Women’s height age or age height index and risk of cesarean delivery

Sir,

Women’s stature has been studied extensively as a predictor of mode of delivery. Short stature of women has shown to be associated with increased risk of cesarean section.1 However, controversy exists on such association. Mahmood et al2 in their study cohort of 563 women, noted that 80% of the women with height less than 160 cm delivered vaginally. Similarly, Moller et al3 suggested the role of regional variation among the women’s height, in predicting the risk for cesarian section.

Women’s age has also been implicated in influencing the route of delivery. Increased risk of cesarean section with advanced maternal age has been shown.4-6 As evident from the epidemiological studies that the variation in the range of women’s height for any population is small, as compared to wide range of women’s reproductive age.

To evaluate the impact of these two variables (height and age) on mode of delivery, a combination of these factors is needed. We established two indices, which included both factors (height and age), the height age index and age height index. We conducted this study to look at these factors in predicting the mode of delivery.

A total of 96 deliveries were reviewed, 48 with spontaneous vaginal delivery and 48 with cesarean section. Only primiparous women were included in the study. Charts were reviewed with respect to women’s height, age and mode of delivery. The women’s height age index was calculated as: height in cm divided by age in years (cm/year) while age height index by dividing age by height (year/cm). Variables are compared by using t-test, with the help of Statistical Package for the Social Sciences (Windows version 6.1), SPSS Inc, Chicago, Illinois.

The results are depicted in Table 1. We were able to demonstrate a statistically significant lower height age index and higher age height index in women who had cesarean sections as compared to the women who had vaginal deliveries. That suggested that these indices might be used, in addition to age or height alone, in predicting the risk of cesarean section in primiparous women.

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References

Table 1 - Summary of the results.

<table>
<thead>
<tr>
<th>Mode of delivery</th>
<th>Vaginal delivery</th>
<th>Cesarean section</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cases(n)</td>
<td>48</td>
<td>48</td>
<td></td>
</tr>
<tr>
<td>Women’s age height index (year/cm)</td>
<td>0.13±0.02</td>
<td>0.14±0.02</td>
<td>p=0.01*</td>
</tr>
<tr>
<td>Women’s height age index (cm/year)</td>
<td>7.1±1.06</td>
<td>6.6±0.86</td>
<td>p=0.02*</td>
</tr>
</tbody>
</table>

Results expressed as mean±standard deviation *p<0.05 was taken as statistical significant
Sir,

Neonatal Varicella has a high mortality and morbidity risk. Many health workers are not familiar with the case fatality of Neonatal Varicella or the significance of chickenpox developing in the mother towards delivery. There is also some misconception regarding the terms congenital and neonatal varicella and the use of Acyclovir. We would like to share our experience in Sultan Qaboos University Hospital recently. A 16-day-old baby born to a mother who developed chickenpox at the time of delivery, presented with neonatal varicella, pneumonia and seizures. Baby had received Varicella Zoster Immunoglobulin (VZIG) prophylaxis in the local hospital and was discharged home. There was a delay between onset of symptoms and initiation of Acyclovir therapy due to lack of awareness of the fatality of neonatal varicella. Baby was moribund and very sick and had to be intubated and ventilated for 10 days and ultimately recovered with Acyclovir, anticonvulsant and other supportive measures.

Chickenpox in the mother during pregnancy can lead to Congenital Varicella Syndrome on Neonatal Varicella depending on the time of onset of infection. Risk of Congenital Varicella embryopathy is highest between 13 and 20 weeks of gestation, with an incidence of 2-3%. Neonatal Varicella on the other hand, may occur when maternal disease develops after 37 weeks.

