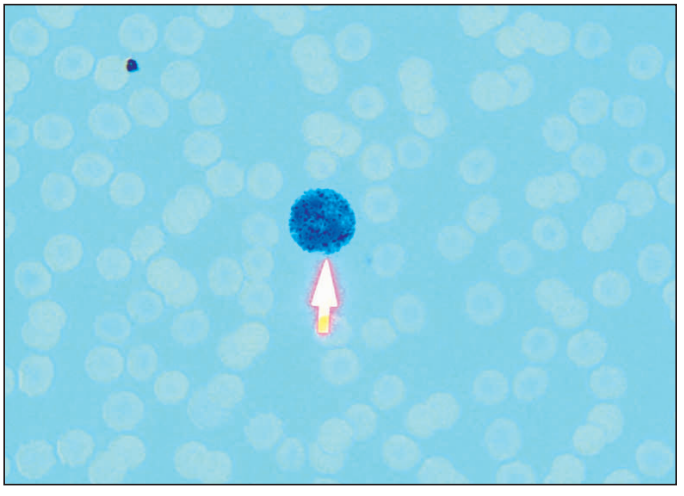

Figure 1: Neutrophil with numerous blue/brown granules.

The normal range of LAP score is 32 to 182. In normal individuals, it is rare to find neutrophils with scores of 3, and scores of 4 should not be present. There is some physiological variation in LAP score.

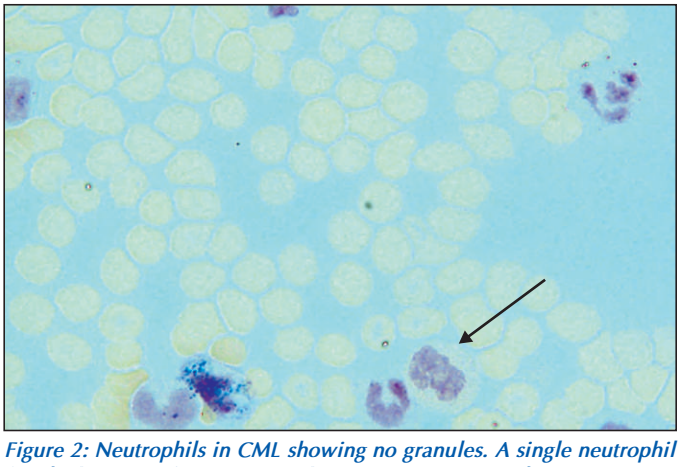

Figure 2: Neutrophils in CML showing no granules. A single neutrophil (marked by arrow) contains moderate number of granules.

Newborn babies, children and pregnant women have high scores, and premenopausal women have, on average, scores one third higher than men. In pathological states, the most significant diagnostic use of LAP score is in chronic myeloid leukemia. In the chronic phase of disease, the score is almost invariably low; usually zero (Figure 2). In myeloid blast transformation or accelerated phase, the score rises. Transient rises may occur with intercurrent infection. Raised LAP score can also be encountered in polycythemia rubra vera, leukamoid reaction and Hodgkin's disease.

Low scores are also found in paroxysmal nocturnal hemoglobinuria and hereditary hypophosphatasia.

Pathology Quiz

Dr Lena Jafri
Resident, Chemical Pathology

A 17-year old female with short stature but no dysmorphic features was referred to AKUH laboratory for growth hormone deficiency evaluation. She came with proper 12 hours of fasting. Two-hour growth hormone insulin test was performed and the results obtained were as follows:

	Glucose (mg/dl)	Growth hormone (ng/ml)
Fasting:	84	19.1
At 30min:	26	12.7
At 45min:	48	14.4
At 60min:	56	16.7
At 90min:	68	22.1
At 120min:	76	59.7

Questions:

1. Are these results normal?
2. Which clinical syndrome did the patient have?
3. Where does the defect lie?
4. What other tests will you order to confirm your diagnosis?
5. What therapy is recommended for such children?

