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The Atypical Lymphocytes

Dr Shabneez Malik and Roohi Noman
Haematology

Atypical lymphocytes are reactive lymphocytes that play an important role in the immune response. It was originally described by Turk as 'Turk Cell' in 1907 in the peripheral blood of a patient with infectious mononucleosis. In 1923, Downey and McKinley, based on the cytoplasmic appearance and the distribution and quantity of chromatin, further classified them into;

- Type 1 Downey Cells (fig. 1): Nucleus is irregular or it may be round or oval (in this case it may be indented). The nuclear chromatin resembles that of a mature lymphocyte. It has a basophilic cytoplasm that may be foamy and vacuoles may be present.

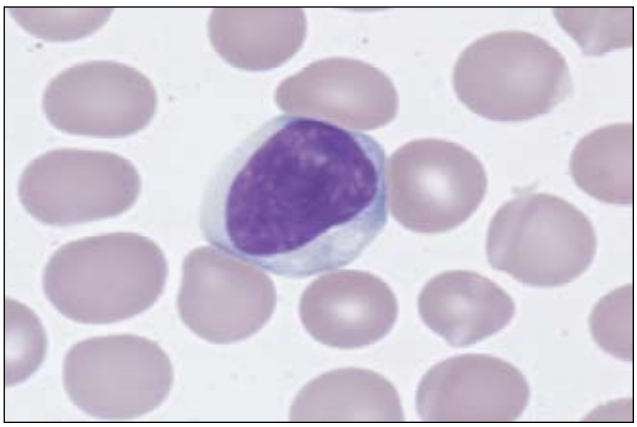

Fig. 1. Type 1 Downey Cell

- Type 2 Downey Cells (fig. 2): Nucleus has coarse chromatin (less than in the type I cell). Cytoplasm has a scalloped appearance (appear to have flowed around adjacent RBCs) and is increased in quantity. It stains light blue around the nucleus but more intense blue at the periphery (basophilic). There may be radial basophilia. Vacuolation is less than in type 1 and azurophilic granules may be seen.

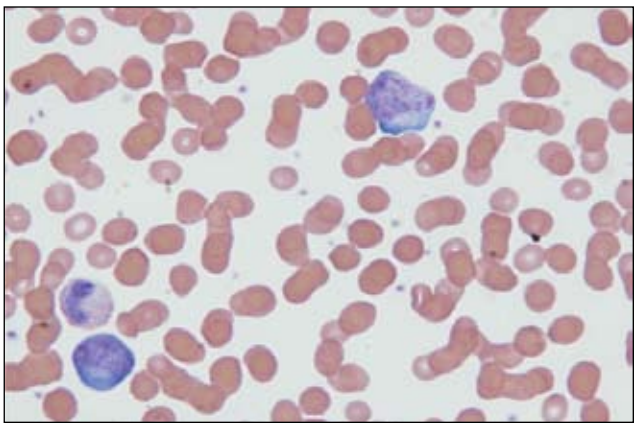

Fig. 2. Type 2 Downey Cell

- Type 3 Downey cells (fig. 3): Nucleus resembles immature lymphocyte and nucleoli (one to four) may be visible. Cytoplasm is highly basophilic and abundant. This cell is larger than types 1 and 2.

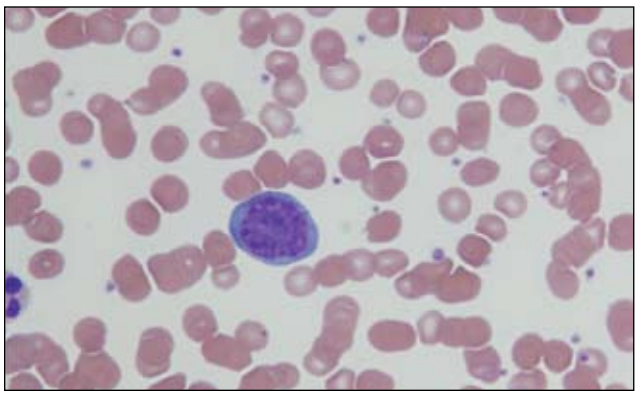

Fig. 3. Type 3 Downey cell

Atypical lymphocytes are present in both viral and bacterial infections. These are usually 10 to 15 µm in diameter. In severe infections, they may develop into plasmacytoid lymphocytes and plasma cells. They are non-malignant and appear as a non-specific response to stress from a variety of stimuli.

Causes of atypical lymphocytosis are listed in Table 1.

