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Severely Jaundiced Body Builder: A Case of Acute Cholestatic Syndrome With Anabolic Androgenic Steroid Use

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INTRODUCTION: Illicit use of anabolic androgenic steroids (AAS) has been growing among athletes and body builders for cosmetic and performance purposes. We report a case of one of the possible hepatic complications of AAS, acute cholestatic syndrome (ACS), in a male with anabolic steroid abuse.

CASE DESCRIPTION/METHODS: 27-year-old Polish man with no past medical history presented with jaundice, itching of the body, dark-colored urine, clay-colored stools for 5 weeks. No abdominal pain was reported. Patient reported taking for 4 months 4 types of AAS (testosterone, oxandrolone, methasterone and trenbolone) for body building, which he stopped taking 6 weeks prior to initial presentation due to fatigue. Vitals were normal, and on exam he appeared to be a muscular, jaundiced male with scratch marks on his skin. Abdominal exam revealed a non-tender, non-distended abdomen, with difficulty palpating the liver edge due to the musculature. Laboratory tests were significant for total bilirubin of 34.8 mg/dL with direct of 23.3 mg/dL, AST 55 U/L, ALT 70 U/L, ALP 212 U/L, GGTT 57 U/L INR 1.1, creatinine 1.1 mg/dL. Liver ultrasound showed hepatomegaly with no biliary dilation and patent hepatic and portal veins. A liver biopsy was obtained showing portal neutrophil predominant inflammation with occasional eosinophils, mild portal fibrosis and cholestasis, suggestive of drug-induced hepatotoxicity. Liver function tests normalized after 4 months of cessation of AAS use, with resolution of his jaundice and pruritus.

DISCUSSION: Body builders usually prefer polydrug use of AAS to maximize the desired effects and hypothetically reduce side effects at the same time. But contrary to their belief, the concomitant use of different illicit AAS may be a factor in their toxicity, making difficult to link an adverse effect with a specific substance. Acute cholestatic syndrome, has been associated with use of the 17α - alkylated anabolic steroids (i.e. oxandrolone, methasterone), thought related to an increase of reactive oxygen. This leads to prolonged direct toxic effects and intrahepatic cholestasis, though the pathophysiology remains unclear. ACS usually occurs 1-4 months after steroid initiation presenting with nausea, fatigue, pruritis; later, jaundice, dark urine, with significantly elevated total bilirubin and ALP but mild aminotransferase elevation can be seen. Management consists of stopping anabolic steroid use, supportive care and symptomatic treatment of pruritus.

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Management of a Massive Liver Hemangioma: Does Size Matter?

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INTRODUCTION: The majority of “giant” hemangiomas remain asymptomatic with no cause for surgical intervention; however, this may not hold true for massive tumors. The following case will review the challenges facing both patients and physicians when managing these atypical tumors.

CASE DESCRIPTION/METHODS: A 49-year-old male with no medical problems presented with complaints of post-prandial bloating, early satiety and mild epigastric discomfort. The bloating was intermittent for several years; however, symptoms have recently curtailed his eating habits. CBC, BMP and liver enzymes were unremarkable. An ultrasound highlighted a massively enlarged liver extending into the pelvis and displacing surrounding organs. The liver parenchyma appeared to be replaced with a homogenous, hyperechoic lesion. An MRI then illustrated a 29.5 × 20.1 × 19.4 cm, strongly hyperintense mass on T-2 weighted sequences consistent with the diagnosis of a hemangioma. He was referred to a hepatobiliary surgeon and an extended right hepatectomy was eventually performed. Histopathology results described vast endothelial lined channels supported by thin fibrous stroma without features of malignancy. The patient returned to clinic four weeks after surgery reporting complete resolution of his symptoms.