Answers on page 8

Synovial Fluid Analysis in Important Joint Diseases

Dr Raihan Sajid
Assistant Professor, Haematology

Synovial fluid is believed to be produced by dialysis of plasma across the synovial membrane and by secretion of hyaluronate-protein complex by the synovial membrane. Synovial membrane lines joints, bursae, synovial tendon sheaths, but not the articular cartilages and menisci. The function of synovial fluid is lubrication and nourishment of the articular cartilage.

Inflammation of the synovial membrane will cause leakiness of synovial membrane and influx of WBC and exudation of proteins into synovial fluid. Any joint can be aspirated, however joint aspiration should be performed by an experienced operator under sterilized conditions.

Synovial fluid analysis comprises of appearance, viscosity, protein, glucose, and TLC, DLC and crystals identification under polarized light. Additional tests include lactate, uric acid, pH measurements and immunologic studies including rheumatoid factor, ANA antibodies and complement measurements. Gram stain and cultures should always be sent if septic arthritis is suspected. In rheumatoid arthritis in about 95% of patient's cells known as RA cells are present which contain small, dark cytoplasmic granules from 0.5 to 2 μ diameters within 5-100% of neutrophils.

Normal synovial fluid is crystal clear and pale yellow in colour. It has high viscosity with WBC less than 200/cumm, protein less than 2 gm/dl and glucose nearly equal to blood levels. Milky fluid may occur with tuberculosis, chronic rheumatoid arthritis and SLE and calcium hydroxyapatite arthropathy. In rheumatoid arthritis the appearance of the synovial fluid shall be turbid with low viscosity, a high WBC count of more than 20,000/cumm with 70% neutrophils and RA cells are usually present.

In septic arthritis, the appearance of the synovial

fluid will be turbid to purulent with low viscosity, high WBC count of 90,000/cumm with more than 90% neutrophils. Gram stain and culture is positive in about 50% of the patients with septic arthritis. In osteoarthritis the appearance of synovial fluid is usually clear yellow, leukocyte count of 700/cumm with 15% neutrophils and collagen fibrils and/or cartilage fragments are usually present.

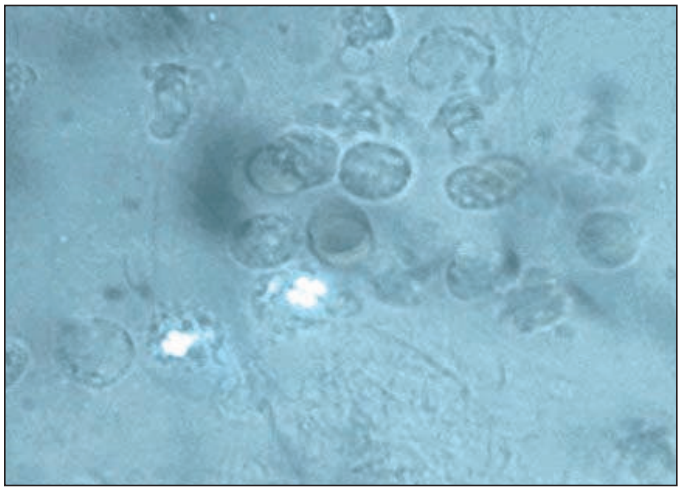

Figure 1: Crystals under polarized light.

Figure 1 shows crystals under polarized light. Table 1 shows categories of synovial fluid based on clinical and laboratory findings.

Table 1: Categories of synovial fluid based upon clinical and laboratory findings.

Categories of Synovia Fluid Based Upon Clinical and Laboratory Finding

Measure	Normal	Noninflammatory	Inflammatory	Septic	Hemorrhagic
Volume, mL (knee)	<3.5	Often >3.5	Often >3.5	Often >3.5	Usually >3.5
Clarity	Transparent	Transparent	Translucent-opaque	Opaque	Bloody
Color	Clear	Yellow	Yellow to Opalescent	Yellow to green	Red
Viscosity	High	High	Low	Variable	Variable
WBC, per mm3	<200	200-2,000	2,000-10,000	>100,000↑	200-2,000
PMNs, percent	<25	<25	≥50	>75	50-75
Culture	Negative	Negative	Negative	Often positive	Negative
Total protein, g/dL	1-2	1-3	3-5	3-5	4-6
LDH (compared to levels in blood)	Very low	Very low	High	Variable	Similar
Glucose, mg/dL	Nearly equal to blood	Nearly equal to blood	>25, lower than blood	>25,much lower than blood	Nearly equal to blood

↑Lower with infections caused by partially treated or low virulence organisms

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1. Clinical Diagnosis and Management by Laboratory Methods, 17th edition. John Bernard Henry. Pages 475-83.