Atypical lymphocytes should be morphologically differentiated from the atypical lymphoid cells of lymphoma or malignancy on the basis of size, nuclear and cytoplasmic features. Atypical lymphocytes are 10 to 15 µm in diameter with an abundant, irregular and basophilic cytoplasm. The nucleus is slightly larger with open chromatin. In comparison, atypical lymphoid cells are variable in size depending on the type of malignancy involved. The cytoplasm is agranular, scanty with varying degree of basophilia and the nucleus is irregular lobed, indented or cleaved.

Infections	Infectious Mononucleosis (in which it is frequently ≥30%) Toxoplasmosis Rubella Mumps Tuberculosis Hepatitis A & B
Drug and toxic reactions	Lead poisoning
Hormonal	Addison's disease Thyrotoxicosis
Auto immune disorders	Rheumatoid arthritis Systemic Lupus Erythematosus (SLE) Idiopathic thrombocytopenic purpura
Malignancy	Hodgkin's disease
Idiopathic	Sarcoidosis Guillain-Barre Syndrane Myasthenia gravis
Graft rejection	Renal
Others	Immunisation, radiation

Table 1: causes of atypical lymphocytosis

High Performance Liquid Chromatography (HPLC): An Effective Tool for the Diagnosis of Various Hemoglobinopathies

Mashhooda Rasool Hashmi
Haematology

Cation Exchange high performance liquid chromatography (HPLC) is a process in which a mixture of molecules (such as normal or variant haemoglobins) with a net positive charge is separated into its components by their adsorption onto a negatively-charged stationary phase in a chromatography column, followed by their elution by a mobile phase. The mobile phase is a liquid with an increasing concentration of cations flowing through the column. After elution, these molecules are provisionally identified by their retention time and then quantified spectrophotometrically.

The application of HPLC to the identification of variant haemoglobins depends on the fact that, for each normal or variant haemoglobin, there is a characteristic period of time, referred to as retention time before the haemoglobin appears in the elute. In this manner, a complete separation of almost all kinds of haemoglobins can be achieved, including various overlapping haemoglobins like S and D.

A complete blood count along with a thorough review of peripheral film is essential in the assessment of haemoglobinopathies to reach a correct diagnosis. Variant haemoglobin identification by HPLC is basically a screening test and any haemoglobinopathy identified through it should be confirmed by molecular studies. Being a screening test, it has an importance in premarital or antenatal screening as well as in the neonatal diagnosis of various haemoglobinopathies, particularly thalassemias.

The automated HPLC instruments currently in use are of a high precision and are moderately rapid. They use especially designed microbore columns, high precision gradient-forming liquid pumps and optical detectors.

HPLC has the following advantages over haemoglobin electrophoresis:

- The technique is less labour sensitive
- A very small sample is adequate
- Quantification of normal and variant haemoglobins is available for each sample
- As haemoglobin A2 is quantified, β- thalassemia trait can be diagnosed in a single procedure
- a large range of variant haemoglobins can be identified
- compound heterozygote can be detected
- fast moving haemoglobins can be detected by manual inspection of chromatogram

Considerable skill and experience is needed in interpreting the results of HPLC as the data produced are quite complex. Typical elution patterns of normal and some of the variant haemoglobins are shown in the following figures (1-4).

References

Barbara J. Bain. Haemoglobinopathy Diagnosis, second edition black well 2001.

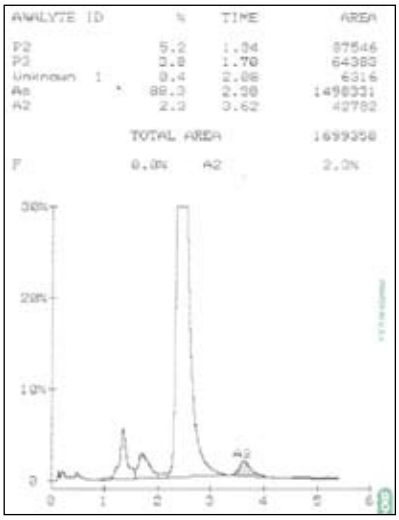

Fig. 1. chromatogram of a normal patient showing predominantly haemoglobin A and A2.

