INSIGHT

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GROUP B STREPTOCOCCUS INFECTION

Since the early 1970's, Group B Streptococcus (GBS) have been identified as the number one cause of life threatening infections in newborn babies. Group B Streptococcus should not be confused with Group A Streptococcus which causes throat infection and may lead to Rh fever and RHD.

Streptococcus group B is normally found in the vagina and lower intestine of 15% to 40% of all healthy, adult women. Those women who test positive for GBS are said to be colonized. Neonates acquire the infection as a vertical transmission from the maternal genital tract in utero or at delivery

Diseases in neonates can manifest in two forms. Early onset and late onset. Early sepsis with GBS is often observed within 24 hr of delivery but it can manifest as late as 7 days after birth. Meningitis, Pneumonia and bacteremia is common etiology with GBS. Premature babies are more susceptible to GBS infection than full term babies. Performance of a cesarean section will not eliminate the risk of infection.

An estimated 12,000 infants in the United States become infected with GBS each year resulting in death of an estimated 2,000 infants, while leaving many others mentally and/or physically handicapped.

GBS infections are more common than other illnesses for which pregnant women are screened, such as rubella, Down's syndrome and spina bifida. Yet, GBS remains generally unknown to the public.

Fortunately**, there is testing and a preventative treatment available** that can help prevent many of these infections

GBS AND PREGNANCY

"Do All Women Carry GBS?"

If 1000 women, regardless of race or socioeconomic status, had a vaginal culture taken, 150-350 would test positive for GBS. Because GBS usually does not cause problems for the adult female, most women carry it and do not know it. Yet, GBS can cause serious illness in babies born to women who carry the bacteria.

"Is GBS a Sexually Transmitted Disease?"

Since GBS is normally found in the vagina and/or rectum of colonized women, one way it can colonize another individual is through sexual contact. However, this bacteria usually does not cause genital symptoms or discomfort and is generally not linked with increased sexual activity. Therefore GBS is not considered to be a sexually transmitted disease.

"How Common Are GBS Infections?"

Out of every 1000 births, three babies will become ill with GBS. Why only certain infants fall victim to this infection is not completely known. An estimated 12,000 babies will suffer from GBS infections each year.

"What Complications Does GBS Cause?"

Most often, GBS colonizes the baby during labor either by traveling upward from the mother's vagina into the uterus, or as the infant passes through the birth canal.

"When is GBS a Threat?"

GBS can be present in a woman's first pregnancy, or in following pregnancies. The bacteria can be a threat both during pregnancy and at the time of delivery. It has been shown that women who carry large amounts of the bacteria are at greatest risk of having a baby infected with GBS. Also, the occurrence of GBS infections are increased in certain high risk situations.

HIGH RISK SITUATIONS:

- When labor is premature;
- When there is premature rupture of the membranes;
- When there is prolonged rupture of membranes (>12 hours) before the baby is born;
- If the mother has a fever (>100.4 F) before or during labor;
- Women who have a history of GBS in previous births.

"Can GBS Infections Be Prevented?"

Yes. There is a fast and effective treatment for many situations. Medical research indicates that giving antibiotics to the mother during labor can greatly reduce the frequency of GBS infection in the baby immediately after birth or during the first week of life.

Treating the mother with oral antibiotics during the pregnancy may decrease the amount of GBS for a short time, but it will not eliminate the bacteria completely and will leave the baby unprotected at birth. Also, waiting to treat the baby with antibiotics after birth is often too late to prevent illness.

Carriers of GBS

Some doctors routinely screen for GBS by doing cultures on their patients during pregnancy. **These cultures must be taken from the lower vagina and rectum,** not the cervix.

Women who are found to carry the bacteria can then be treated as potential GBS risk patients. But, just like any other bacteria in the human body, GBS can be present in small amounts on one day which would result in a negative culture. Therefore, one negative culture result does not guarantee that you will be negative on the day of delivery. .Current studies indicate that a lower vaginal AND rectal culture done late in pregnancy is more than 93% accurate in detecting who will **not** carry the bacteria at delivery.)

At LPL following tests are available

- Streptococcus Group B antigen detection.
- Culture of vaginal and rectal swabs.