The highest risk of fatal infection in the newborn occurs when the mother becomes symptomatic between 5 days before and 2 days after delivery where the mortality is up to 30%. Hence it is worth considering certain facts: 1. The knowledge that Neonatal Varicella is fatal with a mortality up to 30% if the mother develops chickenpox within 4-5 days before delivery and 2 days after delivery, should be disseminated to the health workers and family members in contact with chickenpox; 2. VZIG to have maximum benefit, should preferably be used within 48 hours of exposure. Also administration of VZIG may prolong the incubation period, as well as modify the disease; 3. Intravenous Acyclovir to be fully effective should be given within 24 hours of onset of symptoms; 4. Oral Acyclovir in a dose of 40 mgm/kg/day in 4 divided doses for 5 days can prevent or modify clinical varicella if used late in the incubation period after household exposure. A neonate exposed to maternal varicella 4-5 days before and 2 days after delivery is immunocompromized, as these infants are born before transplacental transfer of maternal antibody and the immune responses of the neonate are insufficient to retard the growth and dissemination of Varicella Zoster Virus. Therefore, there seems to be a point in giving oral Acyclovir to abort or modify clinical varicella in this group of neonates before the disease manifests.

However, a prospective study is required to confirm the efficacy, as well as safety (especially renal) of oral Acyclovir in this neonatal age group, where the disease is fatal with visceral complications like viral pneumonia; 5. Once Varicella vaccine becomes freely available and routinely used, the incidence will probably become less.

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References
Guidelines and Code of Ethics#

Sir,

The implementation of Guidelines and Code of Ethics for peers like fellow authors, printed since January 1999 issue of Saudi Medical Journal, is commendable. Instruction to authors and "conflict of interest" are parts of the "uniform requirements for manuscripts submitted to biomedical journals.1 "Peer review" aims to improve quality, prohibit fraud, preserve "intellectual property" and direct money for research; issues which remain "news focus" under review elsewhere.2 Such pioneering editorial action is most helpful to science and medicine in particular as it encourages input from unknown authors.

Most materials published nowadays may be reruns of old data. New discoveries inducing breakthrough scientific advances may be based on concepts which come from somewhere unexpected and not necessarily from research centres and countries of excellence. Discoveries occur against odds and challenge a received wisdom. It is conceived by observation, implanted in a "lone" mind, stimulated by challenge and feeds on dedicated painstaking work and insomnia.

Concepts, however, could easily be "borrowed" but impossible to return. Discoveries compliment, but are not synonymous with, scientific advances and inventions, as it cannot be patented. Validation through competent scientific research may take years but well equipped teams may reproduce and print it in months. For reasons which have been comprehensively exposed,3-5 the implementation of "open peer review" is a most welcomed step.

David F. Horrobin, Editor of Medical Hypothesis, is the pioneer who was first to ask: "What is peer review for?" Current materialization of "open peer review" was seeded 25 years ago.3 His answer: "Quality control, but it works best when nothing of importance is at stake and is a hinder to scientific innovation".4 Like the unknown innovative scientists, he tried to bring to the attention of peers and editors, he was left "unwept, unhonored and unsung" (Walter Scott).

It is unusual that the main regulatory process for scientific research and publication to lack regulations, particularly in societies that strive on science and has a system that works for regulating everything from rocket launching to supermarket shopping. Every judge rules by law and juries verdict. Editors are the judges in science. Peers are the juries and though by definition have a vested interest, unlike in law they should not withdraw.

No apology for the corrupt. No profession is immune. The corrupt will always be back after finding new loop holes. Honest peers have nothing to fear. They know that comments which cannot be said openly, with references, are not worth saying at all. They recognize old from new. Professor Stehbens has laid foundations for new regulations and warned against "over regulations," leaving honest peers, like innovative authors, with little room to breath.5

As always, a minority of corrupts forces in regulations. The honest majority suffer most and bear the cost. But, should they? If not, who should pay? Scientists should be spared as doctors' altruism should be to the individual patient's care based on evidence-based ethical medicine. Before the whole scientific process of "clinical" research becomes only feasible on a "computer simulation", such issues and others must be also considered.3-5

In a world currently manipulated by the "lahlooh"§ how can honest peers, editors, authors and doctors, such as judges, keep intellectual integrity, compassion, fairness and altruism without having financial independence and safety of intellectual property? The scientific process, such as medicine itself should not be commercialized, valued or manipulated by the "lahlooh". It aims "to cure sometimes, to relieve often and to comfort always".4

§"Lahlooh" is a fictitious international currency unit with roots in Egyptian folklore; meaning: the thing which manipulates all others without ethics, moral or religion.

