

The black dotted line: Controls without BP ($r=0.69$, $p<0.0001$). The solid line: Preterm AGA without BP ($r=0.38$, $p=0.11$), the red dotted: preterms AGA with BP ($r=0.06$, $p=NS$).

At 9 years, preterm AGA with BP ($n=13$) had lower length SDS ($p=0.003$), weight SDS ($p=0.006$) and head circumference SDS and a tendency to lower height catch-up ($p=0.09$) compared to preterm AGA without BP ($n=18$). Fasting levels of IGF-I, insulin and leptin were lower in all Preterms with BP.

Preterms with SP ($n=8$) had a lower height catch-up ($p=0.009$) compared to those without SP ($n=30$).

Conclusion Children born preterm have an increased risk for SP and BP. These disorders are associated with reduced catch up in height.

453 REPEAT COURSES OF ANTENATAL CORTICOSTEROIDS FOR PRETERM BIRTH AND RISK FOR METABOLIC SYNDROME IN YOUNG ADULTHOOD

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Background Preterm birth is associated with later hypertension and diabetes. One explanation for this association could be that exposure to antenatal corticosteroids (ACS), especially if repeated, induce adverse long-term effects. There are no data on whether repeat courses of ACS are associated with health problems later in life. The aim of this study was to assess whether repeat courses of ACS correlate to metabolic syndrome later in life.

Methods In a population-based cohort we measured BMI, blood pressure, arterial stiffness, blood lipids and glucose tolerance in 58 subjects (36 boys, age 14 to 26 years) exposed to 2–9 weekly courses of antenatal betamethasone. Subjects exposed to a single course ($n=25$, 14 boys) and unexposed subjects ($n=44$, 25 boys) were included as comparison groups.

Results As compared to unexposed controls, subjects exposed to repeat courses of ACS did not differ in BMI (mean difference 0.6 kg/m^2 , $p=0.5$), mean systolic or diastolic blood pressure (mean diff 1 mmHg , $p=0.78$ – 0.83), arterial stiffness assessed by pulse wave analysis (mean diff 0.1% , $p=0.50$), triglyceride (mean diff 0.1 mmol/L), total cholesterol (mean diff 0 mmol/L), LDL/HDL ratio (mean diff 0.1), Lipoprotein(a) (mean diff 61 mg/L), Apolipoprotein B/Apolipoprotein A1 ratio (mean diff 0.01), ($p=0.33$ – 0.91) or glucose tolerance assessed by HOMA-index (mean diff 0 , $p=0.84$). Subjects exposed to a single course of ACS did not differ from the other groups in any of the variables above.

Conclusions Repeat courses of ACS do not correlate to metabolic syndrome in young adulthood. This observation has clinical implications for the ongoing discussion about safety of antenatal steroids.

454 HEPATIC GLYCOGENOSIS IN TYPE I DIABETES MELLITUS: REPORT OF TWO CASES AND REVIEW OF THE LITERATURE

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Aim Hepatic glycogenosis is an underrecognized cause of serum transaminase elevations in poorly controlled type I diabetes mellitus, which has a relatively benign course with appropriate treatment.^{1,2} Objective of this study is to describe the aetiology, clinical presenting symptoms and treatment options.

Methods A report of two adolescents with poor controlled diabetes mellitus, hepatomegaly and serum transaminase elevations and a literature review.

Results Both cases presented with abdominal pain and hepatomegaly, combined with nausea and dyspeptic complaints. Laboratory investigation revealed marked elevation of serum transaminase levels. Synthetic function of the liver stayed intact. Abdominal ultrasound showed isolated, homogenous hepatomegaly, without other abdominal abnormalities. In one case liver biopsy was performed, showing hepatic glycogenosis. Other causes for hepatomegaly were excluded. With improved diabetic control all complaints improved within three weeks, with normalisation of serum transaminase levels.

Review of literature that hepatic glycogenosis, not frequently described, is an important complication of type I diabetes mellitus. Hepatic glycogenosis as result of glycogen storage in hepatocytes, caused by periods of hyperglycaemia and frequent insulin boluses. This process is reversible with improved glycaemic control.^{1,2}

Conclusions Hepatic glycogenosis is a important complication of type I diabetes mellitus which can be reversible with the proper treatment. Therefore, medical attention is necessary.

References

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455 IMPROVEMENT OF SERUM TESTOSTERONE IN DIABETIC RATS TREATED WITH METFORMIN AND NIGELLA SATIVA

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Background and Aims To evaluate the effect of metformin and *Nigella sativa* (alone or in combination) on improving the diabetic state of rats.

Methods Male Sprague-Dawley rats weighing 180–200g had induced diabetes using alloxan (150 mg/kg), then diabetic rats were treated daily for 45 days with metformin (0.5 g/Kg.b.wt), *Nigella sativa* (1 g/Kg.b.wt) or a mixture of metformin + *Nigella sativa* ($0.25\text{ g}+1\text{ g/Kg.b.wt}$) in a separated three groups and compared with a group of alloxanated diabetic rats as control. HbA1c, serum glucose, lipid profile, microalbuminuria (MA), ALT, AST, insulin, SHBG and total testosterone were measured using ELISA & spectrophotometer techniques, testis and liver tissue were examined histopathologically.

Results Both metformin and *Nigella sativa* were comparable in reducing serum glucose of the diabetic rats, furthermore, *Nigella sativa* showed a hypolipidaemic effect and it also improved liver functions. The level of serum insulin was significantly increased ($P<0.05$) in three groups. Importantly, using the mixture of metformin and *N.sativa* was less effective in improving diabetic state than using metformin or *N.sativa* alone, although it had improved serum level of testosterone and normalized the structure of testis.

Conclusion Using either metformin or *Nigella sativa* alone was more effective in improving the diabetic state of rats than using them in combination, although this combination was more effective in improving both serum level of testosterone and the structure of testis. This raise basic questions about the effect of interactions that may occur on using this mixture in the treatment of diabetes that necessitate further studies.

456 SEGMENTAL ACUTE SPINAL CORD SYNDROME: "AS A BOMB"

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