Dr Vikas Khillan
Head- microbiology and clinal pathology LPL..

Dr Lal PathLabs

Reactive Hypoglycemia..... A lurking fear

Reactive hypoglycemia or its synonym Post prandial hypoglycemia in otherwise normoglycemic individuals, though not taken too well, is not an unexpected finding in routine Blood Glucose Estimation in Clinical Labs of repute. Though diagnostic labs always have a relook in these reports of comparative low post prandial glucose levels before releasing it to clients or clinicians but then the labs are mentally prepared to answer volley of queries not only from patients/individuals but also from ambitious medical practitioners.

Maintenances of plasma glucose concentration within narrow bounds is essential for health. Lower levels of blood glucose relates to a pathological condition, popularly termed as hypoglycemia, which is a dangerous condition as brain for which primary source of energy substrate is only glucose tends to be vulnerable because it can not utilize circulating fatty acids as energy source as evident in other tissues.

Hypoglycemia can be classified as Postprandial (reactive) or fasting. Low glucose levels occurring in response to and as a follow up after meals is what we call it Reactive(PP) hypoglycemia. Levels of PP glucose falling below fasting could be marginal or more but than any levels falling below 2.5 mmol/litre (45 mg/dl) can lead to adverse effects producing recognizable symptoms which may be autonomic or neuroglycopenic but these are unlikely to surface on levels above 2.8 mmol/l (50mg/dl). It is commonly thought that fasting hypoglycemia is a serious condition with most likely etiopathology of insulinoma whereas reactive hypoglycemia is invariably a benign disorder.

However yet another type of hypoglycemia is drug induced which can be encountered in patients of diabetes mellitus who are either on insulin or oral hypoglycemics. Besides in normoglycemics drugs like alcohol, salicylates,B-blockers and pentamidine can lead to PP hypoglycemia.
Early/pre diabetes can be considered to occur in patients who are suffering from reactive hypoglycemias.

The most common cause of reactive hypoglycemia is Alimentary hyperinsulism. More often such patients have either undergone gastrectomy, pyloroplasty or vagotomy. Hypoglycemia in such patients occur anytime within 1 -2.30 hrs after food/ glucose intake. The reason attributed is excessive and early serum insulin response presumably due to rapid gastric emptying and brisk absorption of glucose thereby depleting glucose more than provide in meals. This feature can also be observed in patients without prior surgery.

Hormone deficiencies viz. growth hormone can also lead to reactive hypoglycemia. Besides glucagons deficiency and enzymes like fructose-1,6-diphosphatase deficiency or genetic derangements such as heredity fructose intolerance can lead to reactive hypoglycemias. Liver diseases may also contribute to pp hypoglycemia by affecting neoglucogenesis. Galactose intolerance ,a metabolic disorder, is also responsible for hypoglycemias. Renal glycosuria is yet another factor for causing postprandial reactive hypoglycemia

Overall high insulin sensitivity is tipped to be the most important cause for reactive hypoglycemias. The most usual cause of increased insulin response is most often assumed to be insulin resistance related hyperinsulinemia.

Under circumstances quoted above it is observed that glucose levels fall more rapidly than insulin thereby resulting in insulin-glucose imbalance and eventually leading to hypoglycemias.

The phenomena of reactive hypoglycemia was observed by Harris in 1924 who reported five cases of hypoglycemia following meals and the term was coined by him. He postulated that reactive hypoglycemia was some kind counterpart of diabetes mellitus resulting due to hyperinsulinism or dysinsulinism. It was a debatable question and finally in third international conference/symposium on hypoglycemia held in Rome on 22/23 sept'1986 a consensus statement was released that it was an overdiagnosed entity but than there was no doubt that some patients exhibit postprandial symptoms suggesting hypoglycemia in everyday life and if these symptoms are accompanied by blood glucose levels of 2.5mmol/litre than the diagnosis of postprandial or reactive hypoglycemia may be correct.

Symptoms of reactive hypoglycemia, unless PP glucose level is < 45mg/dl , may not be acknowledged by patient at all. However the most common symptoms are as listed below. Patient may suffer with one or all of them.