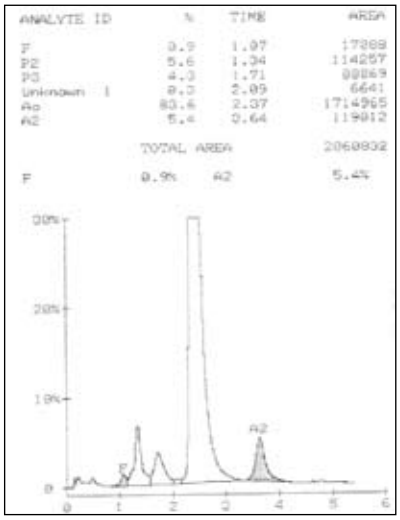

Fig. 2. chromatogram of a subject with β-thalassemia trait showing high A2

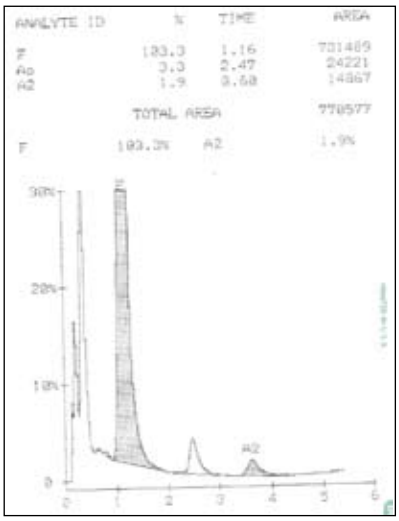

Fig. 3. chromatogram of a patient with β- thalassaemia major showing HbF only

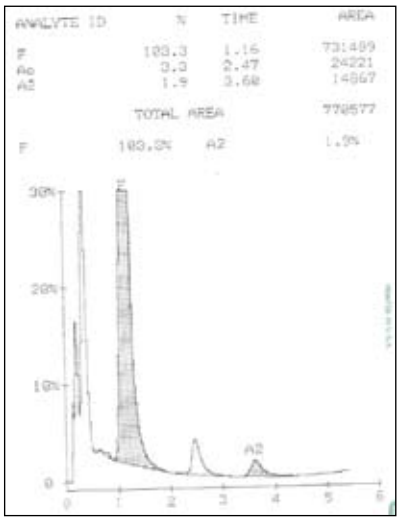

Fig. 4. fast moving haemoglobin

Platelet Transfusions: What We Need to Know

Mohammed Salman
Haematology

Platelet transfusions remain an integral part of treatment of haematology/oncology and surgical patients. Besides, these may be required in liver disorders, trauma setting and transplant setup.

Indications of platelet transfusions

Platelet transfusions are indicated in presence of active bleeding secondary to low platelets or platelet function defects (therapeutic usage). They may be required when bleeding is expected in the presence of very low platelet count (prophylactic usage).

1) Therapeutic usage

Platelets are transfused in a patient with active bleeding secondary to low platelets < 20 X 10^9/l or bleeding secondary to platelets function defect. Platelets are required in these circumstances if other measures (anti-fibrinolytic agents, DDVAP etc) have failed to secure haemostatsis.

It should be noted that patients having fever, sepsis, coagulopathy or anatomic bleeding may have higher threshold for platelets transfusion.

2) Prophylactic usage

Prophylactically, platelets are given to a patient with platelet count <10 X 10^9/l even in the absence of bleeding. The reason being such patients are at risk of intracranial bleeding. Other indications for prophylactic use include:

i. Non-bleeding patients with temporary myelo-suppression due to chemotherapy or radiotherapy or underlying disease (e.g. leukemia) in the presence of fever or minor haemorrhagic signs.

ii. Impending surgery or invasive procedures involving the CNS or other critical areas in which micovascular bleeding is harmful.

iii. Other minor surgery or invasive procedures where the external pressure can be utilised to maintain haemostasis can be performed at a platelet count of 50 X 10^9/l.

iv. Qualitative platelet function defect should be considered for patients with von Willebrand disease or qualitative function defect undergoing invasive procedures.

v. Open heart surgery patients with micro vascular bleeding having coagulopathy or low platelet or their dysfunction.

Efficacy of platelet transfusion

This can be assessed clinically and by observing the haemostatic response. The response can also be monitored in the laboratory by evaluating platelet count one hour and 23 hours after platelet transfusions which should demonstrate an increment in platelet concentration.

However, the efficacy of platelets transfusions can be influenced by conditions such as uremia, medications, coagulation disorders, alloimmunisation to platelet HLA antigens, infections or splenomegaly.

Some important considerations while transfusing platelets

i. ABO identical or compatible platelets are preferred. However, in adults ABO incompatible platelets may be used because the amount of plasma is very rarely of clinical concern.

ii. Platelets do not carry Rh or D antigen. Therefore Rh grouping is never a matter of concern while transfusing patients. However, Rh negative females of reproductive age group should be given Rh negative platelets because of the risk of possible red cells contamination of platelets or be considered for Rh immunoglobulins in such cases.

iii. Patients at risk for transfusion associated graft-versus host disease (TA-GVHD) should receive gamma irradiated platelets.

Types of Platelets available at AKUH

There are two types of platelet products that are available at AKUH:

Random donor platelets (RDP)

This is derived from a unit of whole blood after

centrifugation (Fig. 1). Each random donor platelet contains 50x10^9/l platelets in approximately 50 ml of plasma. The recommended dose of random donor platelet is 1 unit/10kg body weight. The expected increment in platelets count is 5-10 X 10^9/l per unit of platelet transfused.

