FAQ

Frequently Asked Questions On CML

Focus on 2nd Generation TKIs

English

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FAQ

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Specially compiled for

Friends of Max

Q. 1 What is Leukaemia?

Leukaemia is blood cancer. Our bone marrow produces blood and blood cancer begins from the bone marrow. There are three types of blood cells and Leukaemia is cancer of the white blood cells.

Q. 2 What is Chronic Myeloid Leukaemia and how common is it?

There are four main types of leukaemia. Two of them are acute and two are chronic. The Acute Leukaemias are Acute Myeloblastic Leukaemia (AML) and Acute Lymphoblastic Leukaemia (ALL) while the Chronic Leukaemias are Chronic Myeloid Leukaemia (CML) and Chronic Lymphocytic Leukaemia (CLL). Acute leukaemia develops quickly while Chronic Leukaemia develops slowly. In CML, white cells are present in excess. Their production goes out of control. These cells fill the bone marrow and also come out in the blood.

CML is the commonest blood cancer in India and other Eastern countries. CLL is the commonest blood cancer in Western countries.

Q. 3 What is the age at which CML occurs?

In India, most commonly, CML affects the age group between 30-60 years. In the Western world, it affects the age group between 50-70 years. Rarely, it can occur in children and very old persons as well. It affects both males and females.

Q. 4 Is CML a genetic disease? If I have it, will my children have higher chance of getting it?

No. CML is not a genetic disease. Children or any other member of the household has no higher chance of getting it. CML is not inherited.

Q. 5 Is CML a contagious disease? Can it spread to my spouse, parents or children?

No. CML is not an infection and hence it is not a contagious disease. It cannot spread to other members in the house.

Q. 6 Why did I get CML? What are the risk factors?

The exact answer to your question is unknown. In Japan, after the atom bomb explosion at Hiroshima and Nagasaki, a large number of affected individuals developed CML after a few years. Hence, there is a role of radiation. A nuclear accident can result into CML. However, in routine civil life, the reason one gets CML still remains under research. During the course of day to day living there is no way one would be exposed to the kind of radiation that would result in CML.

Diagnosis

The **diagnosis** of CML is usually made with information from blood and bone marrow tests.

Blood Tests

The testing for CML includes blood cell counts and a blood cell examination.

Blood cell counts. The doctor orders a test called a complete blood count to check the numbers of blood cells. With CML, the red cell count is lower than normal. The number of white cells is higher than normal and may be very high. The number of platelets may be higher or lower than normal.

Blood cell examination. The cells are stained (dyed) and looked at (dyed) and looked at with an instrument called a light microscope. A person with CML has a small number of developing cells called "blast cells" in his or her blood. Blast cells are not found in the blood of healthy individuals.

Bone Marrow Tests and Cytogenetic Tests

Some signs of CML do not show in blood tests. The doctor has to look at a small number of cells (a sample) from the marrow. The samples of cells are obtained with tests known as a **bone marrow aspiration** and a **bone marrow biopsy.**

Samples of marrow cells are examined under a microscope. This is called a cytogenetic analysis. The examiner looks at a map of the chromosomes in the cell. The map is called a "karyotype." The Ph chromosome in a CML cell can be detected on the karyotype.

FISH or fluorescence in situ hybridization is a special test used to detect CML cells that may not show up on a standard cytogenetic test for the Ph chromosome. A PCR test can be done on cells from blood or marrow.

PCR also known as Polymerase Chain Reaction is a very sensitive test that can detect CML cells that are not found by the FISH test. This test can detect a very small number of CML cells. A PCR test can be done on cells from blood or marrow.

Accelerated Phase CML

In the accelerated phase, the patient may develop **anemia,** the number of white cells may go up or down, or the number of platelets may drop. The number of **blast cells** may increase and the **spleen** may swell. People with accelerated-phase CML may feel ill.

Blast Crisis Phase CML

Patients with blast crisis phase CML have an increased number of blast cells in the **marrow** and blood. The number of red cells and platelets drops. Patients may have infections or bleeding. They may also feel tired and have shortness of breath, stomach pain, or bone pain.

Signs and Symptoms

People with CML may not have any symptoms at the time of diagnosis. They may be diagnosed following a medical examination for another condition or as part of a periodic checkup. CML **signs and symptoms** tend to develop gradually. Some signs and symptoms of CML are:

Tiring more easily

Shortness of breath doing usual day-to-day activities

Pale skin color

Enlarged **spleen** leading to a "dragging" feeling on the upper left side of the abdomen

Night sweats

An inability to tolerate warm temperatures

Weight loss.