Hunger, nervousness, anxiety, perspiration , cold sweat, confusion, dizziness , sleepiness, light headedness or weakness.
Interestingly hyperinsulinemia is responsible for enhancing epinephrine, norepinepherine and cortisol secretion in response to hypoglycemia which collectively are involved in producing postprandial adrenergic syndrome.

Once it has been observed that PP levels of glucose are low , if not insisted upon for repeat, Oral Glucose Tolerance Test (OGTT) is advised after 10hrs of fasting observing all diet/ drugs and alcohol limitations. Since plasma glucose levels in healthy persons may be usually below 2.8 mmol/litre without any specific symptoms therefore OGTT has not been considered as a choice investigation though surely it still remains as first line investigation . Interestingly reactive hypoglycemia is quite a normal occurrence after OGTT whether or not individual suffers with hypoglycemia after meals in everyday life.

Therefore it is imperative to state that glucose tolerance test alone is not fully reliable means of establishing diagnosis of reactive hypoglycemia. It is suggested that no doubt the test should be carried out but than it must be combined with insulin response(Insulin Glucose tolerance) test. And glucose levels must be correlated with symptoms of hypoglycemia. Additional tests like C-peptide and proinsulin can also be advised.

Finally the reactive hypoglycemia, a functional disorder, suffered from apt approach in past as a result of inadequate knowledge regarding its mechanism but now as more facts are being revealed, is now considered to be a “non disease” by most Physicians.

A positive and well reasoned approach by clinicians and pathologists will definitely allay all lurking fears from mind of our patients. It is definitely not a lab error.

Dr. Kapil Bhalla
Consultant Biochemist &
Head Validation

HYPERTENSION

Hypertension is a condition of having high blood pressure. It is very common and is the major cause of morbidity & mortality.

The term hypertension means elevation in diastolic pressure, or systolic pressure. Elevation in either diastolic or systolic pressure represent a significant risk factor to a patient. Hypertension is often defined as a diastolic pressure of 90mm Hg or above or a systolic pressure of 140 mm Hg or above.

In most patients (90-95%) presenting with hypertension, the cause is unknown. This condition is called Primary hypertension. The remaining 5-10% of hypertensive patients have hypertension that results secondarily from renal disease, endocrine disorders, or other causes. This form of hypertension is called Secondary hypertension.

Hypertension is much more than “cardiovascular” disease as it affects other organs of the body such as :--

- Heart—leads to heart attack and heart failure
- Brain—leads to stroke and internal bleeding
- Kidneys—leads to renal failure and the need for dialysis
- Eyes—leads to blindness
- Peripheral Blood Vessels—leads to limping and tissue death (gangrene)
- If left untreated , the disease will progress and will eventually lead to death.

Hypertension is a Silent Killer disease without apparent symptoms yet the body is slowly being destroyed by high blood pressure.

A WHO sample study claims that one in every four senior citizens in the national capital suffers from hypertension. The findings are quite alarming—the incidence of hypertension was found to be about 25%. Epidemiological studies show a steadily increasing trend in the prevalence of hypertension across India over the last 40 years in contrast to the developed countries where a significant decrease has occurred

The prevalence of heart disease in urban areas in India is now between 15-30% which is four fold higher than in USA. A rapid increase in stroke mortality has also been reported. Hypertension and stroke occur at a relatively younger age in India. Indians develop coronary artery disease 5-10 years earlier than people in united states. Higher socioeconomic groups have a greater incidence of hypertension and coronary heart disease in India

Hypertension is a big problem in Philippinesas it has 21% prevalence rate. Roughly 8.6 million Filipions are hypertensive

It affects around 50 million Americans of which about 30% are not yet diagnosed

Hypertension is an under diagnosed disease because it causes damage to the body with no symptoms or mild symptoms.

Lead Poisoning

Lead, mercury, arsenic and cadmium are ranked first, second, third and sixth respectively in the U.S. Agency for Toxic Substances and Disease Registry which lists all hazards present in toxic waste sites according to their prevalence and severity of toxicity.