Single donor platelets (SDP)

In contrast to RDP, it is obtained from a single donor by the process of aphaeresis which takes 1-2 hours for completion (Fig. 2 and 3). A single donor platelet contains 200-300 ml of plasma and has 300 X 10^9 platelets approximately. For adults one single donor platelets unit is the therapeutic dose and for children, a single donor platelet unit may be split or alliquoted. The expected platelet count increment from a single donor platelet unit in an adult is 25-50 X 10^9/l.

Difference between RDP and SDP

The use of single donor platelets obtained by apheresis has increased substantially in the past decade in part because of perceptions that single donor platelets are a better transfusion product. SDP are more expensive than random donor platelet concentrates, and comparative studies have shown that post-transfusion platelet increments and the incidence of alloimmunisation are similar, using the two preparations.

With improved donor screening, it is unlikely that there would be any detectable difference between the two platelet products in terms of the incidence of infectious disease transmission. Though some studies have suggested a reduction in bacterial transmission by transfusion with the use of single-donor platelets. Thus, except in cases where histocompatible donors are needed for alloimmunised patients, there is no compelling reason to choose single donor over random donor platelets.

How platelets are stored in a blood bank

Both RDP and SDP can be stored up to five days at 20-24°C with continuous agitation to prevent clumping. Storage is limited to five days due to the risk of bacterial contamination.

Quality Assurance of Platelets

Safeguarding the safety and quality of platelets is of crucial importance.

Currently, AKUH blood bank assures quality according to FDA and AABB guidelines for various blood products: firstly blood is collected only from healthy donors. Secondly, we monitor pH and sterility of the platelets bag. Also, storing platelets in an incubator within temperature limits assures safety from bacterial contamination.

References

1. Consensus Conference on Platelet Transfusion (1998); Synopsis of background papers and consensus statement. British Journal of Haematology. 101:609-617.

2. Guidelines for the Use of Platelet Transfusions (2008). British Journal of Haematology.

3. American Association of Blood Bank; sixteenth edition.

Fig. 1: Random donor platelets

Fig. 2: Single donor platelets

Fig. 3: A donor donating platelets by apheresis

Measuring Homocysteine Levels

Dr Fareeduddin Mazhar
Chemical Pathology

Homocystiene is a sulphur-containing amino acid, formed during metabolism of methionine. It is metabolized by pathways, remethylation and transsulfuration of methionine (Fig 1). Approximately 50 per cent of homocysteine generated from methionine is metabolized to cystathionine by cystathionine beta synthase (CBS). This is a one-way reaction that permanently removes homocysteine from the methionine cycle and initiates the transsulfuration pathway for the synthesis of cysteine and glutathione. In remethylation, homocystiene acquires a methyl group either from N5 methyltetrahydrofolate (in all tissues) or from betaine (in liver and kidneys) to form methionine. The former methylation step requires folate and vitamin B12 as cofactors in transsulfuration pathways are effective when there is excess of methionine or cysteine synthesis is required.

Elevated plasma homocysteine is associated with increased incidences of cardiovascular mortality, stroke, dementia and Alzheimer's disease, bone fracture, and higher prevalence of chronic heart failure. It was also shown that elevated plasma homocysteine is a risk factor for preeclampsia and neural tube defects (NTD). Homocysteine has been implicated in atherosclerotic and thrombotic vascular disease in the general and in end-stage renal disease (ESRD) population as well.

Most studies so far have indicated normal plasma homocystiene level to be in the range of 5 to 15µmol/L. However, this does not imply that there is of risk associated with homocysteine levels between 10 to 15 µmol/L.

In 1990, the cutoff for homocysteine level was greater than 20 µmol/ L. This number continued to go lower, and today a level somewhere around 12 µmol/ L is considered cutoff. Homocysteine analyses in NHANES III survey results shed some light into what constitutes 'normal' homocysteine levels in the US population. A 1999 study by NHANES investigators (published in Ann Intern Med 1999; 131: 331) showed that homocysteine levels continue to increase as a person ages, and that men have a higher mean concentration than women.

In AKUH clinical laboratory homocysteine levels are determined by Fluorescence polarization immunoassay (FPIA). Normal range is taken from 4.72 -14.05, both for males and females.

