manifests clinically as a yellowish mass in the lateral corner of the eye, which becomes more evident with retropulsion of the globe[2].

The imaging tests that can facilitate the diagnosis of subconjunctival fat prolapse are CT and magnetic resonance imaging (MRI) of the orbits, the most important radiological finding being that of a mass with fat density or fat-like signal intensity, respectively, located in the temporal aspect of the orbits, contiguous with intraconal fat.

The treatment consists of transconjunctival excision, a simple, safe and effective surgical procedure. The rate of recurrence after transconjunctival excision is reported to be approximately 9%[3].

Making a clinical diagnosis of subconjunctival fat prolapse is relatively easy. However, due to its rarity, it can be misdiagnosed as conjunctival dermolipoma, lymphoma, epidermoid cyst, or lacrimal gland prolapse[4]. The main differential diagnosis is conjunctival dermolipoma, which consists of a benign lesion, usually present at birth[5], that affects young women, the mean age of such patients being 22 years[6]. Although the clinical presentation of conjunctival dermolipoma is similar to that of the subconjunctival fat prolapse, the former is typically unilateral and fairly immobile. On CT and MRI, conjunctival dermolipoma presents as a crescent-shaped fatty mass in the temporal aspect of the orbit, not in communication with the intraconal fat[1].

In conjunctival dermolipoma, surgical resection is indicated mainly for aesthetic purposes and tends to be more conservative[1]. Although resection of a conjunctival dermolipoma is a simple procedure, there can be severe complications, including blepharoptosis, diplopia, and keratoconjunctivitis sicca. Therefore, a number of different surgical techniques aimed at a lowering the rate of complications and improving the aesthetic results have been described, including resection with conjunctival flap rotation[7].

Subconjunctival fat prolapse and dermolipoma present clinically as a fatty epibulbar masses in the lateral corners of the orbits, and in some cases their differentiation by clinical aspects can be difficult. The subject is little known among radiologists, and there have been few reports of related cases. Therefore, given the difference between these two entities in terms of treatment, it is necessary that radiologists be familiar with both, in order to recognize them promptly and make the differential diagnosis through the use of imaging tests.

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**Cynthia Ramos Tejo[1], Péricles Almeida da Costa[1], Rafaella Martins Batista[1], Yuri Raoni Ramalho Rocha[1], Marcelle Alves Borba[1]**

1. Universidade Federal do Rio Grande do Norte (UFRN), Natal, RN, Brazil. Mailing address: Dra. Cynthia Ramos Tejo. Hospital Universitário Onofre Lopes. Avenida Nilo Peçanha, 620, Petrópolis. Natal, RN, Brazil, 59012-300. E-mail: cynthiatejo@gmail.com.


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### Ogilvie syndrome after use of vincristine: tomographic findings

*Dear Editor,*

A 33-year-old female patient with diffuse large B-cell non-Hodgkin lymphoma was evaluated two days after the end of the first cycle of chemotherapy. The chemotherapy regimen comprised a five-day cycle, including rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisone on the first day, whereas prednisone alone was administered on the four remaining days. She reported left pleuritic pain and flatus with evacuation. She was afebrile. The abdomen was flaccid and peristaltic, without painful decompression. Because she had neutropenia, she was hospitalized, after which she evolved to having no bowel movements, with the smell of feces on her breath and painful abdominal decompression. Computed tomography (CT) of the chest and abdomen showed left pleural effusion, intestinal obstruction in the descending colon adjacent to the splenic flexure, that segment being of normal caliber, without occlusive lesions, although the transverse ascending colon and cecum were dilated, the latter being 14 cm in diameter (Figures 1 and 2). There was gas in the rectal ampulla. These findings were suggestive of acute colonic pseudo-obstruction. Colonoscopic

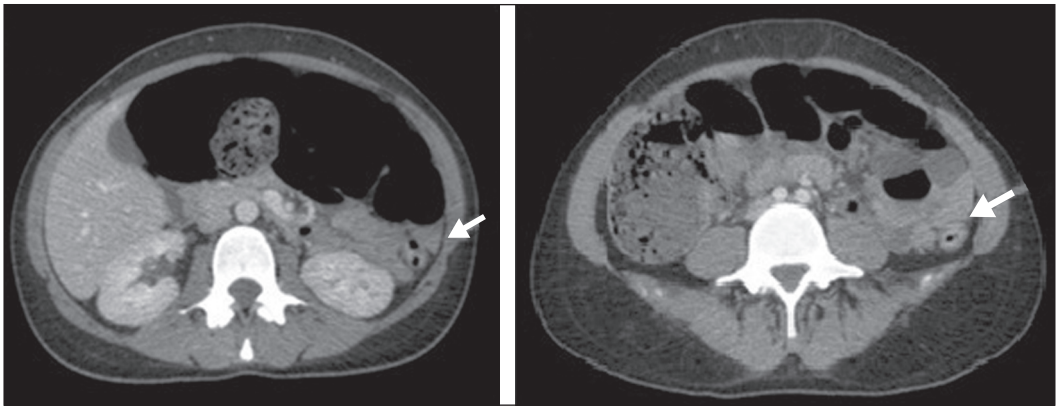

**Figure 1.** CT scan of the abdomen, in axial sections, obtained 60 s after injection of iodinated anionic contrast. Note the intestinal obstruction at the level of the proximal descending colon, adjacent to the splenic flexure. Distension of the transverse colon, ascending colon, and cecum, with the presence of fecal matter. The transitional zone can be seen at the level of the splenic flexure (arrow), with no evident obstructive material.

