

clinicians are unaware of some aspects that explain why its diagnosis may be initially missed, making the patient susceptible to unnecessary exploration or treatment. This study aimed to describe DILI characteristics linked to ACC in the LATINDILI registry.

**Materials and Methods:** We revised data concerning DILI-ACC in the LATINDILI registry during the last decade, looking for information on latency, pattern, severity, and evolution. Baseline characteristics were described using mean, median, and percentages; Student's t-test or a chi-squared test was used to determine the difference between mean and frequencies. A P-value of less than 0.05 was considered statistical significance.

**Results:** We identified 61 DILI-ACC episodes in 60 patients from the LATINDILI registry. The mean age was 58 years (19-90 y), and 54% were male. Median latency was 21 days, with median ALT and ALP at DILI onset of 282 U/L (range 34-2130) and 585 U/L (range 96-1626), respectively; a cholestatic/mixed pattern predominated in 43 cases. In 53 cases, the liver injury appeared with a mean of 13 days (range 2-39 d) after treatment ended. Twenty patients (33%) had allergic immune features, 79% were jaundiced, and 61% required hospitalization. The mean total bilirubin values increased by 7.5 mg/dl (1.5-16) from the onset in 24 of 42 evaluable patients after ten days (range 2-30). Table 1 shows the comparison between groups. Resolution of liver injury occurred on average 64 (14-270) days, one patient did not normalize after 365 days, and no death was consigned.

**Conclusions:** Jaundice linked to a cholestatic/mixed pattern appearing after stopping therapy was a frequent presentation of ACC in our analysis. This clinical presentation may be missed when using ACC and explaining the delayed diagnosis. Worsening bilirubin value is frequent and may be related to longer treatment duration and prolonged latency.

**Table 1.** Comparison between groups with and without bilirubin increment after the initial evaluation. (\*delay in the reduction of more than 50% of maximum bilirubin value).

	Total population (n 61)	Bilirubin increment after onset (n 24)	No bilirubin increment after onset (n 18)	P value
Male (%)	33 (54)	13 (54)	11 (61)	0.65
Mean age (years)	58	59	59.7	0.89
Cholestatic/Mixed Pattern (%)	43 (71)	18 (75)	12 (67)	0.55
Duration of treatment (days)	9.2	11.1	6.9	<b>0.01</b>
Latency (days)	21 (1-46)	25 (6-46)	16 (1-38)	<b>0.004</b>
Dechallenge (days)*	21 (4-51)	22 (6-51)	17 (4-40)	0.1
Bilirubin at DILI recognition	5.7 (0.4-15.7)	7.5 (1-15.7)	5.0 (0.4-15)	0.055
Mean peak bilirubin (mg/dL)	8.7 (0.4-22)	14.4 (2.8-22)	5.0 (0.4-15)	
Mean time to peak bilirubin (days)	10 (2-30)	10 (2-30)	-	

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### O-38 FULMINANT WILSON: A WELL-DEFINED ENTITY. HOW TO DIAGNOSE IT QUICKLY TO PREVENT MORTALITY

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**Introduction and Objectives:** McCullough published in 1983 a series of 3 cases from the Mayo Clinic and nine from the rest of the world, proposing a specific clinical entity of Wilson's disease (WD) with a fatal outcome if not transplanted. Costa Rica has the highest incidence of Wilson's disease in the world (5.2/100,000 inhabit); we found in a total population of patients, 5.8% of fulminant Wilson (FW) (7/120 patients), and in a pediatric population, 14.7% (5/34 children).

This study aimed to define FW as an entity by its clinical, biochemical, histological and genetic characteristics to diagnose earlier because of its high mortality.

**Materials and Methods:** We analyze the publications and cases of WF from Costa Rica up to 2020 and also review the literature around the world.

**Results:** WF is diagnosed principally in patients without the previous diagnosis of WD, principally in female patients (90%), with an onset between 10 and 21 years of age. Manifestations usually start with encephalopathy in the first eight weeks of symptoms, associated with fever peaks higher than 38°C, rapidly progressing jaundice, significant leukocytosis, sudden coombs negative hemolytic anemia, with total hyperbilirubinemia greater than 35 mg/dl, mild elevation of transaminases and alkaline phosphatase, prothrombin less than 20%, acute renal failure, ceruloplasminemia less than 10mg/dl, urinary cooper greater than 1000µg and elevated serum cooper. Micro vesicular steatosis, submassive necrosis, regenerative nodules, canalicular cholelithiasis, levels greater than 600ug/g dry weight in the liver, and hemoglobinuric necrosis in the kidney are also seen. Genetically all with a homozygous Pn1270 S mutation. With a fatal clinical course if not transplanted.

**Conclusions:** Patients with fulminant liver failure with coombs negative hemolytic anemia and clinical, biochemical, histological and genetic characteristics define FW, therefore, requesting priority one liver transplantation in identified patients it's a necessity.

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### O-39 CLINICAL CHARACTERISTICS AND OUTCOME OF DRUG-INDUCED LIVER INJURY DUE TO ANTINEOPLASTIC AND BIOLOGICAL AGENTS

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**Introduction and Objectives:** Idiosyncratic drug-induced liver injury (DILI) caused by antineoplastic and biological agents is an emerging clinical burden in oncologic patients. However, clinical characteristics of DILI due to these drugs remain poorly understood. This study aimed to assess the clinical presentation and outcome of