Intraductal Papillary Mucinous Tumor (IPMT) of the Pancreas: A Diagnostic Challenge

Drs Zishan Haider*, Ishtiaq Chishti*, Farhan Ahmed**, Dawar Khan*; *Assistant Professor, ** Senior Instructor, Radiology

Primary cystic pancreatic neoplasms are rare tumors, with an approximate prevalence of 10% of cystic pancreatic lesions. The natural history of intra-ductal papillary mucinous tumours of the pancreas (IPMTs) is unknown. In clinical practice the signs and symptoms are non-specific. Diagnosis is usually not easy without using new imaging modalities such as CT or MRI. Considering number of cystic lesions of pancreas, diagnosis of pancreatic IPMT is usually challenging as it is relatively newer diagnostic entity. Multidetector CT is helpful in the preoperative differentiation of malignant and benign pancreatic IPMT. Definitive diagnosis is often possible when the lesions show typical radiologic appearances (Figures 1 and 2).

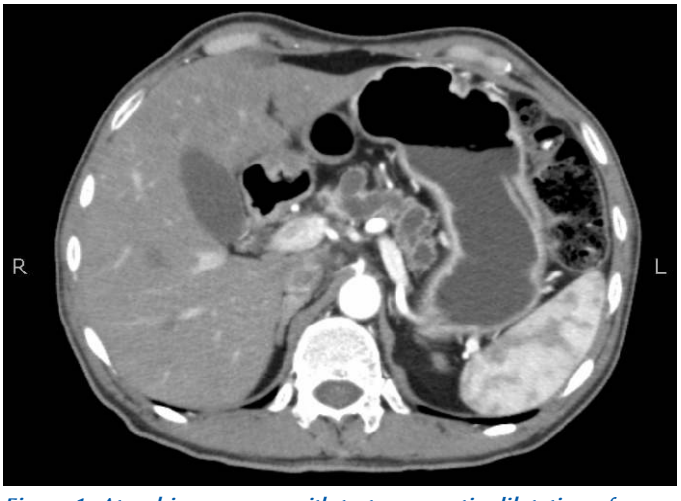

Figure 1: Atrophic pancreas with tortuous cystic dilatation of pancreatic duct on axial contrast enhanced CT scan.

IPMT of the pancreas can be divided into three clinically distinct sub-types: main duct type, branch duct type and mixed type. IPMT is characterized by diffuse dilatation of the main pancreatic duct and/or side branches with inner defects related to mucin or tumor, or mucin extrusion from a patent ampulla. IPMT has a low potential for malignancy, with a low growth rate, a low rate of metastatic spread and postsurgical recurrence. Over the last few years,

major advances have been made in the diagnostic and therapeutic management of this tumor.

The presence of a dilated main pancreatic duct, mural nodules, thickened wall and peripancreatic haziness can be predictive signs of malignancy. Although it has been reported that the branch duct type IPMT is less invasive than the main duct type IPMT, some times branch duct type IPMT may have poor prognosis.

The use of CT scan with high-resolution MPR images significantly improves diagnostic performance for demonstrating connection between pancreatic cystic lesions and the main pancreatic duct, which is useful for the diagnosis of branch duct-type IPMT. If prompt diagnosis is established then it can improve patient care and optimal surgical treatment can be planned.