Many of the signs and symptoms for CML are common to other illnesses. Most people with these signs and symptoms do not have CML.

Also, exposure to certain chemicals, especially benzene over a period of time can result in blood cancers including CML. Benzene is a chemical in petrol and used in the rubber industry. However, exposure to petrol etc. in routine civil life cannot produce CML.

Q. 7 What is the Philadelphia chromosome?

We have 23 pairs of chromosomes (genetic material). In CML, the genetic material from chromosome 9 and 22 get translocated resulting into a shortened chromosome 22. This produces a new gene called bcr-abl. This shortened chromosome 22 was first detected in 1960 by scientists in the city of Philadelphia. Since then, the so called Philadelphia chromosome has been shown to be a consistent finding in patients of CML. This is of diagnostic importance.

Q. 8 Was I born with Philadelphia chromosome?

No. One is not born with the Philadelphia chromosome or bcr-abl gene. It develops later on. Bcr-abl leads to excessive production of an enzyme (protein) called Tyrosine Kinase (TK) which causes increase in white cells.

Q. 9 What are the symptoms of CML?

CML may not have any symptoms as it develops slowly. Many patients are diagnosed during routine blood tests carried out for one reason or the other.

Symptoms of CML are vague. Tiredness, weight loss, excessive sweating, fullness in the left upper part of the stomach , discomfort or lump in the same region and feverish feeling may occur. These symptoms are very common while CML is rare.

Once the disease is advanced (for example after few years into blastic phase), there can be anaemia (lack of blood) leading to pale appearance, bruises on the surface of the skin due to bleeding , infection leading to fever and development of glands in the neck or at other areas (lymphadenopathy).

Q. 10 How is CML diagnosed?

Usually, CML is suspected because of enlarged spleen (lump in the left upper part of stomach). The GP or your family physician, in such cases would ask for blood counts which in turn may show an increase in white cell counts. Please remember that the increase in white cells most commonly occur also due to infections causing fever, cough and diarrhoea etc. Hence, one should not get unduly worried just because the white cells are more.

Subsequently, the haematologist or medical oncologist will look at the type of blood cells and also carry out bone marrow examination looking for the presence of Philadelphia chromosome or bcr-abl gene which will confirm the diagnosis.

Q. 11 Cancer has stages. Does CML also have stages?

Chronic Myeloid Leukaemia has phases i.e. chronic phase, accelerated phase and blast phase. These phases may be loosely called stage I, II and III or early, intermediate and late.

Most of the patients are diagnosed in the Chronic phase or early phase itself. The disease remains stable for years during which there are not many complaints and one can work normally. It is during this phase that the treatment is started which is usually in the form of oral pills (Glivec-Imatinib) taken daily. No one requires hospitalization in the Chronic phase. During this phase, there are very few or no blast cells in the blood or marrow.

The next phase is the Accelerated phase which can sometimes develop very rapidly. In this phase, there are more blasts in the blood or marrow. The patient feels more weak and tired. He may also develop fever (infection) or bleeding. Spleen becomes larger and may stop responding to treatment.

Subsequently, the Blast phase develops. This is like acute leukaemia. Here, the number of blasts in the blood or marrow is maximum. Patient becomes very weak and often requires blood transfusion and even hospitalization.

Q 12. How is CML treated?