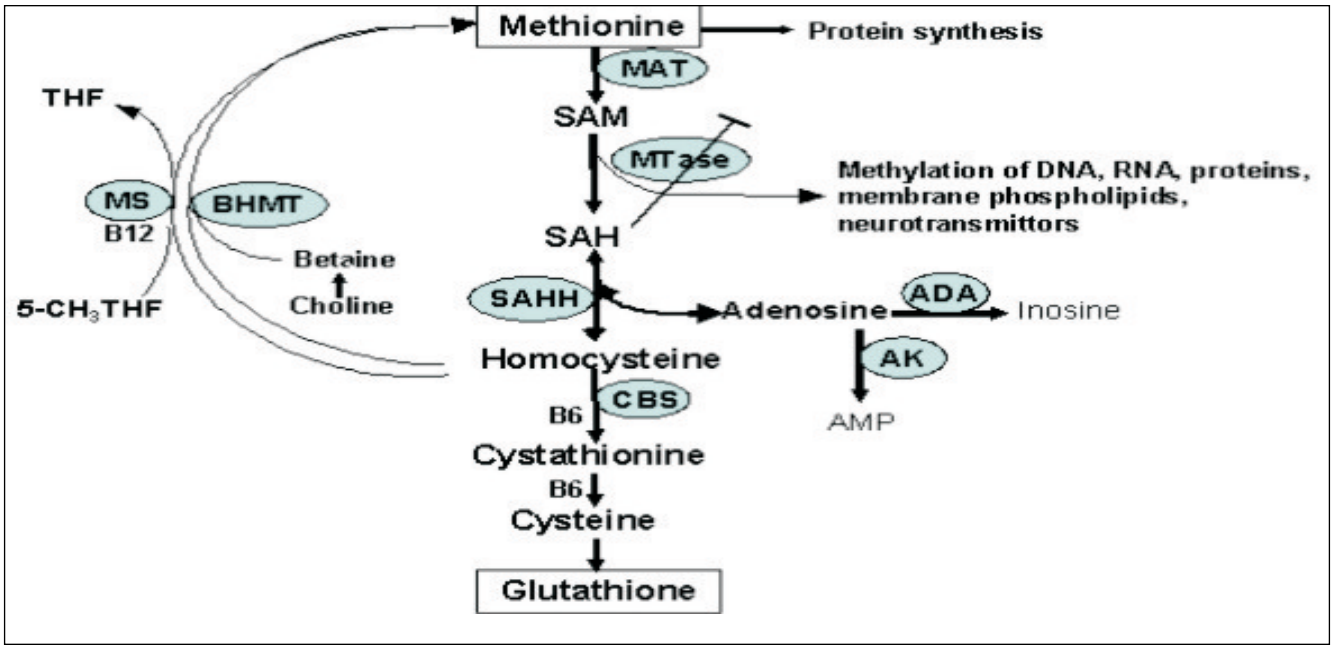

Fig. 1: Metabolism of Homocysteine

Parathyroid Hormone: A Marker of Vitamin D Deficiency and Insufficiency

Dr Aysha Habib Khan
Chemical Pathology

Vitamin D (25OHD) and parathyroid hormone (PTH) are important in both bone health and disease. In the past decade, there has been an increasing appreciation of the complexity and importance of their regulation and actions.

Widespread prevalence of Hypovitaminosis D has been identified worldwide. Surprisingly it is more common in people living in tropical zones than inhabitants of the countries where sunshine exposure is considered insufficient. Countries at low latitude have also shown a high prevalence of vitamin D deficiency, ranging from 50 to 97%. The suggested mechanism is customary clothing in countries like Middle East leading to a high prevalence of vitamin D deficiency. Similarly, a high prevalence of vitamin D deficiency is also reported from Pakistan.

Levels of 1,25-dihydroxyvitamin D (1,25(OH)2D), which is the biologically active form of vitamin D, are tightly regulated by parathyroid hormone (PTH), phosphate and calcium. Vitamin D deficiency causes a compensatory increase in PTH, in an effort to maintain calcium (secondary hyperparathyroidism). However, not all patients with hypovitaminosis D will necessarily show hyperparathyroidism. The term 'vitamin D insufficiency' defines a level at which the decreased vitamin D causes an increase in PTH. It has been suggested that the lowest vitamin D level that does not produce a rise in PTH should be selected as optimal. Magnesium deficiency may cause a blunted response to PTH.

Assumptions about vitamin D status should not be made based on PTH and calcium values, and 25(OH) vitamin D measurements should be requested when vitamin D deficiency is clinically suspected, irrespective of biochemical results. This depends on cost-benefit ratios that various physicians ascribe to testing for deficiency versus treating for suspected deficiency. Vitamin D deficiency can have several causes, and the pattern of laboratory values with respect to 25-hydroxyvitamin D and PTH and response to vitamin D supplementation may help lead to identification of an underlying pathology.

Surrogate Measures of Insulin Sensitivity

Dr Sahar Iqbal
Chemical Pathology

Both insulin resistance and impaired pancreatic beta cell function contribute to the chronic hyperglycemia in type 2 diabetes. Whereas insulin resistance is present several years before the manifestation of diabetes, impaired beta cell function is usually not seen until glucose tolerance becomes impaired. The relative contribution of impaired beta cell function and insulin resistance in the development of glucose intolerance and diabetes can be assessed by different methods.