Metals such as lead and mercury are xenobiotic and can exert toxic effect at any level of exposure. Other metals such as copper and selenium are trace elements and are essential for normal metabolic function, however at high levels these are toxic.

Lead:- The twentieth century saw the greatest-ever exposure of the general population to lead and an extraordinary amount of new research on lead toxicity. The Centers for Disease Control and Prevention reported that one million children in the US have lead blood levels high enough to cause irreversible damage to their health.

Source:- Lead exposure is chiefly via paints, cans, plumbing and leaded gasoline. Other environmental sources include leafy vegetables grown in lead-contaminated soil, improperly glazed ceramics, lead crystal and certain herbal remedies.

Metabolism:- Elemental lead and inorganic lead compounds are absorbed via ingestion and inhalation; organic lead (tetraethyl lead, additive in gasoline) is absorbed through skin as well. Children absorb 50% of lead ingested compared to 10% absorbed by adults. Lead crosses the blood brain barrier and placenta and accumulates in bone and soft tissues; the skeleton contains >90% of the body's total lead burden. Up to 99% of lead in blood is present in the red cells bound to hemoglobin. Lead is excreted in urine and feces and is also present in sweat, saliva, breast milk, hair and nails. The half life of lead in blood is 25 days, in soft tissues 40 days and in bone >25 years.

Clinical Toxicology:- In children symptoms of lead toxicity appear at blood levels > 80 ug/dL and include abdominal pain, anorexia, irritability followed by lethargy, pallor, ataxia and slurred speech. In severe cases convulsions, coma and death occur due to generalized cerebral edema and renal failure. Subclinical lead poisoning can cause mental retardation and deficits in language, cognitive function, balance, behaviour and school performance. Lead's effect on intellectual capacity is probably dose-related and occurs at levels below 30ug/dL and is greatest when exposure is of long duration.

In adults symptoms are apparent at levels >80ug/dL and include abdominal pain, headache, irritability, joint pain, fatigue, anemia, peripheral motor neuropathy and deficits in short term memory. A lead line on gums is sometimes seen after prolonged exposure to high doses. Chronic subclinical exposure causes interstitial nephritis, tubular damage with inclusion bodies, hyperuricemia, decrease glomerular filtration and chronic renal failure.

Lead levels between 7 and 35 ug/dL are associated with increase blood pressure.

Lead that is dormant in bone may pose a threat at times of increased bone resorption such as pregnancy, lactation and osteoporosis. Hyperthyroidism can cause lead toxicity by mobilizing lead accumulated in bone.

In children lead levels should be maintained at <10ug/dL and in adults <40ug/dL.

In the US measurement of blood levels in children 6 months to 5 years of age is mandated; workers with occupational exposure to lead are also required to have blood lead levels monitored.

Investigations:- Anemia is usually normochromic and normocytic; basophilic stippling may be present.

Heme precursors (delta aminolevulinic acid) in plasma and urine can be elevated at blood lead levels as low as 15ug/dL

In children azotemia and pyuria can occur and in adults elevated serum creatinine and decreased creatinine clearance.

Lead lines ie increased metaphyseal plate density of long bones develops in children.

Prolonged nerve conduction time occurs due to peripheral demyelination.

Estimation of blood lead levels is required for definitive diagnosis of lead poisoning and for monitoring dangerous levels in asymptomatic individuals in order to prevent irreversible damage in both adults and children.

Detection Methods:- Colorimetric methods and anode stripping voltametry have been replaced by atomic absorption spectrophotometry for routine analysis of blood lead levels. Flame atomic absorption could detect blood levels of 60ug/dL; this was improved with the use of the Delves cup but was not very reproducible as it was operator dependent.

With the development of Graphite Furnace atomic absorption (GFAA) the detection capability was improved 200 times (0.1 ppb). A major advantage of the GFAA is automation with very little off-line sample preparation. Another major advantage is decreased interference from matrix components that are driven off before atomization at 2700°C, hence very low levels of lead can be accurately determined. The next major milestone in AS was the development of Zeeman correction which compensates for nonspecific

Absorption and structured background produced by complex biological matrices like blood and urine; this allows for the use of aqueous standards. GFAA has been the accepted method for blood lead estimation for more than 20 years.

