Impact of revised atlanta classification of acute pancreatitis on generation of a score employing modified CT severity index

Sir,

We read with great interest the article titled “Severity assessment of acute pancreatitis using CT severity index and modified CT severity index: Correlation with clinical outcomes and severity grading as per the Revised Atlanta Classification” by Sahu et al. published in the April–June 2017 issue of the Indian Journal of Radiology and Imaging. The manuscript is highly informative. We, however, would like to make the following pertinent observations.

One of the principal aims of the present study was to calculate the degree of concordance between the CT scoring indices and the clinical grading as per the Revised Atlanta Classification for Acute Pancreatitis. The former included CT Severity Index (CTSI) and Modified CT Severity Index (MCTSI), both of which generate a score based on CECT findings, thus, stratifying acute pancreatitis (AP) as mild, moderate, or severe. Revised Atlanta Classification for Acute Pancreatitis revolutionized the management of AP by simplifying clinical as well as morphological classifications and coining newer nomenclature for radiological findings to bring in more objectivity. Naturally, calculation of CT scoring indices, for e.g., MCTSI on the basis of newer terminology may alter the final score. For example:

a. Revised Atlanta Classification for Acute Pancreatitis considers any intrapancreatic collection straightaway parenchymal necrosis, which is in strong disagreement with the older terminology used to compute MCTSI. The latter grades intrapancreatic collection and parenchymal necrosis as two separate entities. Hence, MCTSI as per the latter (the older terminology) would be 2 or 4 (depending on the percentage of necrosis whether more than or less than 30%), while the former would generate a score of 4.

b. The nomenclature of fat islands in peripancreatic collections is yet another point of disagreement. While older terminology used to calculate MCTSI considers it as acute fluid collections without necrosis, revised terminology labels it to be acute necrotic collection (in acute stage).

The present study defines the CT parameters as per the Revised Atlanta Classification for Acute Pancreatitis. However, MCTSI, if computed using the recent classification, may yield a different score compared to when calculated as per the older terminology. Hence, it would be of enormous help if the authors could clarify our doubts.

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Nil.

Conflicts of interest
There are no conflicts of interest.
OHVIRA and OSVIRA syndrome

Sir,

I read with great interest the article titled, “Herlyn–Werner–Wunderlich syndrome presenting with infertility: Role of MRI in diagnosis” by Ahmad et al. in Indian Journal of Radiology and Imaging.[1] The manuscript is informative. However, I would like to make the following contributions.

OHVIRA, also known as Herlyn–Werner–Wunderlich syndrome, abbreviates a complex urogenital anomaly whose embryopathogenesis is still putative. It reads out as Obstructed HemiVagina and Ipsilateral Renal Agenesis/Anomaly.[1] Besides, it has a didelphic uterus.[1] Central to understanding of the pathology is embryogenesis of vagina, a disputed topic as yet. While classically the upper vagina is believed to have Mullerian (paramesonephric) roots akin to fallopian tubes, uterus and cervix with sinovaginal bulbs forming the remainder of the lower vagina, recent studies debunk this age‑old concept.[2] Acien[2] proposed Wolffian (mesonephric) origin of vagina in entirety – a notion which has been proved in experiments on female rats by Sanchez.[3] Using Acien’s hypothesis, all three components of OHVIRA can be fully explained.[2]

A faulty development of mesonephric duct fails to induce the metanephric blastema, the future kidney. Also because vagina is Wolffian in origin, it too does not develop. Further, lack of growth factors from the mesonephros disturbs the proper positioning and placement of the paired paramesonephric ducts, resulting in nonfusion (uterus didelphys). Hence, the result is OHVIRA syndrome.[2]

On a parallel track is a constellation of urogenital anomalies in males grouped under the so‑called Zinner syndrome (ZS). It comprises atresia of unilateral ejaculatory duct that leads to obstruction and dilation of seminal vesicle (seminal vesicle cyst) with ipsilateral renal agenesis.[4] Because all the components of this syndrome are mesonephric in origin, Aswani et al. postulated similar embryopathogenesis of ZS in males as that of OHVIRA in females (as per new hypothesis of Wolffian origin of vagina).[5] This concept thus places ZS as a male equivalent of OHVIRA, unlike previously where ZS was thought to be a male counterpart of Mayer–Rokitansky–Küster–Hauser (MRKH) syndrome in females.[6] The caveat here is that MRKH is a Mullerian anomaly, while ZS is Wolffian in origin.[5]

Finally, Aswani et al. proposed OSVIRA as an acronym for ZS, similar to its female equivalent OHVIRA, which expands as Obstructed Seminal Vesicle and Ipsilateral Renal Agenesis.[5]

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Raj Bharatkumar Koticha
Department of Radiology, HBT Medical College and RN Cooper Hospital, Vile Parle West, Mumbai, Maharashtra, India
E‑mail: rkoticha@gmail.com

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