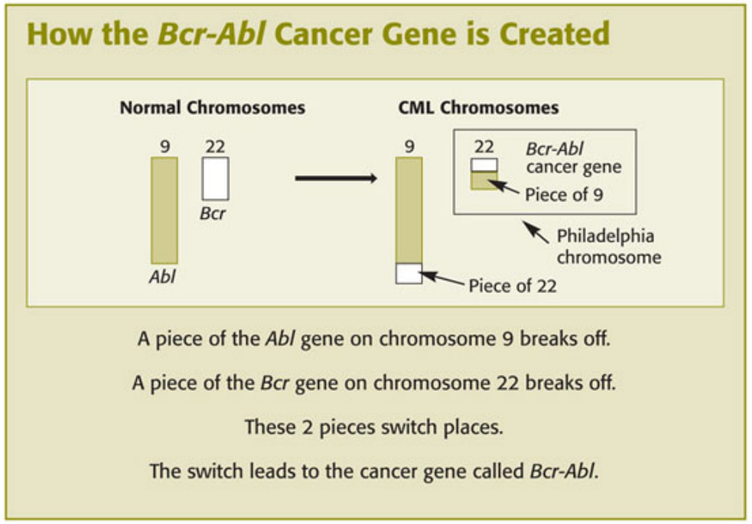
Chronic Myeloid Leukemia is a form of blood cancer arising from a specific cell, referred to as a stem cell, which give rise to series of different cells including the myeloid cells both in normal and abnormal states. Hence it is sometimes referred to as a stem cell disorder. The reason for initiation and progression to a cancerous process is because of the acquisition and persistence of a specific abnormality within the stem cells. This abnormality is related to the chromosomes, which are again specific to the 9th and 22nd chromosomes. Because of rearrangement in some chromosomal materials within these two sets of chromosome an abnormality develops resulting in the form of generation of an abnormal protein called the BCR-ABL fusion protein which has unregulated enzymatic activity and eventually causes the proliferation of the cells, driving the progression of the disease. It is this proliferation that manifests as high counts with spleen enlargement and constitutional symptoms typical of CML. The objective of treating CML therefore revolves around blocking the activity of this protein using small molecules thereby gaining control of the disease by suppressing the protein activity and allowing the existing normal stem cells to function. However, this suppression can only occur after sustained and prolonged therapy with these small molecules, which are referred to as tyrosine kinase inhibitors of which we are more familiar with Imatinib, Nilotinib and Dasatinib.

Q 13. What is meant by 2nd generation TKIs?

Any drug or molecule for treatment of conditions in medicine undergoes an evolutionary process wherein there might be small alterations in their structure which gives desirable properties in terms of activity, potency, stability and side effect profile. These are, for purpose of recognition, categorized in terms of generations. With regards to CML we have the first generation TKIs in Imatinib while the 2nd generation TKIs available are Nilotinib and Dasatinib. Currently there is also the 3rd generation TKIs in the form of Ponatinib. The 2nd generation TKIs, Nilotinib and Dasatinib are much more potent molecules as compared to Imatinib. They are also quite distinct from one another in terms of their side effect profile though comparable

Chronic myelogenous leukemia (CML)

Chronic myelogenous leukemia: A chronic malignant disease in which too many **white blood cells** belonging to the myeloid line of cells are made in the bone marrow. CML patients have what is called the "Philadelphia Chromosome" (Ph chromosome). Chromosomes are structures in the cells that contain genes. Every cell with a **nucleus** has **chromosomes**. Genes give instructions to the cells. The Ph chromosome is made when a piece of chromosome 22 breaks off and attaches to the end of chromosome 9. A piece of chromosome 9 also breaks off and attaches to the end of chromosome 22. The break on chromosome 9 involves a **gene** called Abl. The break on chromosome 22 involves a gene called Bcr. The Bcr and Abl genes combine to make the CML-causing gene called the **Bcr-Abl cancer gene**.



Phases of CML

There are three phases of CML:

- The chronic phase
- The accelerated phase
- The blast crisis phase

Chronic Phase CML

Most patients are in the chronic phase of the disease when their CML is diagnosed. In this phase, CML symptoms are milder. **White cells** can still fight infection. Once patients in the chronic phase are treated, they can go back to their usual activities.

SOME WEBSITES THAT MAY BE OF HELP IN ACCESSING INFORMATION

www.cancer.gov

www.leukemia-lymphoma.org

www.cancer.org

www.ncrn.org.uk

www.cancerworld.org

www.medterms.com

www.cmlhealthcare.com

www.cmlsupport.org.uk

www.newcmldrug.com

www.cmlsupport.com

www.cmladvocates.net

www.gistsupport.org

www.gistsupportuk.com

in terms of their effectiveness in treating newly diagnosed CML patients. They would also be useful when there is failure of adequate response to Imatinib which could result from mutations occurring in the BCR ABL protein binding sites.

Q 14. When are 2nd generation TKI's used as first line of treatment?