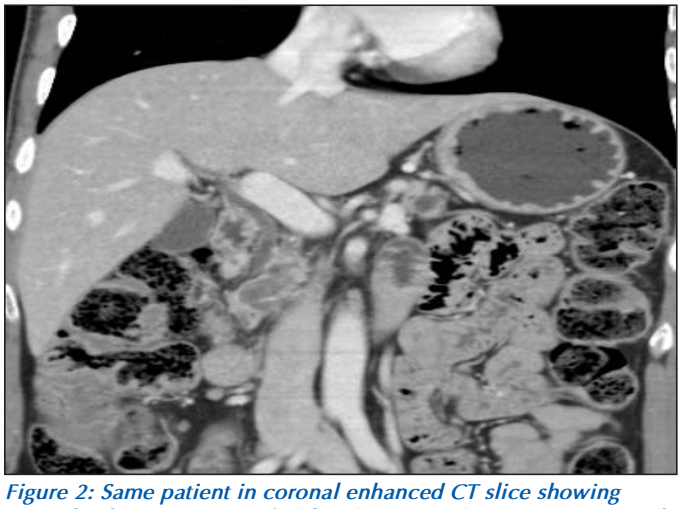

Figure 2: Same patient in coronal enhanced CT slice showing normal caliber of CBD with dilated tortuous duct containing subtle high density material suggestive of IPMT.

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2. Lim JH, Lee G, Oh YL. Radiologic spectrum of intraductal papillary mucinous tumor of the pancreas. Radio-Graphics 2001; 21(2):323337.
3. De Lima JE Jr, Javitt MC, Mathur SC. Mucinouscystic neoplasm of the pancreas. Radio-Graphics 1999; 19(3):807811.

Hepatitis Delta Virus (HDV)

Ms Hina Noureen,
Assistant Technologist, Molecular Pathology

Hepatitis D virus (HDV, also called the delta virus) is a defective pathogen and considered to be a sub-viral satellite because it can propagate only in the presence of hepatitis B virus. Transmission of HDV can occur either via simultaneous infection with HBV (co-infection) or via infection of an individual previously infected with HBV (super infection). The delta antigen was first detected by an Italian physician Mario Tizzetto in hepatocytye of patients with chronic type B hepatitis.

Genetic Structure of HDV

This is a 36 nm virus with a 1.7 kb circular negative strand RNA genome which is folded as a rod like structure. It has approximately 1,700 nucleotides, the smallest genome of all known human pathogens. The HDV virion is composed of an outer lipoprotein envelope made of the surface antigen of HBV (HBsAg) and an inner ribo-nucleoprotein structure in which the HDV genome reside. The nucleo-capsid consists of a smaller HD Ag-S and a large HD Ag-L peptide which play different biological roles in the viral life cycle. HD Ag-S is produced in the early stages of an infection and is required for viral replication. HD Ag-L, is in contrast, is produced during the later stages of an infection, acts as an inhibitor of viral replication and is required for assembly of viral particle.

Symptoms

Co-infection (acute):
Patients infected with HBV and HDV simultaneously suffers from fatigue, lethargy, anorexia, nausea, headache, jaundice. Incubation period is 3 to 7 weeks.

Super Infection

Post-infected with HBV and later infected with HDV suffers from jaundice, hepatic encephalopathy, changes in personality, coagulopathy, disturbance in sleep, and confusion, and acute massive destruction of large portion of liver.

Chronic Infection
Similar symptoms to above but severe ongoing liver inflammation, cirrhosis leads to liver failure, death from bleeding, hepatic infection and kidney failure.

Diagnosis of HDV
Diagnosis is done primarily by serological assays and PCR. Due to the dependence of HDV on HBV, the presence of HBsAg is necessary for the diagnosis of HDV infection. The additional presence of IgM antibody to hepatitis B core antigen (IgM anti-HBc) is necessary of diagnosis of acute HBV/HDV coinfection. High levels of IgM and IgG anti-HD are associated with progression to chronicity.

HDV RNA is extracted using High Pure Viral RNA kit (Roche, USA) then cDNA is synthesized and amplified by RT- PCR. The PCR product is run on the gel electrophoresis which gives picture of bands indicating (L) ladder, i.e. reference dye, positive control, negative control (not shown in the figure below), and positive patient's (Pt) sample.

