Aguda e Plaquetopenia severa (< 5000 plaquetas). Exame para hemólise positivos: Haptogobina 2 mg/dL, presença de hemácias fragmentadas, LDH 1402 U/L e Coombs direto negativo. Marcadores reumatológicos negativos. Durante investigação, identificada causa de turvação visual como descolamento seroso de retina bilateral por coriorretinopatia hipertensiva relacionada à hemólise. Portanto, paciente com quadro pouco característico para PTT apesar de PLASMIC score de 6 pontos. Solicitada dosagem de ADAMTS 13, porém com previsão de liberação do resultado em 14 dias. Realizada corticoterapia com Prednisona inicialmente (1 mg/Kg/dia) e, após, Dexametasona (40 mg/dia), com melhora clínica e laboratorial. Dentro de duas semanas, resultado do ADAMTS 13 liberado pelo laboratório com baixa atividade, confirmando o diagnóstico de PTT. Recebe alta hospitalar para seguimento ambulatorial. Em Setembro de 2022, apresenta novo episódio de sangramentos e plaquetopenia (Plaquetas 3000). Interna em UTI por hemólise franca: Hb: 6,7 mg/dL, Haptoglobina 1 mg/dL, BT 5,6 mg/dL (BD: 2 mg/dL), LDH 2068 U/L e Coombs direto negativo. ADAMTS 13 < 0,4%. Realizada plasmaférese (10 sessões) + Pulsoterapia com Metilprednisolona, porém sem resposta satisfatória. Optado então por Ciclosporina em dose terapêutica, porém ainda sem resposta, com persistência de esquizócitos em sangue periférico e ascensão de LDH. Iniciado tratamento com Rituximabe (375 mg/m^2), tendo apresentado melhora da hemólise inicialmente, porém mantendo plaquetopenia após o C1 (60.000). Realizados 5 ciclos de Rituximabe, com resposta completa após o término do tratamento, recuperando contagem de plaquetas (212.000) e ADDAMTS 13 44%. Segue acompanhamento ambulatorial. **Discussão:** O uso de plasmaférese tornou-se a terapia padrão para a PTT, reduzindo as taxas de mortalidade de 90% para 10%–30%. Porém, alguns pacientes podem se apresentar com casos refratários e recidivas, que pioram o prognóstico e podem necessitar de terapias adicionais, como no caso da paciente em questão, que respondendeu apenas após o uso de Rituximabe. Além disso, evidencia-se a forma mais agressiva da doença em um caso de recaída e aponta para a importância do acompanhamento a longo prazo dos níveis de ADAMTS 13 como preditor de novas recaídas e pior prognóstico para o paciente. **Conclusão:** Este relato exemplifica como doenças raras muitas vezes demandam tratamentos alternativos e como o acesso às essas terapias não padronizadas como primeira linha em serviços públicos pode dificultar e retardar o manejo da enfermidade e um melhor prognóstico para o paciente.

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MUTATIONAL STATUS OF ONCOGENE KRAS AND RISK OF VENOUS THROMBOSIS IN PATIENTS WITH METASTATIC COLORECTAL CANCER

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Background: Venous Thromboembolism (VTE) is a common cause of death and complications in patients with cancer. Choice of anticoagulant is challenging because of high risk of bleeding and recurrent VTE. Some tumor oncogenes, such as KRAS, a GTPase signaling protein that regulates proliferation, differentiation and cell survival can modulate the expression of genes at the core of hemostasis control. Clinically, KRAS mutation status is a validated predictive biomarker that is currently used to determine those mCRC (metastatic colorectal cancer) patients likely not to benefit from anti-EGFR therapy. A retrospective cohort study with 172 patients with mCRC has suggested that KRAS mutation (codons 12/13) could increase the risk of VTE. **Aim:** To analyze the risk VTE of patients with mCRC based on KRAS status. **Methods:** Records of 111 patients with metastatic or unresectable locally advanced colorectal cancer, attended at Clinica AMO (Salvador, Brazil) between January 2015 and January 2020, tested for mutational status of KRAS were reviewed. Seven mutations within codons 12 e 13 of KRAS detected by PCR (Idylla technique) or were considered for this study. Non-metastatic cases, those with insufficient clinical data, VTE provoked by catheter use and superficial vein thrombosis were excluded. This study was approved by an institutional Ethics Committee. Odds Ratios and 95% Confidence Intervals were obtained from the regression coefficients. A value of $p < 0.05$ was statistically significant. **Results and discussion:** One-hundred eleven patients were selected; the median age was 64 (22–79) and 44.1% were male. Sixty-three patients (56.7%) had a KRAS activating mutation and 48 (43.2%) were found to be KRAS wild-type (Fig. 1), the incidence being comparable to the literature (approximately 30%–50%) (6). Twenty-three (20,7%) patients suffered at least 1 episode of VTE, 5 (4,5%) Pulmonary Embolism (PE), 15 (13.5%) low-extremity Deep-Vein Thrombosis (DVT) and 3 (2.7%) presented both PE and DVT. All six patients with thrombosis in catheter had PE or DVT and there was no need to be excluded. KRAS mutated status was not associated with an elevated risk of thrombosis (15.8% vs. 27%, OR = 0.50, 95% CI 0.20–1.28) as well for the outcome of PE, DVT and both PE and DVT (Table 1). Although Ades, et al. (2015) found mutated KRAS as risk factor for VTE and remained significant despite adjustment for Khorana score and bevacizumab treatment, data are insufficient and conflicting. A Cohort with 194 patients, 41 patients (21.1%) who experienced VTE, ECOG ≥2 was the only independent predictor. Moreover, an abstract of 184 patients with mCRC shown limited evidence that mCRC patients harboring KRAS mutations may be at greater risk for VTE. In Table 2, we described VTE patient's characteristics according to oncogenic status. More than a half were metastatic and all patients had performance status ECOG 0–1 at diagnosis. Personal characteristics and treatment may interfere in VTE outcome and may be considered in further investigations to analyze if KRAS has impact on patient care and associated morbidity. **Conclusion:** In our study, biomarker KRAS have not been associated with an increased risk of VTE in advanced stage CRC patients and there is no evidence to select patients to whom VTE thromboprophylaxis should be offered.

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