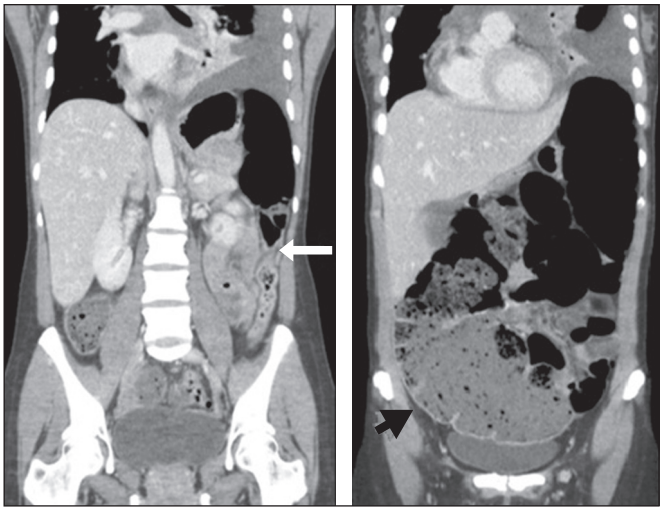

**Figure 2.** Coronal reconstruction of a CT scan, providing a better view of the transitional zone, where an abrupt transition to a normal caliber segment is observed, with no evident occlusive lesion (arrow). Note the marked dilation of the cecum, which measured 14 cm in diameter (arrowhead). Left pleural effusion can also be seen.

decompression and enema use were not considered because of the risk of cecal perforation. Therefore, the pseudo-obstruction was confirmed surgically. Thereafter, the patient was treated with gastric rest and her electrolyte levels were monitored.

Ogilvie's syndrome was named after William Heneage Ogilvie, who, in 1948, described a disorder of gastrointestinal motility, with dilation of the cecum and right colon in the absence of mechanical obstruction, that was autonomic in origin, with suppression of parasympathetic activity and activation of sympathetic activity[1].

The acute form of Ogilvie's syndrome arises from an autonomic imbalance, with a mismatch between parasympathetic and sympathetic activity, which are downregulated and upregulated, respectively. The distal colon is often atonic, whereas the proximal colon can still be functional[2]. Some chemotherapeutic agents have been implicated, such as those in the rituximab-cyclophosphamide-doxorubicin-vincristine-prednisone regimen, as have factors such as trauma, acute myocardial injury, electrolyte disturbances, hypothyroidism, renal failure, and neuropathy[2]. Lee et al.[3] observed that cancer patients developed Ogilvie's syndrome two to ten days after infusion of vincristine, the syndrome resolving after its discontinuation. Sandler et al.[4] found that patients treated with vincristine experienced abdominal pain and constipation within the first 4–72 hours after receiving the drug. Neutropenia and the use of antibiotic therapy have also been implicated in the development of the syndrome[3].

The symptoms of Ogilvie's syndrome include abdominal distension, abdominal pain, vomiting of fecal matter, and constipation[1,5]. Signs of peritonitis can indicate cecal perforation with pneumoperitoneum[6], especially when the distension is greater than 12 cm and lasts for more than six days. For evaluating diseases of the colon, CT has been shown to be the method of choice[7–11]. In Ogilvie's syndrome, CT is a useful for identifying the obstruction and determining the underlying cause[12], the main findings being dilation extending from the cecum to the transverse colon, with a transition zone in the splenic flexure, where the caliber of the adjoining loop is considerably smaller. The treatment involves the use of parasympathomimetic agents that increase colonic motility[13], endoscopic decompression or right hemicolectomy, the last being required in the presence of cecal ischemia or perforation.

Colonic pseudo-obstruction is associated with the use of chemotherapy. It is characterized by dilation of the loops of the colon and transitional zone. Attention should be paid to signs of perforation and the risk of death from cecal rupture.

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**Fernanda Miraldi Clemente Pessôa[1], Leonardo Kayat Bittencourt[2], Alessandro Severo Alves de Melo[1]**

1. Hospital Universitário Antonio Pedro – Universidade Federal Fluminense (HUAP-UFF), Niterói, RJ, Brazil. 2. Universidade Federal Fluminense (UFF), Niterói, RJ, Brazil. Mailing address: Dra. Fernanda Miraldi Clemente Pessôa. Rua Ouro Branco, 66, ap. 301, Vila Valqueire. Rio de Janeiro, RJ, Brazil, 21321-560. E-mail: fernandamiraldi@hotmail.com.


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### Pontine tegmental cap dysplasia accompanied by a duplicated internal auditory canal

*Dear Editor,*

A 48-year-old female with cognitive and auditory deficits presented for evaluation prior to cochlear implantation. Among her parents and four siblings, there was one brother with mental disability of unknown cause. Physical examination revealed ataxia. An electrophysiological study of hearing revealed the absence of waves from the cochlear nerve and of auditory brainstem pathways evoked by 95 dB nHL clicks and 500–1000 Hz tone bursts (also at an intensity of 95 dB nHL). There was also an absence of otoacoustic emissions in both ears, indicating profound sensorineural hearing loss. Computed tomography (CT) of the ears showed a narrow, duplicated internal auditory canal, one canal containing the facial nerve and the other containing