2nd generation TKIs are considered many times more potent than Imatinib in terms of their capability to bind with the BCR ABL protein and suppressing or blocking its activity. They have the capability to induce quicker and deeper responses in newly diagnosed patients with CML, which we today recognize as an important goal towards better control and the concept of treatment free remissions. It is perceived that these goals of sustained deep molecular or complete molecular responses are better achieved with 2nd generation TKIs. Under the circumstances there is an inclination towards using 2nd generation TKIs in newly diagnosed patients. However, there is no data that has conclusively shown that it has improved overall survival over Imatinib. There is also a situation of using 2nd generation TKIs in newly diagnosed CML when there is indication that it is a high-risk disease, which the clinician ascertains through a scoring system called the SOKAL score. There is a suggestion that 2nd generation TKI are better suited when SOKAL risk score is high. Again 2nd generation TKIs are used when there is intolerance to Imatinib, which is rare but not uncommon.

Q 15. Why does physician prescribe dasatinib vs nilotinib or vice versa? What is the difference between the two?

Physician can use either nilotinib or dasatinib in first line. However there may be specific instances based on presence of comorbidities for using either. If the patient has pre-existent diabetes or peripheral vascular disease or any cardiac arrhythmia we may avoid nilotinib as first choice. Similarly if the patient has pulmonary problems, autoimmune disorder or hypertension etc physician may avoid dasatinib. However these are only relative.

Both molecules are completely different with different affinity for targets. Their activity in second line is sometimes dependent on presence or absence of specific mutations, if present, some of which are resistant to either nilotinib or dasatinib. These are defined and determine the physician's choice of either.

These molecules have their own unique toxicities too which have to be monitored and decisions taken thereafter for their continuance or cessation.

The patient on nilotinib can be switched to dasatinib and vice versa provided there is no specific mutation for the particular molecule for which the alternative has been used.

Q 16. How should Nilotinib / Dasatinib be taken?

Nilotinib is available in the form of pale yellow capsules in strengths of 150mg and 200mg. The dosing is usually 300 to 400mg per day in divided doses. The intake of the medication has to be time bound and care should be taken to consume it with water one hour before or 2 hours after food. The capsules should not be opened or tampered with thus ensuring appropriate dosing.

The recommended starting dosage of Dasatinib for chronic phase CML is 100 mg administered orally once daily. The recommended starting dosage of Dasatinib for accelerated phase CML, myeloid or lymphoid blast phase CML, or Ph+ ALL is 140 mg administered orally once daily. Tablets should not be crushed or cut; they should be swallowed whole.

Q 17. Can Nilotinib / Dasatinib be taken with food? Is there any food to avoid?

Care should be taken when taking Nilotinib. It is ideally consumed orally with a glass of water on an empty stomach, which means the medication should be taken one hour before food or 2 hours after food at a fixed time preferably 12 hours apart.

Dasatinib should be taken one fixed time a day, with or without food, either in the morning or in the evening.

The drugs can interact harmfully with many medicines and supplements and increase the chance for serious and life-threatening side effects – it is advisable to inform your doctor about all other medicines including over-the-counter medicines, vitamins, antacids and herbal supplements.
When on TKIs, foods to be avoided include grapefruit products and grapefruit supplements and extracts.

Q 18. How can a patient tell if the drug is working?

TKIs are potent BCR-ABL kinase inhibitors, the culprit protein. Following suppression of the protein activity the first response we notice is the normalization of blood counts, normalization of type of cells in peripheral circulations (the hematological response). This situation also parallels the improvement in symptoms, decrease in spleen size and disappearance of constitutional symptoms. This is followed by gradual decline in the chromosomal abnormality in the bone marrow (cytogenetic response), which also parallels the decline in the quantity of bcr-abl protein levels in the circulation (molecular response). The pace of these responses can be variable with hematological responses occurring latest by 12 weeks, cytogenetic responses within 6 to 12 months and deep molecular responses by 12 months. These responses are ascertained by specific tests which includes complete blood counts, specific chromosomal analysis through cytogenetic testing and molecular response analysis using specialized tests such as polymerase chain reaction (PCR).

Q 19. What are the common side effects of Nilotinib?

There could be a host of side effects that can occur with Nilotinib. These could be categorized as immediate or late. Some of them may be sporadic and self-limiting while some could be chronic and persistent. The immediate side effects include nausea, rash, non specific headache, feeling of lethargy and tiredness, itching, vomiting, intermittent diarrhea, cough, constipation, muscle and joint pain, nasal stuffiness and rhinitis, sneezing, sore throat. Sometimes there could be a drug fever and associated night sweats. Other more serious side effects could relate to aberrations leading to abnormal cardiac rhythm which can be potentially serious requiring attention. This can particularly occur in presence of electrolyte disturbances, use of

cells make up almost half of the blood.