ICP-MS is 50-100 times more sensitive than GFAA, however cost is a limiting factor for routine clinical use.

With the use of AAS the blood lead level considered dangerous in children was lowered from 60 ug/dL to 10ug/dL; AAS has also helped in identifying the environmental sources of contamination.

Modern AAS instrumentation has in addition helped enormously in understanding other trace elements such as the toxic effects of arsenic and chromium and the nutritional benefit of selenium and has impacted on the quality of human lives.

Dr. Anjali Kale
H.O.D.
Okhla Lab

Symptoms of hypertension includes :-

No symptoms or mild or vague symptoms

Headache (morning headache)

Dizziness

Confusion

Papilloedema

Symptoms due to complications of hypertension eg. heart disease,

Kidney disease, eye disease etc.

Sleep Apnea

Who are at risk ?

Smokers

Over weight persons

In take of salty, fried or greasy food

Alcoholics

Those under stress

"Sit down" life style

Man above 35 yrs.

Family history of hypertension

If your cholesterol level is over 200 mg/dl

Diabetics

Heart or renal failure patients

You had a stroke

Ways for a healthy lifestyle :-

Exercise regularly

Watch your weight

Keep stress under control

Avoid salty and fatty foods

Avoid too much of caffeine

Limit alcohol intake

Stop smoking

Take your medication properly

A frequent cause of failure to control blood pressure is the use of inappropriately low dosages of drugs inevitably leads to multiple drug regimens. Combination therapies containing a diuretic are

More effective .The new combinations of angiotensin

converting enzyme inhibitors and calcium channel blockers offer promise to control hypertension.