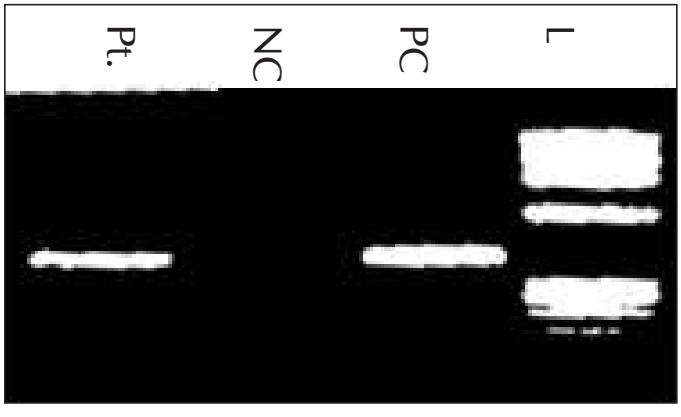

Figure 1: Different stages of the epilome enzyne

Transmission
Blood borne, sexual, percutaneous, permucosal and rarely perinatal.

Prevention
Abstinence from alcohol, Percutaneous of personal contact with HBV infected Patients is recommended. The vaccine against hepatitis B also prevents delta hepatitis.

Treatment
There is no effective antiviral therapy available for treatment of acute or chronic type D hepatitis. Liver transplantation has been helpful for treating fulminant acute and end- stage chronic hepatitis.

Answers to Pathology Quiz

1. No. Growth hormone levels are abnormally high.

2. Laron's Syndrome. Some children with idiopathic short stature show very high levels of growth hormone during growth hormone insulin test. It is not an uncommon condition. It is a familial disorder with an autosomal recessive form of inheritance. Laron and his colleagues were the first to describe short stature with characteristic features of isolated growth hormone deficiency but with elevated growth hormone levels. It is also known as growth hormone insensitivity syndrome.

3. The fault lies in the inability of the growth hormone target cells to respond to growth hormone due to defective growth hormone receptor on cell surface.

4. Serum assays for insulin-like growth factor -1(IGF-1) and insulin growth factor binding protein -3 are now commercially available. Patients with Laron's syndrome have normal or elevated circulating growth hormone but low IGF-1 and low IGFBP-1. They should be included in complete evaluation of such patients but unfortunately these tests are not available in our laboratory at present.

5. Effects of recombinant human IGF-1 on linear growth of children with receptor mutations have proven beneficial and it has been recommended as a long term therapy for patients with Laron's Syndrome.

Thyroid Autoimmune Diseases and Thyroid Peroxidase (TPO) Antibody

Dr Sahar Iqbal
Resident, Chemical Pathology

The autoimmune diseases are the frequent causes of thyroid disorders. Important targets for auto antibodies are thyroid receptors (TSHR), thyroglobulin (Tg) and thyroid peroxidase (TPO). In up to 90% of autoimmune thyroid disease thyroid peroxidase antibody (TPO-Ab) are seen, whereas anti thyroglobulin(Tg-Ab) is found in less than 20% of the cases. TSH receptors antibodies (TSHR-Ab) are specific for Graves' disease, in contrast to Tg-Ab and TPO-Ab. Almost all patients with Graves' hyperthyroidism have detectable TSHR-Ab when measured by sensitive assays.

TPO is a glycoprotein present as a dimer on the apical surface of thyroid follicular cells as well as in cytoplasm. It catalyses the thyroid hormone synthesis and thought to represent the cell surface antigen involved in cell mediated cytotoxicity. TPO-Ab inhibits TPO enzyme activity, resulting in decrease synthesis of thyroid hormones.

Measurement of TPO-Ab is important in the diagnosis of autoimmune thyroid diseases especially Hashimoto's thyroiditis. Prevalence of TPO-Ab is also found to be high in other autoimmune disorders and many studies have shown the association of presence of TPO-Ab with other diseases including pernicious anaemia, diabetes mellitus, poly cystic ovarian syndrome, urticaria and arthritis.