Methods available for assessment of insulin sensitivity

1) **Hyper-insulinemic euglycemic clamp**
   This test is considered as gold standard for the assessment of insulin sensitivity but is laborious and expertise is required to carry out the test.

2) **Intravenous glucose tolerance test**
   This test is also compatible with hyper-insulinemic euglycemic clamp but also requires expertise.

3) **Homeostasis model assessment insulin resistance index (HOMA – IR)**
   This test estimates insulin sensitivity from measurement of fasting insulin and glucose.

4) **Insulin sensitivity index (Si)**
   This test estimates the insulin sensitivity from insulin and glucose response during an oral glucose tolerance test (OGTT).

The relative assessment of the risk of type 2 diabetes mellitus can be estimated by an insulin sensitivity index as resistance to insulin is developed before the manifestation of metabolic syndrome (hyperglycemia, hypertension, hyperlipidemia and hyperinsulinism which can lead to coronary artery disease and stroke).

In the AKUH clinical laboratory the insulin sensitivity test is conducted through OGTT in which two fasting blood samples of insulin and glucose are taken (at -15 and 0 minutes) followed by administering 75 g of glucose orally; then six more blood samples are taken for insulin and glucose at 30 minutes intervals for 180 minutes post-load. Whole body insulin sensitivity index (WBISI) is calculated through a calculation with fasting and mean of post-load values of serum insulin and glucose. Normal WBISI in normal glucose tolerance is 2.02, 2.16; however the lower the sensitivity the higher the resistance is expected.

Serum Insulin levels are measured at section of Clinical Chemistry through MEIA technique at AXYM (Abbott diagnostics) having functional sensitivity of 1.0 µU/ml.

Reference

Yeckel CW, Weiss R, Dziura J, Taksali SE, Dufour S, Burgert TS, et al. Validation of insulin sensitivity indices from oral glucose tolerance test parameters in obese children and adolescents. J Clin Endocrinol Metab. 2004 Mar; 89 (3): 1096-101.

Foetal Autopsy

Dr Sidra Arshad
Resident, Histopathology

An autopsy-also known as a post-mortem examination or autopsia cadaverum is a medical procedure that consists of a thorough examination of a corpse to determine the cause and manner of death and to evaluate any disease or injury that may be present. It is usually performed by a pathologist.

The question is what is the significance of a foetal autopsy? Clinicians should advise parents of the usefulness of a foetal autopsy in ascertaining the cause of death and this would in turn help in counseling their future pregnancies.

Since it is a useful and necessary tool for helping determine the cause of foetal death, it should be approached in a systematic and meticulous manner by paying close attention to every fine detail of external and internal gross examination followed by microscopic examination.

Components of the foetal autopsy

In order to be consistent and thorough in approach for every autopsy, the Autopsy Committee of the College of American Pathologists, with representatives of other interested organisations, prepared a guideline to assist pathologists in reporting foetal autopsies.

The complete medical record is reviewed to obtain the clinical history, with attention to medical obstetrical (past and present), and genetic conditions potentially associated with still birth.

The following examinations are commonly performed:

- Appropriate length of gestation
- General conditions: well preserved or macerated?
- Gross external examination with detailed description and photographs of abnormalities, dysmorphic features, and pertinent negative findings
- Gender
- Foetus weight and external measurement
  - Crown-to-rump length (crown to ischial tuberosities, ‘sitting height’)
  - Crown-to-heel length (crown to heel of extended leg)
  - Foot length
  - Hand length
  - Head circumference (i.e. occipitofrontal circumference - above ears)
  - Chest circumference (around nipples)
  - Abdominal circumference (at umbilical insertion)
- Gross description of the placenta and cord: weigh placental membranes, cord length and number of

vessels. A short cord can be a sign of neuromuscular compromise, a long cord can be a sign of heart failure, while a single umbilical artery can be associated with genitourinary anomalies (eg. renal agenesis, horseshoe kidney)

- Gross and microscopic examination of major organs, anomalies, including the weight of each organ. Paired organs are weighed together
- X-rays may reveal unrecognised skeletal malformations, or can be obtained to further evaluate suspected skeletal abnormalities. Plain whole body anteroposterior and lateral radiographs are taken with the infant's head 'straight'
- Foetal radiographs should be examined carefully by the autopsy pathologist for specific findings, such as:
  - Appropriate ossification of bones for gestational age
  - Documentation and timing of fractures (which should be sampled histologically)
  - Presence of ectopic mineralisation/ossification of non-bony tissues

Sections from the foetal and placental tissues should be processed for histological examination in all cases, even when severely autolysed or structurally normal. Important pathologic findings that have clinical relevance beyond that of identifying a cause of death can still be diagnosed in severely autolysed tissues.