Diagnostic Tests recommended & available at LPL*:--

*Routine urinalysis

a blood cell count

*Comprehensive Lipid Profile

*KFT

*Diabetic profile

*Calcium

ECG

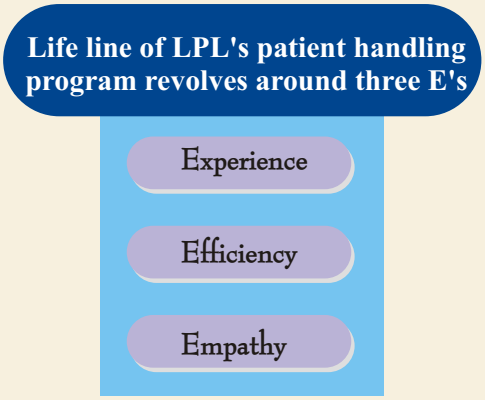
BP Check regularly

Dr. A.K.KAPOOR
Research Scientist

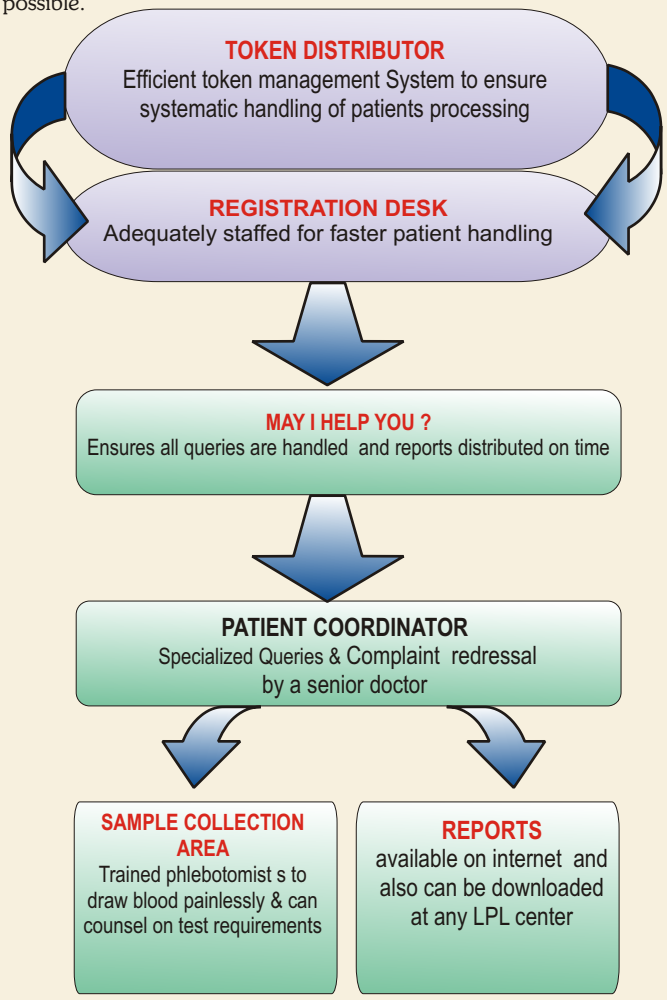
Patient is the King at LPL

It has been more than five decades that LPL is serving the nation. in the field of diagnosis utilizing latest techniques in Laboratory Medicine. We are today proud to be the most trustworthy and Quality conscious laboratory in India. (LPL is the first to attain laboratory accreditations and quality certifications. e g CAP, NABL, ISO)

Focus through out has been our patient ***..... our sole target audience***



The smoothness of operations here is that of a conveyer belt. At each stage hurdles are anticipated and eradicated so that our patients are not only satisfied but delighted. An insight into the planed structure of our MAIN LAB-**Registration-May I help you?-Patient Coordination and Phlebotomy area** shows that as soon as the patient enters our premises efforts are made to make his / her visit as short, hassle free, comfortable, painless and informative as possible.



Furthermore we take pride in saying that at LPL we nurture long term relationships. A dedicated customer care team ensures that patient feedback is collected , assessed and analysed to enable management in formulating policies for further patient satisfaction

Usha M Shanker
Quality Assurance

From the Editors Desk

Friends,

It has been really nice to receive all your encouragement and support. It is great to get comments and views on various topics that have been highlighted in our previous issue of INSIGHT.

In order to serve our patients better we are selecting the right combination of high Quality, Accuracy, Precision and Competitive pricing as we move along.

In this issue the article on "Hypertension" is included because it is a common and major cause of morbidity and mortality. Another article on "Lead Poisoning" is very informative. There is a great exposure of general population to lead toxicity. An article on "Reactive Hypoglycemia" explains the causes of low blood sugar values after carbohydrate meal in PP samples than that of fasting samples in non diabetic patients which is a common complaint of patients. Article on Group B streptococcus highlights the seriousness of this life threatening infection in new born babies. Last but not the least .....KING AT LPL.

A Red letter day in the history of LPL was on 21st January 07 when we organized the First Winter Cricket Tournament which had over enthusiastic response nearly 70 employees who were full of energy & enthusiasm participated in the tournament --A Report.

Last but not the least is a report on patient care who is treated like a King at LPL.

Thank you for having entrusted your faith in us . We look for your similar patronage in future as well.

Best wishes,

Dr. Meenu Beri
H.O.D. SDA Lab.

Test Report of the Cricketing Skills of the Employees of LPL

The Khalsa Cricket Ground, Delhi University, was full of enthusiastic cricketers of Dr. Lal Path Labs on 21st January,07. This was the first Winter Cricket Tournament organized by the company. In the beginning, we had thought of participation of a few amateur cricketers in this tournament but we were pleasantly surprised to receive more than 70 employees, all full of energy and enthusiasm.

The participants were subjected to selection process, which was supervised by a few professional players and coaches, specially invited to assess the abilities and worthiness of the players. We were extremely surprised that more than 90% of the players had top most skills and played with near perfection, like professional cricketers.

On the basis of the observation of the selectors, various teams were formed and then the matches started. Final match was played on 28th Jan,2007 at the same ground. Some of the players, especially, Anil Sharma, Rajesh Kumar, Lalit Singh, Gaurav Mittal, Ajay Gupta and Bhopal Singh played exceptionally well. The matches were played under strict professional rules and regulations.

The Tournament was widely covered by the press. We have now formed the LPL cricket team and are looking forward to playing matches with various cricket clubs and companies, which will not only boost the morale of the players but will also give wide publicity to the Company.

Roopak Vasishtha
Head-HR