In relation to pregnancy the presence of thyroid autoimmunity, appears to be a determining factor in future pregnancy loss, not only in hypo-and hyperthyroid women but also in euthyroid ones. In the early 1990s it was discovered that unselected euthyroid women who present with thyroid antibodies to TPO and Tg in the early trimesters of pregnancy have a two-to-four-fold increase in their miscarriage rates.

The prevalence of TPO-Ab among pregnant women is 10-12% in different studies.

Many other studies showed high prevalence of TPO-Ab positive results in pregnant women and its association with adverse outcomes including miscarriage or abortion, preterm delivery, low birth weight, postpartum thyroiditis. Hypothyroidism during pregnancy may lead to the impaired neuropsychological development of the child.

In our laboratory both TPO-Ab and Tg-Ab are performed by Microparticle enzyme immunoassay (MEIA) for detection of thyroid autoimmune disease.

Therapeutic Monitoring of Tacrolimus

Ms Noureen Niaz Ali
Staff Technologist, Chemical Pathology

Tacrolimus is an immunosuppressive drug which was discovered during routine screening and fermentation of streptomyces tsukubaensis fungi. It is also known as FK 506. After its discovery, it was soon recognized that tacrolimus effectively suppresses and prevents organ graft rejection by inhibiting the early events in T cells activation and proliferation by suppression of IL-2 and other cytokines transcription.

Therapeutic monitoring of blood level of tacrolimus is required for effective and safe use because its therapeutic window is narrow and its toxic effect correlates with whole blood levels. In addition, it produces renal vasoconstriction and hypertension. It is also diabetogenic and is capable of causing central nervous system toxicity.

Several methods have been developed to measure tacrolimus (FK506). In AKUH laboratory, we use IMX (instrument) which works on microparticle enzyme immunoassay technology (MEIA) requiring monoclonal antibody. Whole blood is the preferred sample for the measurement of tacrolimus levels. EDTA is the preferred anticoagulant. False positive results can occur when blood sample of patient clots in the tube.

Meeting Report: 32nd Annual Conference of Pakistan Association of Pathologists

Reported by Dr Lena Jafri
Resident, Chemical Pathology

The 32nd annual conference of Pakistan Association of Pathologists (PAP) was held from October 24 to 26, 2008 at Abbottabad, the City of Pines. The conference was hosted by Ayub Medical College away from the hustle and bustle of city life. Pathologists and scientists from all over Pakistan were invited for a positive and interactive learning forum.

The inaugural session was marked by a welcome address by Professor Syed Humayun Shah, Chief Patron PAP Conference on October 24. This was followed by a plenary lecture by Dr Lubna Naseem from the Pakistan Institute of Medical Sciences, Islamabad on 'Blood Transfusion Practices: Past, Present and Future'. The key aspect of her discussion was better and safe transfusion practices. It was an informative and insightful talk highlighting the risk of transfusion-transmissible infections as a result of poor blood donor recruitment and selection practices and the use of untested units of blood.

The conference had two half-day free papers interactive sessions in different disciplines of Pathology. Participants shared their researches with the audience. It provided an opportunity for the participants to give presentations to their peers in their areas of expertise. Many questions were elicited which made for great discussion among all. The evening grand finale culminated with a Banquet dinner followed by a vibrant cultural evening.

Day 2 commenced with the 'State of the Art Lecture' delivered by renowned Professor Anwar ul Haque from Pakistan Institute of Medical Sciences, Islamabad. His prolific talk was on 'Giant Cell Tumor of Bone: Is it a Neoplasm?'

An inspirational Plenary Session by Dr Farooq Ghani, Associate Professor and Consultant Chemical Pathologist Aga Khan University, Karachi was delivered on 'Cardiac Markers Update'. The utility of Brain Natriuretic Peptide (BNP), myeloperoxidase and myoglobin as evolving cardiac markers were emphasized. This was followed by free paper session. Numerable scientific posters were displayed which portrayed research advances in the field of pathology.