Cytogenetic studies may be performed on the stillborn's blood, tissue, or body fluids (for example bile, urine, vitreous humour), as long as the cells are viable. Blood can be collected from the umbilical cord and skin samples are usually taken from the thigh after cleaning with normal saline. Fresh tissue samples should be placed in sterile medium (for example Hanks balanced salt solution) from the cytogenetics laboratory, or sterile saline solution, and kept at room temperature (do not use fixatives such as formaldehyde). In cases of moderate or marked autolysis, the umbilical cord or chorionic plate vessel can be sampled for karyotype analysis. Microbiologic studies can be performed on foetal blood taken from the heart or umbilical cord; however, foetal blood is more easily obtained from the foetal lung at autopsy.

Foetal autopsies should be performed with the care and respect that all autopsies are given. Also, it is not uncommon for a family to decide to view the body after the postmortem examination has been done; therefore careful dissection to avoid any disfigurement is important. Even the smallest, youngest (in gestational age), and most autolysed cases can receive a thorough post-mortem examination and be returned in a condition suitable for viewing management. Finally, autopsy reports should be completed promptly so that a family meeting with the clinicians can be timely.

Laboratory Diagnosis of Vaginitis due to *Trichomonas Vaginalis*

Dr Naima Fasih
Resident, Microbiology

Introduction

Trichomoniasis is a sexually transmitted protozoal infection caused by *Tricomonas Vaginalis*. Women and men may be asymptomatic carriers, or they may experience most commonly, foul smelling purulent discharge and itching. It sometimes occurs as co-infection with other STDs and therefore is a public health concern. *T. vaginalis* inhabits the vaginal and urethral tissues, causing direct damage to the epithelium, leading to microulcerations. Symptoms typically occur after an incubation period of 4-28 days.

Laboratory Diagnosis

Following are the commonly used tests for the diagnosis of *T. vaginalis:*

- Wet mount
- Culture
- Giemsa stain

Other tests that can also be used in the laboratory are:

- DNA probe
- Latex agglutination
- Enzyme-linked immunoassay
- Direct fluorescent antibody staining
- RNA probe semi automated system

Direct Wet Mount

The identification of *T. vaginalis* is usually based on the examination of a wet preparation of vaginal and urethral discharges, prostatic secretions or urine sediments. Specimens are diluted with a drop of saline and are examined within one hour under low power. The presence of actively motile organisms with jerky motility is diagnostic. The movement of the undulating membrane may be seen as the motility of the trophozoite diminishes. Pus cells are often present. The sensitivity of the wet preparation is between 50 and 70 per cent and is largely affected by the time between collection and examination of the specimen. This test is available at the clinical laboratory of Aga Khan University Hospital.

Culture

Culture has increased sensitivity (>80 per cent) than the wet mount and is considered as a gold standard. Specimens must be collected properly and inoculated as soon as possible on appropriate medium, i.e. Modified Diamonds, Trichosel or Hollanders medium. Immediate processing is mandatory for maximum recovery of organisms. This approach is not routinely used in clinical laboratories. Culture systems which allow direct inoculation, transport, culture and microscopic examination are commercially available.

Giemsa Stain

This stain is also used routinely but the sensitivity is low as organisms are often difficult to recognize.

References

1. Henry D Isenberg. Clinical Microbiology Procedure Handbook, 3rd ed. Vol. 1 ASM Press.

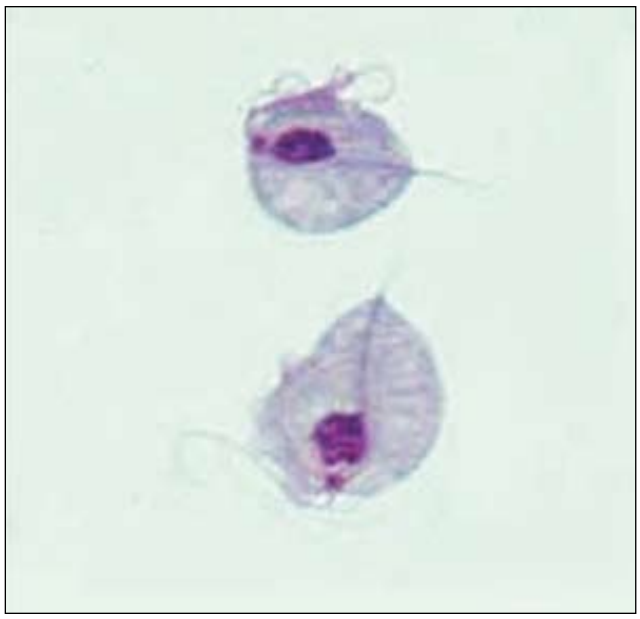

Fig. 1: Trophozoites of T. vaginalis (Image taken from www.dpd.cdc.gov/dpdx/HTML/ImageLibrary/Trichomoniasis_il.htm)