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References

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Primary Hyperparathyroidism

Sir,

I read the interesting article of "Primary Hyperparathyroidism and Pregnancy" by Dr. Mona A. Fouda, but I think there are some other valuable points worth mentioning. For example, the parathyroid adenoma of women can be asymptomatic during pregnancy and with the evaluation of a symptomatic infant after delivery (including hypocalcemia and tetany), the diagnosis of adenoma being confirmed.1,2 Other complications for the fetus include: increase intrauterine fetal growth retardation, spontaneous abortion and stillbirth.3 In addition, to maternal complaints described by Dr. Fouda, the patient may present herself with symptoms of toxemia of pregnancy, so differentiation between pre-eclampsia and the hyperparathyroidism should be kept in mind, due to similar symptomatic and clinical findings.4,5 Some authors explained that the mother presented herself first with the symptoms and signs of acute pancreatitis (including vomiting, nausea and abdominal pain). So the evaluation of the parathyroid glands for a co-existence of parathyroid adenoma during pregnancy should be considered in acute pancreatitis.6 Consequently, determination of calcium serum concentration of every trimester of pregnancy and regular intervals after delivery and also serial ultrasound evaluation of the fetal growth are recommended.3,4 And finally, about the mortality of this co-existence, some authors believe that this is related to delayed resection of parathyroid adenoma.6

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Sir,

I read with interest the recent article "primary hyperparathyroidism and pregnancy." The author has successfully alerted the readers to the coexistence of primary hyperparathyroidism and pregnancy that is a very rare and an easily overlooked situation. Hereby, I would like to elaborate important points about calcium-phosphate relationship and certain pitfalls in their interpretation from chemical pathological point of view.

1. It is worth stating that for diagnosing primary hyperparathyroidism there is nearly always hypercalcemia with occasionally serum calcium is only raised intermittently. However an increase in circulating PTH is usually, but not always, present and is not a consistent finding and so the results of PTH assay must be interpreted with caution. In the presence of hypercalcemia due to causes other than primary hyperparathyroidism, PTH production from the parathyroid glands should be suppressed to below normal range (i.e. undetectable). A PTH concentration even in the normal range in association with hypercalcemia is considered, therefore, inappropriate and suggests autonomous PTH secretion. Thus, primary hyperparathyroidism can be defined as a disturbance of parathyroid where circulating level of PTH is high or even inappropriately normal for the prevailing high concentration of plasma calcium (cf ADH secretion and hypo-osmolality in SIADH, insulin secretion and hypoglycemia in insulinoma). The identification of the laboratory contribution in the diagnosis of primary hyperparathyroidism is of paramount importance as nowadays, only about 20% of these patients have urolithiasis, and radiographically detectable bone disease is rare.

2. An important diagnostic criterion for diagnosing primary hyperparathyroidism is the deranged state of phosphate which the author didn’t make use of. The paradoxical relationship between serum calcium and phosphate (hypercalcemia with hypophosphataemia) occurs almost exclusively (in the absence of renal impairment) in primary hyperparathyroidism. This occurs due to the phosphaturic effect of PTH on renal tubules. Hence, a low (or even low-normal serum phosphate) with hypercalcemia are considered to be diagnostic for primary hyperparathyroidism. Furthermore, tests based on the renal response to excess PTH particularly concerning its phosphaturic effect are considered to be a further diagnostic prove. This can be elicited by careful assessment of the renal handling of phosphate. Measurement of 24 hours urine phosphate excretion, as such, is of limited value because of its variability and low specificity. It is, therefore, not surprising for the 24 hour urine phosphate to be low in the reported three cases (which is against the sounder physiological principles of excess PTH). Phosphate excretion was 3.0 mmol/day in patient 1, 15.2 mmol/day in patient 2 and 11.4 mmol/day in patient 3 (reference range 13-42 mmol/day).