AKU delegates at 32nd PAP Conference held at Abbottabad.

Dr Farooq Ghani receiving a shield from the Chief Guest following his Plenary Lecture at PAP 2008.

Exhibition of latest equipments with the most modern technologies was arranged for demonstration. Professional attention was offered on most of the booths for interested participants. The evening was marked by the closing ceremony addressed by Professor Jamil A. Mirza, President PAP. Post-conference tour to scenic Nathiagali and Ayubia was offered to the participants on the third day of the conference.

The attendees were remarkably diverse, with high level of enthusiasm. Whatever their field of expertise, attendees left with valuable information. The goal of providing a forum of information exchange among health professionals was successfully achieved. It was an inspirational weekend!

From left to right:
Drs Farhan Dar and Lena Jafri, Ms Sadia Tabassum, Dr Raihan.

AKU Clinical Laboratory Continuing Medical Education (CME) Seminar in Bahawalpur

Reported by Dr Romena Qazi
Assistant Professor, Molecular Biology

The CME Seminar was held at Quaid-e-Azam Medical College (QMC), Bahawalpur on October 14, 2008 and covered a broad range of topics on infectious and genetic diseases. It was well attended by health care professionals as well as students, staff and faculty members from local area hospitals and academic institutions. Professor Dr Mazhar-ul-Haq Attiq, Principal of the College, presided over the session.

Dr Usman Shaikh receiving his shield from the Chief Guest.

In his opening statement, Dr Asghar Javed, Staff Pathologist from Multan shared the mission of AKUH Clinical Laboratory of conducting such seminars and underscoring the role of AKUH pathologists in educating as well as providing high-quality diagnostic services for physicians and hospitals nationwide. He invited the Chief Guest for his welcome remarks.

Professor Mazhar-ul-Haq Attiq applauded the effort and initiative of AKU pathologists in terms of disseminating knowledge and sharing their experiences with health care professionals in other parts of the country. He further highlighted the role of these seminars in educating the students and staff

of academic institutions. He appreciated the fact that AKUH clinical laboratory has opened hundreds of collection points throughout the country to help those who do not have direct access to high-quality diagnostic facilities in their respective areas.

Dr Qazi Masroor receiving his shield from the Chief Guest.

After the welcome address Professor Mazhar-ul-Haq Attiq delivered a seminar on Hepatitis-C Virus (HCV) in which he presented epidemiology of HCV in Pakistan with special emphasis on data gathered locally to show its rising burden of disease in Bahawalpur and its vicinity.

Dr Aysha Habib Khan, Assistant Professor Chemical Pathology at AKU provided an overview of the various metabolic bone diseases. She emphasized the important role of vitamin-D in the pathogenesis of two common bone diseases, osteomalacia and osteoporosis. She shared her data gathered on ambulatory care patients and healthy asymptomatic adults from Karachi, showing high prevalence of vitamin-D deficiency.

Dr Qazi Masroor Ali, Associate Professor of Medicine at QMC talked very passionately about the epidemiology, treatment and duration of treatment for hepatitis-B virus (HBV). He presented a video clip of the HBV infectious cycle, and compared the response of different drugs used for treating HBV infected individuals.

Dr Usman Sheikh, Assistant Professor and Consultant Haematologist impressed the audience with a lucid and engaging presentation on Thalessemia. He referred to data obtained by the AKUH clinical laboratory as well as scans to reveal the true picture of thalessemias within the country. He emphasized early diagnosis of thalessemia and counseling of parents.

The last talk was delivered by Dr Romena Qazi, Assistant Professor and Consultant Molecular Pathologist at AKUH. She imparted essential information about the Human Immunodeficiency Virus (HIV) to her audience, in which she highlighted the importance of viral genotype as well as load in patient's treatment and management. She discussed the various commercially available tests for detecting and also emphasized that HIV is an emerging public health problem which can be curtailed through mass education. Dr Asghar Javed closed the meeting with a vote of thanks.

Participants of the CME at Bhawalpur.

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