AKUH Clinical Laboratory Continuing Medical Education (CME) Seminars

Reported by
Seema Vaqar

CME Seminar in Nawabshah

The CME Seminar was held at Latif Hall, Peoples Medical College & Hospital (PMCH), Nawabshah with the collaboration of Department of Pathology and Microbiology on March 2, 2010. It covered a broad range of topics on infectious hepatitis B, C, and D diseases and swine flu, aplastic anaemia and role of histopathologist as diagnostician & prognostician in the management of cancer.

Dr Usman Shaikh addressing the audience at CME Seminar

It was well attended and about 600 health care professionals, students, staff and faculty members from local area hospitals and academic institutions participated. Professor Dr Azam Hussain Yousifani, Principal of the College, presided over the session.

In his opening statement, Professor Dr Rasool Bux Shaikh, pathologist from PMCH shared the mission of AKUH clinical laboratory by conducting such seminars. He highlighted the role of AKUH pathologists in education and provision of high quality nationwide diagnostic services and invited the Chief Guest, Professor Azam Hussain Yousifani for his welcome remarks.

Professor Azam Hussain Yousifani applauded the effort and initiatives of the speakers from AKU 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.

Dr Seema Irfan delivering a talk on viral hepatitis

Dr Usman Sheikh, Assistant Professor and Consultant Haematologist, AKU impressed the audience with a lucid and engaging presentation on Aplastic Anaemia. He referred to data obtained by the AKUH clinical laboratory of Aplastic Anemia within the country and emphasized the early diagnosis and management of the disorder.

Dr Abrar Barakzai addressing the audience at CME Seminar

Question-answer session at the Seminar

Dr Seema Irfan, Assistant Professor AKU delivered a lecture on hepatitis B, C and D Virus and discussed the interpretation of serological and PCR results and epidemiology of HCV and HBV in Pakistan, especially in Sindh. She highlighted the importance of viral genotyping as well as testing for viral load in diagnosis and treatment of hepatitis B and C.

Dr Abrar Barakzai, Assistant Professor, AKU provided an overview of various tumors and cancer. He emphasized the significant role of histopathologists in the diagnosis and prognosis of cancer management and showed national cancer data reflecting a high prevalence of oral cavity and lung cancers in Sindh as compared to other regions of Pakistan. He also highlighted the role of immuno-histochemistry in the proper diagnosis of cancer.

Dr Anwar Ali Akhund, Professor of Pathology, PMCH Nawabshah presented a lecture on Swine Flu infection and discussed prevention of Swine Flu and its management.

The seminar was successful as indicated by the interest of the audience in the question and answer session.

Dr Abdul Qayoom Memon, Professor of Medicine, ended the seminar by delivering a vote of thanks. Before closing, the chief guest, Prof. Azam Hussain Yousifani presented shields and *Ajrak* to the guest speakers.

Participants at the Seminar

Speakers of CME Seminar along with Principal and Faculty of Peoples Medical College and Hospital, Nawabshah

CME Seminar in Rahim Yar Khan

This year the clinical laboratory has started 'Focus Continuing Medical Education' seminars. The objective is to involve with local pathologists and physiciansin order to develop a strong and long-term relationship.

Our first focused CME for 2010 was held in Rahim Yar Khan on March 25th. It was well attended - about 50 doctors from all major specialties including medicine, surgery, gastroenterology and the pathology departments of Sheikh Zaid Hospital were present on the occasion.

In his opening statement Dr Asghar Javaid, Staff Pathologist of Multan Stat Lab shared the mission of AKUH Clinical Laboratory of conducting such seminars and Focus groups. He also highlighted the role of AKUH pathologists in educating as well as providing high quality diagnostic services for physicians and hospitals nationwide.

Dr Romena Qazi, Section Head of Molecular Pathology spoke on Detection, Genotyping of hepatitis C. She also shared the data for the prevalence of hepatitis C in the world. She emphasised the importance of proper diagnosis of hepatitis C.

Dr Romena Qazi addressing the audience at CME Seminar

Dr Irfan Ahmed, gastroenterologist and Professor of Medicine, Sheikh Zaid Hospital spoke on the management of hepatitis C. He also highlighted the symptoms and the root cause of hepatitis C and also gave the standard protocol for the treatment of hepatitis C.

Finally there was a lively question and answer session, the event concluded on a successful note.

Participants at CME in Rahim Yar Khan