DISCUSSION: Hemangiomas are the most common benign solid tumor of the liver with little to no risk of malignant transformation. Often discovered incidentally on imaging studies, the majority of these tumors remain indolent without the need for routine surveillance. Rarely these tumors become symptomatic, often correlating with tumor size. The definition of “giant” liver hemangioma remains controversial, with most authors assigning the label to tumors greater than 4cm or 3cm in size. It is for this reason that management of giant hemangiomas remains highly debated (i.e. observation versus resection). Recent studies have shown that tumors greater than 20cm in size pose a higher risk for GI symptoms related to mass effect on surrounding organs as well as causing a disturbance in the hematologic and coagulation systems. Surgical resection should be considered for symptomatic or complicated lesions, or when the diagnosis remains inconclusive. It is our belief that size classifications for giant hemangiomas requires further subgrouping to consider the danger of these massive tumors as well as the increased morbidity of surgery. Proper management of these tumors should be individualized to each patient and include a multidisciplinary team approach.
INTRODUCTION: Neurofibromatosis (NF) has been frequently associated with multiple cancers and the expansive growth of these tumors often is one of the reasons for their short life expectancy. The incidence of gastrointestinal stromal tumors (GIST) and malignant peripheral nerve sheath tumors (MPNST) in patients with NF tends to be higher than the general population. The literature describes patients with only one of these tumors. The patient presented in our report likely had both tumors (MPNST) in patients with NF. The incidence of gastrointestinal stromal tumors (GIST) and malignant peripheral nerve sheath tumors (MPNST) in patients with NF tends to be higher than the general population. Our patient presented with a mass that had features of both in the same tumor making the diagnosis difficult and its management uncertain. Reporting of other such cases may help establish a pattern regarding this entity.

CASE DESCRIPTION/METHODS: A 39-year-old male with neurofibromatosis type 2 affecting the spine presented to the ED with RUQ abdominal pain, nausea, vomiting and jaundice. Abdominal CT and SBP showed intrahepatic bile duct dilation with obstruction of the common duct at the gallbladder level. EUSC showed a localized biliary structure with dilation of the common hepatic, left and right hepatic, and intrahepatic ducts. A stent was placed, but brush cytology was non-diagnostic. EUS with FNA was performed, which was also non-diagnostic. Repeat MRI a month later revealed interval growth, comprising the porta hepatis. Exploratory laparotomy demonstrated a perihilar mass (5 cm × 5 cm) involving the entire CBD and part of the left lobe of the liver. A second small bowel tumor (2 cm × 2 cm) was also present. He underwent a radical resection of the peri-hilar mass, CBD, Roux-en-Y hepatico-jejunostomy to the intra-hepatic bile ducts, retroperitoneal and peri-portal lymphadenectomy, left partial hepatectomy and small bowel resection. The small bowel mass showed a GIST with typical morphology, a low mitotic rate and a conventional immunophenotype, with expression of CD117 and DOG-1, negative for epithelial membrane antigen, rare expression of SOX10 and retention of H3K27me3. The hilar mass was histologically active, malignant-appearing spindle cell neoplasm with necrosis. Its immunophenotype was not concordant with its morphologic features since it was found to be positive for both CD117 and DOG-1, scattered expression of SOX, diagnostic for GIST. The hilar mass showed complete loss of expression of H3K27me3, an immunohistochemical finding typically associated with malignant peripheral nerve sheath tumors.

DISCUSSION: GIST and MPNSTs are reported in patients with NF. Our patient presented with a mass that had features of both in the same tumor making the diagnosis difficult and its management uncertain. Reporting of other such cases may help establish a pattern regarding this entity.

GIST or MPNST or Both? An Enigmatic Mass

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Too Much TAE: A Case of Tumor Lysis Syndrome After Repeat Transarterial Embolization of Hepatocellular Carcinoma

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INTRODUCTION: Hepatocellular carcinoma (HCC) is the sixth most common cancer in the world & the second highest cause of cancer-related deaths globally. In the US, HCC incidence has almost tripled since the 1980s & has become the fastest rising etiology of cancer-related deaths. Tumor lysis syndrome (TLS) is a condition that results when malignant cells release their intracellular contents, either spontaneously or in response to cytotoxic treatment. The cellular contents result in hyperuricemia, hyperkalemia, hyperphosphatemia, & hypocalcemia; these disturbances can lead to erythropoietic, AKI & death. TLS is common in leukemia & lymphomas but is rare in solid tumors.

CASE DESCRIPTION/METHODS: A 50-year-old female with biopsy proven HCC without cirrhosis was admitted for planned transarterial chemoembolization (TACE) of her large right lobe & medial left lobe HCC, measuring 13.3 × 7.9 × 21.8 cm (Image 1), as too advanced for surgical resection or transplant & she had large liver-lung shunt preventing radioembolization treatment. Intentional Radiology (IR) placed two vials of LC beads containing doxorubicin 100mg & bland transarterial embolization (TAE) via 2.5 vials of Embospheres into right hepatic artery. Afterwards, a mass that had features of both in the same tumor making the diagnosis difficult and its management uncertain. Reporting of other such cases may help establish a pattern regarding this entity.

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