Relapsed CML. CML that responded to treatment but then returned.

Remission. No sign of the disease and/or a period of time when the disease is not causing any health problems.

Resistance. When a drug does not work or stops working.

Stem cell. A type of cell found in marrow that makes red cells, white cells and platelets.

Tyrosine kinase inhibitor (TKI). A drug that blocks cell growth. Gleevec (Imatinib), Sprycel (Dasatinib) and Tasigna (Nilotinib) are TKIs that are used to treat CML.

Hematologist. A doctor who treats blood cell diseases.

Hemoglobin. A protein in red cells that carries oxygen. A blood test can tell how much hemoglobin you have in your blood.

HLA (Human Leukocyte Antigens) Typing. A blood test that determines a person's compatability for purposes of a bone marrow or stem cell transplant based on the types of antigens present

Immune response. The reaction of the body to foreign material. Examples of foreign material are an infection-causing microorganism, a vaccine or the cells of another person when those cells are used for an allogeneic stem cell transplant.

Immune system. Cells and proteins in the body that defend it against infection. Immunoglobulins. Proteins that fight infection.

Immunotherapy. The term for treatments that can boost the body's immune system.

Karyotype. A map of the 46 human chromosomes of a cell. There are 22 matched pairs and the sex chromosomes, shown as a separate pair (XX for females or XY for males).

LFTs. Liver Function Tests. A group of blood tests that can help to show how well a person's liver is working. LFTs include measurements of albumin, various liver enzymes (ALT, AST, GGT and ALP), bilirubin, prothrombin time, cholesterol and total protein. All of these tests can be performed at the same time

Leukemia. A cancer of the marrow and blood.

Lymphocyte. A type of white cell that is part of the immune system and fights infection.

Marrow. The spongy material in the center of bones where blood cells are made.

Oncologist. A doctor who treats people who have cancer.

Pathologist. A doctor who identifies diseases by studying cells and tissues under a microscope.

PCR. The short name for a lab test called “polymerase chain reaction,” a very sensitive test that can measure the presence of a blood cancer cell marker in the blood. PCR is used to detect blood cancer cells that are below the level of detection by cytogenetic tests (for example, FISH).

Plasma. The liquid part of the blood.

Platelet. A type of blood cell that helps prevent bleeding. Platelets cause plugs to form in the blood vessels at the site of an injury.

Red cell. A type of blood cell that carries oxygen to all parts of the body. In healthy people, red

concommittant medications such as specific antihistamines and certain anti acidity drugs and even certain antibiotics. Nilotinib can also induce derangement in glucose metabolism resulting in elevated glucose levels and poor diabetic control in patients with pre-existing diabetis. It can cause low potassium and magnesium levels in the body resulting in symptoms of tiredness. It can cause increase in the bilirubin levels which again may be self-limiting. During initial treatment nilotinib can rarely cause bone marrow suppression which manifests as low blood counts. The delayed side effects attributed to nilotinib have been peripheral vascular disease due to which there may be narrowing of vessels particularly in the extremities resulting in compromised blood flow. There have been concerns too about accelerated coronary narrowing especially in predisposed individuals having diabetis. Some patients rarely develop hypertension too after starting nilotinib. It is important to recognize these problems and take remedial measures and if required stop its use in presence of no remitting problems.

Q 20. What are the serious side effects of Nilotinib?

While any of the side effects if persistent can be serious, it is those related to the cardiovascular system that is of concern. Electrolyte imbalances and direct effects on the heart can result in abnormal rhythms which if not recognized is life threatening. The increase in sugar levels and poor control of diabetes induced or aggravated by Nilotinib can be detrimental if not controlled. Liver dysfunction induced by nilotinib may require cessation of the medication to allow recovery. The peripheral vascular disease resulting from long-term nilotinib ought to be recognized to prevent heart attacks and peripheral vascular (blood) insufficiency resulting in poor circulation to the extremities.

Q 21. How will the doctor monitor for possible side effects?

Proper interviewing of the patient’s well-being, physical examination, relevant investigations (hematological and biochemical), proper reporting by the patient of symptoms, concomitant medicines being taken for other conditions would help in identifying, preventing and alleviating side effects.