An improvement in the validity of the phosphaturic effect of PTH on the kidney can be achieved by measurement of the indices of tubular reabsorption of phosphate. Although these tests have been replaced by the newer PTH immunoassay, however, their usefulness may still be considered in district hospitals using the commonly available simple data before further referral. These tests include the following:

- A. Ratio of phosphate clearance of creatinine clearance (Cp/Ccr) which gives the proportion of phosphate filtered at the glomeruli, which has been reabsorbed in the tubules. Normally the ratio is <0.15 and is often raised in primary hyperparathyroidism. This is calculated as: \( \frac{Cp}{Ccr} \) where: \( Ctr = 1 - \frac{Cp}{Ccr} \) X 100. With normal range being 84-95% and it is usually decreased in primary hyperparathyroidism.
- Percentage tubular reabsorbed phosphates (TRP) where: \( TRP = \left(1 - \frac{Cp}{Ccr}\right) \times 100 \). With normal range being 84-95% and it is usually decreased in primary hyperparathyroidism.
- Phosphate excretion index (PEI) which allows for changes in Cp and Cp/Ccr which can result from changes in serum phosphate and in phosphate intake. PEI is calculated as: \( PEI = \left(\frac{Cp}{Ccr}\right) - 0.05 \) with normal value being 0.12 to + 0.12 and it is often increased in primary hyperparathyroidism. An additional advantage in using these phosphate reabsorption and excretion indices is that their measurement does not necessitate timed 24-hour urine specimen. Instead, a random urine sample can be used for measuring urine phosphate and creatinine and together with the corresponding values in serum sample, calculation of these parameters can be made. It would, therefore, be advantageous to derive these indices in the reported three cases using the available data. Measurement of serum calcium and its interpretation in the light of other commonly available results (biochemical bone profile) including: serum phosphate, alkaline phosphatase, albumin (for correcting calcium), urea (for excluding renal impairment) and bicarbonate (for detecting any associated metabolic acidosis consequent upon inhibition of tubular bicarbonate reabsorption by excess PTH) may be sufficient for diagnosis. This may obviate the need for measurement of PTH, which is of very limited availability, even in the well-equipped hospital laboratory.

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Reply from the author

Sir,

I have received two correspondences from you regarding my manuscript published in your esteemed journal "primary hyperthyroidism and pregnancy". The first one from Dr. Payam S Pahlavan from Shaheed Beheshi University of Medical Sciences, Tehran, Iran with interesting expansion on the complications that could happen to the mother and the fetus. He confirmed what has been stressed in my manuscript on the need for routine screening for serum calcium level during pregnancy, and early resection of the parathyroid adenoma during pregnancy when feasible.

The second correspondence is from Dr. Waad Allah S. Mula-Abed from College of Medicine, University of Mosul, Mosul, Iraq. With his detailed biochemical analysis of the disturbed relationship between the calcium and phosphorus minerals and the PTH. However, his statement "hence a low or even low normal serum phosphate with hypercalcemia are considered to be diagnostic for primary hyperparathyroidism", is not totally true, since other causes of inappropriately high PTH or PTH-like peptides could account for a similar presentation, e.g. solid tumors, lithium therapy etc. The twenty-four-hour urinary phosphate excretion even though could be of further help as not very diagnostic, and the renal phosphate handling as provided by the equations quoted by Dr. Mula-Abed could be helpful, but since the advent of PTH essays it has been customary to do this since it also helps in the differential diagnosis of hypercalcemia especially the new standard intact PTH essay.

I would like to thank both correspondents for their interest in the manuscript and their valuable comments. With best regards.

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References