Q 22. Should any symptoms alert a patient on the drug to contact his doctor immediately?

Any chest discomfort in the form of chest pain, palpitation (abnormal heart beat), and breathlessness should trigger a discussion with the doctor and attention by a medical team. Tingling sensation in lower limbs, discoloration of the lower limbs and pain in the lower limbs on exertion would indicate circulatory compromise and need to be addressed.

Q 23. How long will patient need to take these drugs?

It is generally considered a lifelong therapy. However, in the more recent understanding the concept of cessation after achieving a durable and sustained response for defined length of time have been explored in trial settings with promising results. However these aspects are to be considered only within the framework of a clinical study and not as routine practice.

Q 24. What should a patient do if he forgets to take the daily dose?

It would be best not to try and compensate the dose. A dose missed remains missed and the patient should go onto the next dose. However it has to be emphasized that compliance to therapy is of utmost importance.

Q 25. What should a patient do if he over doses on the drug?

Nothing really is to be done. However, one may consider adjusting with the next dose or even omitting the next dose. Again it is best to be conscious of your dosing and avoid such occurrences on a routine basis.

Q 26. How should the drug be stored?

The medicine should be stored strictly out of the reach of children at room temperature of 15 degrees to 30 degrees C. The medicine should not be used after the “expiry date” which is mentioned on the carton (box) after EXP

Q 27. Can a patient become pregnant when on these drugs?

It is advisable not to become pregnant while on the drugs. Any TKI has a potential to have an adverse impact on the growing fetus especially in the first 3 months of pregnancy with reports of significant neural abnormalities being reported. If absolutely desirous of pregnancy it is important to understand that the patient should have achieved a sustained deep response which has sustained for at least 2 years following which they could explore cessation of TKI and thereafter try to conceive with close monitoring for any disease activity.

Q 28. Can a patient on these drugs breast-feed her baby?

There could be excretion of these drugs in breast milk and therefore breast-feeding is best avoided.

Q 29. If a male patient desires to be a father, can he continue on the drug?

The recommendation for avoiding conception holds good for either sex while on the drug. However the chances that it will have an adverse effect when the patient is a male desirous of conceiving is remote.

Q 30. How is CML monitored when a patient is on these drugs?

The guidelines for monitoring a patient on 2nd generation TKIs is not any different from the general recommendation while on any TKI. The blood counts, biochemical parameters are routine assessments. Specific assessment would include cytogenetic response assessment on the bone marrow if possible. More importantly the molecular monitoring for decline of the BCR ABL protein is to be done at fixed intervals (ideally 3 months but at least every 6 months) in order to understand the ongoing response to therapy and also identifying any sluggish responses or loss of response.

Glossary of commonly used terms

Anemia. A decrease in the levels of hemoglobin in the blood.

Antibiotics. Drugs that are used to treat infections caused by bacteria and fungi. Penicillin is one type of antibiotic.

Antibodies. Proteins made by plasma cells in the blood. Antibodies help to fight infection in the body.

Blast cells. Early bone marrow cells.

Bcr-Abl. A gene and also a protein created by the translocation of Chromosomes 9 and 22

Bone Marrow. The soft, spongy tissue found in the center of most large bones that produces the white cells, red cells and platelets

Bone marrow aspiration. A procedure to remove marrow cells so that they can be examined to see if they are normal. A liquid sample of cells is taken from the marrow and then the cells are looked at under a microscope.

Bone marrow biopsy. A procedure to remove marrow cells and examine them to see if they are normal. A very small amount of bone filled with marrow cells is taken from the marrow, and the cells are looked at under a microscope.

CBC. Complete Blood Count

Chemotherapy or drug therapy. Treatment with chemical agents to treat CML and other diseases.

Chromosomes. Any of the 23 pairs of certain basic structures in human cells. Chromosomes are made up of genes. Genes give the instructions that tell each cell what to do. The number or shape of chromosomes may be changed in blood cancer cells.

Differential Count. A test that measures the relative numbers of white blood cells (WBCs) in the blood; it also includes information about abnormal cell structure and the presence of immature cells (Blasts or myeloblasts)

FISH. The short name for a test called “fluorescence in situ hybridization.” This is a test to measure the presence in cells of a specific chromosome or gene. This test can be used to plan treatment and to measure the results of treatment.

Hematocrit. The amount of blood that has red cells.